UNIVERSITY MOHAMMED V-RABAT
FACULTY OF MEDICINE AND
PHARMACY RABAT

YEAR: 2020 ***THESIS N°: 313***

MALIGNANT TEMPORAL LOBE GLIOMA

THESIS

Publicly presented in: ………………………………

BY

Mr. TALOUNI AYMENE

Born in 26/08/1994

Royal school of medical military service - Rabat

To obtain the medical doctorate degree

Key words : Malignant gliomas, temporal lobe, surgery, radiotherapy.

JURY

Mr Miloud GAZZAZ Professor of neurosurgery	**PRESIDENT**
Mr CHERIF EL ASRI Abad Professor of neurosurgery	**PROTRACTOR**
Mr Adyl MELHAOUI Professor of neurosurgery	**JUDGES**
Mr Hassan Ennouali Professor of radiology	
Mr Hassan SIFAT Professor of radiotherapy	

بسم الله الرحمن الرحيم

سبحانك لا علم لنا إلا ما علمتنا
إنك أنت العليم الحكيم

صدق الله العظيم

سورة البقرة: الآية: 31

UNIVERSITY MOHAMMED V
FACULTY OF MEDICINE AND PHARMACY
RABAT

HONORQRY DEANS :

1962 – 1969: Professor Abdelmalek FARAJ

1969 – 1974: Professor Abdellatif BERBICH
1974 – 1981: Professor Bachir LAZRAK
1981 – 1989: Professor Taieb CHKILI
1989 – 1997: Professor Mohamed Tahar ALAOUI
1997 – 2003: Professor Abdelmajid BELMAHI
2003 - 2013: Professor Najia HAJJAJ – HASSOUNI

ADMINISTRATION :

Dean	**Professor Mohamed ADNAOUI**
Vice-*Dean in charge of academic and student affairs*	Professor Brahim LEKEHAL
Vice-Dean in charge of research and cooperation	Professor Toufiq DAKKA
Vice-Dean in charge of pharmaceutical affairs	Professor Younes RAHALI
General secretary	Mr. Mohamed KARRA

1 - ENSEIGNANTS-CHERCHEURS MEDECINS ET PHARMACIENS

PROFESSEURS DE L'ENSEIGNEMENT SUPERIEUR :

Décembre 1984

Pr. MAAOUNI Abdelaziz	Médecine Interne – ***Clinique Royale***
Pr. MAAZOUZI Ahmed Wajdi	Anesthésie -Réanimation
Pr. SETTAF Abdellatif	Pathologie Chirurgicale

Décembre 1989

Pr. ADNAOUI Mohamed	Médecine Interne –***Doyen de la FMPR***
Pr. OUAZZANI Taïbi Mohamed Réda	Neurologie

Janvier et Novembre 1990

Pr. KHARBACH Aîcha	Gynécologie -Obstétrique
Pr. TAZI Saoud Anas	Anesthésie Réanimation

Février Avril Juillet et Décembre 1991

Pr. AZZOUZI Abderrahim	Anesthésie Réanimation- ***Doyen de FMPO***
Pr. BAYAHIA Rabéa	Néphrologie
Pr. BELKOUCHI Abdelkader	Chirurgie Générale
Pr. BENCHEKROUN Belabbes Abdellatif	Chirurgie Générale
Pr. BENSOUDA Yahia	Pharmacie galénique
Pr. BERRAHO Amina	Ophtalmologie
Pr. BEZAD Rachid	Gynécologie Obstétrique ***Méd. Chef Maternité des Orangers***
Pr. CHERRAH Yahia	Pharmacologie
Pr. CHOKAIRI Omar	Histologie Embryologie
Pr. KHATTAB Mohamed	Pédiatrie
Pr. SOULAYMANI Rachida	Pharmacologie- ***Dir. du Centre National PV Rabat***
Pr. TAOUFIK Jamal	Chimie thérapeutique

Décembre 1992

Pr. AHALLAT Mohamed	Chirurgie Générale ***Doyen de FMPT***
Pr. BENSOUDA Adil	Anesthésie Réanimation
Pr. CHAHED OUAZZANI Laaziza	Gastro-Entérologie
Pr. CHRAIBI Chafiq	Gynécologie Obstétrique
Pr. EL OUAHABI Abdessamad	Neurochirurgie
Pr. FELLAT Rokaya	Cardiologie
Pr. JIDDANE Mohamed	Anatomie
Pr. TAGHY Ahmed	Chirurgie Générale

Pr. ZOUHDI Mimoun	Microbiologie

Mars 1994

Pr. BENJAAFAR Noureddine	Radiothérapie
Pr. BEN RAIS Nozha	Biophysique
Pr. CAOUI Malika	Biophysique
Pr. CHRAIBI Abdelmjid	Endocrinologie et Maladies Métaboliques ***Doyen de la FMPA***
Pr. EL AMRANI Sabah	Gynécologie Obstétrique
Pr. ERROUGANI Abdelkader	Chirurgie Générale – ***Directeur du CHIS***
Pr. ESSAKALI Malika	Immunologie
Pr. ETTAYEBI Fouad	Chirurgie Pédiatrique
Pr. IFRINE Lahssan	Chirurgie Générale
Pr. RHRAB Brahim	Gynécologie –Obstétrique
Pr. SENOUCI Karima	Dermatologie

Mars 1994

Pr. ABBAR Mohamed*	Urologie ***Inspecteur du SSM***
Pr. BENTAHILA Abdelali	Pédiatrie
Pr. BERRADA Mohamed Saleh	Traumatologie – Orthopédie
Pr. CHERKAOUI Lalla Ouafae	Ophtalmologie
Pr. LAKHDAR Amina	Gynécologie Obstétrique
Pr. MOUANE Nezha	Pédiatrie

Mars 1995

Pr. ABOUQUAL Redouane	Réanimation Médicale
Pr. AMRAOUI Mohamed	Chirurgie Générale
Pr. BAIDADA Abdelaziz	Gynécologie Obstétrique
Pr. BARGACH Samir	Gynécologie Obstétrique
Pr. EL MESNAOUI Abbes	Chirurgie Générale
Pr. ESSAKALI HOUSSYNI Leila	Oto-Rhino-Laryngologie
Pr. IBEN ATTYA ANDALOUSSI Ahmed	Urologie
Pr. OUAZZANI CHAHDI Bahia	Ophtalmologie
Pr. SEFIANI Abdelaziz	Génétique
Pr. ZEGGWAGH Amine Ali	Réanimation Médicale

Décembre 1996

Pr. BELKACEM Rachid	Chirurgie Pédiatrie
Pr. BOULANOUAR Abdelkrim	Ophtalmologie
Pr. EL ALAMI EL FARICHA EL Hassan	Chirurgie Générale
Pr. GAOUZI Ahmed	Pédiatrie

Pr. OUZEDDOUN Naima	Néphrologie
Pr. ZBIR EL Mehdi*	Cardiologie ***Directeur HMI Mohammed V***

Novembre 1997

Pr. ALAMI Mohamed Hassan	Gynécologie-Obstétrique
Pr. BIROUK Nazha	Neurologie
Pr. FELLAT Nadia	Cardiologie
Pr. KADDOURI Noureddine	Chirurgie Pédiatrique
Pr. KOUTANI Abdellatif	Urologie
Pr. LAHLOU Mohamed Khalid	Chirurgie Générale
Pr. MAHRAOUI CHAFIQ	Pédiatrie
Pr. TOUFIQ Jallal	Psychiatrie ***Directeur Hôp.Ar-razi Salé***
Pr. YOUSFI MALKI Mounia	Gynécologie Obstétrique

Novembre 1998

Pr. BENOMAR ALI	Neurologie ***Doyen de la FMP Abulcassis***
Pr. BOUGTAB	Abdesslam Chirurgie Générale
Pr. ER RIHANI Hassan	Oncologie Médicale
Pr. BENKIRANE Majid*	Hématologie

Janvier 2000

Pr. ABID Ahmed*	Pneumo-phtisiologie
Pr. AIT OUAMAR Hassan	Pédiatrie
Pr. BENJELLOUN Dakhama Badr.Sououd	Pédiatrie
Pr. BOURKADI Jamal-Eddine	Pneumo-phtisiologie ***Directeur Hôp. My Youssef***
Pr. CHARIF CHEFCHAOUNI Al Montacer	Chirurgie Générale
Pr. ECHARRAB El Mahjoub	Chirurgie Générale
Pr. EL FTOUH Mustapha	Pneumo-phtisiologie
Pr. EL MOSTARCHID Brahim*	Neurochirurgie
Pr. TACHINANTE Rajae	Anesthésie-Réanimation
Pr. TAZI MEZALEK Zoubida	Médecine Interne

Novembre 2000

Pr. AIDI Saadia	Neurologie
Pr. AJANA Fatima Zohra	Gastro-Entérologie
Pr. BENAMR Said	Chirurgie Générale
Pr. CHERTI Mohammed	Cardiologie
Pr. ECH-CHERIF EL KETTANI Selma	Anesthésie-Réanimation
Pr. EL HASSANI Amine	Pédiatrie - ***Directeur Hôp.Cheikh Zaid***
Pr. EL KHADER Khalid	Urologie
Pr. GHARBI Mohamed El Hassan	Endocrinologie et Maladies Métaboliques

Pr. MDAGHRI ALAOUI Asmae	Pédiatrie

Décembre 2001

Pr. BALKHI Hicham*	Anesthésie-Réanimation
Pr. BENABDELJLIL Maria	Neurologie
Pr. BENAMAR Loubna	Néphrologie
Pr. BENAMOR Jouda	Pneumo-phtisiologie
Pr. BENELBARHDADI Imane	Gastro-Entérologie
Pr. BENNANI Rajae	Cardiologie
Pr. BENOUACHANE Thami	Pédiatrie
Pr. BEZZA Ahmed*	Rhumatologie
Pr. BOUCHIKHI IDRISSI Med Larbi	Anatomie
Pr. BOUMDIN El Hassane*	Radiologie
Pr. CHAT Latifa	Radiologie
Pr. DAALI Mustapha*	Chirurgie Générale
Pr. EL HIJRI Ahmed	Anesthésie-Réanimation
Pr. EL MAAQILI Moulay Rachid	Neuro-Chirurgie
Pr. EL MADHI Tarik	Chirurgie-Pédiatrique
Pr. EL OUNANI Mohamed	Chirurgie Générale
Pr. ETTAIR Said	Pédiatrie - ***Directeur Hôp. Univ. Cheikh Khalifa***
Pr. GAZZAZ Miloudi*	Neuro-Chirurgie
Pr. HRORA Abdelmalek	Chirurgie Générale ***Directeur Hôpital Ibn Sina***
Pr. KABIRI EL Hassane*	Chirurgie Thoracique
Pr. LAMRANI Moulay Omar	Traumatologie Orthopédie
Pr. LEKEHAL Brahim	Chirurgie Vasculaire Périphérique ***V-D chargé Aff Acad. Est.***
Pr. MEDARHRI Jalil	Chirurgie Générale
Pr. MIKDAME Mohammed*	Hématologie Clinique
Pr. MOHSINE Raouf	Chirurgie Générale
Pr. NOUINI Yassine	Urologie
Pr. SABBAH Farid	Chirurgie Générale
Pr. SEFIANI Yasser	Chirurgie Vasculaire Périphérique
Pr. TAOUFIQ BENCHEKROUN Soumia	Pédiatrie

Décembre 2002

Pr. AL BOUZIDI Abderrahmane*	Anatomie Pathologique
Pr. AMEUR Ahmed *	Urologie
Pr. AMRI Rachida	Cardiologie
Pr. AOURARH Aziz*	Gastro-Entérologie ***Dir.-Adj. HMI Mohammed V***
Pr. BAMOU Youssef *	Biochimie-Chimie
Pr. BELMEJDOUB Ghizlene*	Endocrinologie et Maladies Métaboliques

Pr. BENZEKRI Laila	Dermatologie
Pr. BENZZOUBEIR Nadia	Gastro-Entérologie
Pr. BERNOUSSI Zakiya	Anatomie Pathologique
Pr. CHOHO Abdelkrim *	Chirurgie Générale
Pr. CHKIRATE Bouchra	Pédiatrie
Pr. EL ALAMI EL Fellous Sidi Zouhair	Chirurgie Pédiatrique
Pr. EL HAOURI Mohamed *	Dermatologie
Pr. FILALI ADIB Abdelhai	Gynécologie Obstétrique
Pr. HAJJI Zakia	Ophtalmologie
Pr. JAAFAR Abdeloihab*	Traumatologie Orthopédie
Pr. KRIOUILE Yamina	Pédiatrie
Pr. MOUSSAOUI RAHALI Driss*	Gynécologie Obstétrique
Pr. OUJILAL Abdelilah	Oto-Rhino-Laryngologie
Pr. RAISS Mohamed	Chirurgie Générale
Pr. SIAH Samir *	Anesthésie Réanimation
Pr. THIMOU Amal	Pédiatrie
Pr. ZENTAR Aziz*	Chirurgie Générale

Janvier 2004

Pr. ABDELLAH El Hassan	Ophtalmologie
Pr. AMRANI Mariam	Anatomie Pathologique
Pr. BENBOUZID Mohammed Anas	Oto-Rhino-Laryngologie
Pr. BENKIRANE Ahmed*	Gastro-Entérologie
Pr. BOULAADAS Malik	Stomatologie et Chirurgie Maxillo-faciale
Pr. BOURAZZA Ahmed*	Neurologie
Pr. CHAGAR Belkacem*	Traumatologie Orthopédie
Pr. CHERRADI Nadia	Anatomie Pathologique
Pr. EL FENNI Jamal*	Radiologie
Pr. EL HANCHI ZAKI	Gynécologie Obstétrique
Pr. EL KHORASSANI Mohamed	Pédiatrie
Pr. HACHI Hafid	Chirurgie Générale
Pr. JABOUIRIK Fatima	Pédiatrie
Pr. KHARMAZ Mohamed	Traumatologie Orthopédie
Pr. MOUGHIL Said	Chirurgie Cardio-Vasculaire
Pr. OUBAAZ Abdelbarre *	Ophtalmologie
Pr. TARIB Abdelilah*	Pharmacie Clinique
Pr. TIJAMI Fouad	Chirurgie Générale
Pr. ZARZUR Jamila	Cardiologie

Janvier 2005

Pr. ABBASSI Abdellah	Chirurgie Réparatrice et Plastique

Pr. ALLALI Fadoua	Rhumatologie	
Pr. AMAZOUZI Abdellah	Ophtalmologie	
Pr. BAHIRI Rachid	Rhumatologie	***Directeur Hôp. Al Ayachi Salé***
Pr. BARKAT Amina	Pédiatrie	
Pr. BENYASS Aatif	Cardiologie	
Pr. DOUDOUH Abderrahim*	Biophysique	
Pr. HAJJI Leila	Cardiologie	*(mise en disponibilité)*
Pr. HESSISSEN Leila	Pédiatrie	
Pr. JIDAL Mohamed*	Radiologie	
Pr. LAAROUSSI Mohamed	Chirurgie Cardio-vasculaire	
Pr. LYAGOUBI Mohammed	Parasitologie	
Pr. SBIHI Souad	Histo-Embryologie Cytogénétique	
Pr. ZERAIDI Najia	Gynécologie Obstétrique	

AVRIL 2006

Pr. ACHEMLAL Lahsen*	Rhumatologie
Pr. BELMEKKI Abdelkader*	Hématologie
Pr. BENCHEIKH Razika	O.R.L
Pr. BIYI Abdelhamid*	Biophysique
Pr. BOUHAFS Mohamed El Amine	Chirurgie - Pédiatrique
Pr. BOULAHYA Abdellatif*	Chirurgie Cardio – Vasculaire. ***Directeur Hôpital Ibn Sina Marr.***
Pr. CHENGUETI ANSARI Anas	Gynécologie Obstétrique
Pr. DOGHMI Nawal	Cardiologie
Pr. FELLAT Ibtissam	Cardiologie
Pr. FAROUDY Mamoun	Anesthésie Réanimation
Pr. HARMOUCHE Hicham	Médecine Interne
Pr. IDRISS LAHLOU Amine*	Microbiologie
Pr. JROUNDI Laila	Radiologie
Pr. KARMOUNI Tariq	Urologie
Pr. KILI Amina	Pédiatrie
Pr. KISRA Hassan	Psychiatrie
Pr. KISRA Mounir	Chirurgie – Pédiatrique
Pr. LAATIRIS Abdelkader*	Pharmacie Galénique
Pr. LMIMOUNI Badreddine*	Parasitologie
Pr. MANSOURI Hamid*	Radiothérapie
Pr. OUANASS Abderrazzak	Psychiatrie
Pr. SAFI Soumaya*	Endocrinologie
Pr. SOUALHI Mouna	Pneumo – Phtisiologie
Pr. TELLAL Saida*	Biochimie
Pr. ZAHRAOUI Rachida	Pneumo – Phtisiologie

Octobre 2007

Pr. ABIDI Khalid	Réanimation médicale
Pr. ACHACHI Leila	Pneumo phtisiologie
Pr. ACHOUR Abdessamad*	Chirurgie générale
Pr. AIT HOUSSA Mahdi *	Chirurgie cardio vasculaire
Pr. AMHAJJI Larbi *	Traumatologie orthopédie
Pr. AOUFI Sarra	Parasitologie
Pr. BAITE Abdelouahed *	Anesthésie réanimation
Pr. BALOUCH Lhousaine *	Biochimie-chimie
Pr. BENZIANE Hamid *	Pharmacie clinique
Pr. BOUTIMZINE Nourdine	Ophtalmologie
Pr. CHERKAOUI Naoual *	Pharmacie galénique
Pr. EHIRCHIOU Abdelkader *	Chirurgie générale
Pr. EL BEKKALI Youssef *	Chirurgie cardio-vasculaire
Pr. EL ABSI Mohamed	Chirurgie générale
Pr. EL MOUSSAOUI Rachid	Anesthésie réanimation
Pr. EL OMARI Fatima	Psychiatrie
Pr. GHARIB Noureddine	Chirurgie plastique et réparatrice
Pr. HADADI Khalid *	Radiothérapie
Pr. ICHOU Mohamed *	Oncologie médicale
Pr. ISMAILI Nadia	Dermatologie
Pr. KEBDANI Tayeb	Radiothérapie
Pr. LOUZI Lhoussain *	Microbiologie
Pr. MADANI Naoufel	Réanimation médicale
Pr. MAHI Mohamed *	Radiologie
Pr. MARC Karima	Pneumo phtisiologie
Pr. MASRAR Azlarab	Hématologie biologique
Pr. MRANI Saad *	Virologie
Pr. OUZZIF Ez zohra *	Biochimie-chimie
Pr. RABHI Monsef *	Médecine interne
Pr. RADOUANE Bouchaib*	Radiologie
Pr. SEFFAR Myriame	Microbiologie
Pr. SEKHSOKH Yessine *	Microbiologie
Pr. SIFAT Hassan *	Radiothérapie
Pr. TABERKANET Mustafa *	Chirurgie vasculaire périphérique
Pr. TACHFOUTI Samira	Ophtalmologie
Pr. TAJDINE Mohammed Tariq*	Chirurgie générale
Pr. TANANE Mansour *	Traumatologie-orthopédie
Pr. TLIGUI Houssain	Parasitologie
Pr. TOUATI Zakia	Cardiologie

Mars 2009

Pr. ABOUZAHIR Ali *	Médecine interne
Pr. AGADR Aomar *	Pédiatrie
Pr. AIT ALI Abdelmounaim *	Chirurgie Générale
Pr. AKHADDAR Ali *	Neuro-chirurgie
Pr. ALLALI Nazik	Radiologie
Pr. AMINE Bouchra	Rhumatologie
Pr. ARKHA Yassir	Neuro-chirurgie ***Directeur Hôp.des Spécialités***
Pr. BELYAMANI Lahcen *	Anesthésie Réanimation
Pr. BJIJOU Younes	Anatomie
Pr. BOUHSAIN Sanae *	Biochimie-chimie
Pr. BOUI Mohammed *	Dermatologie
Pr. BOUNAIM Ahmed *	Chirurgie Générale
Pr. BOUSSOUGA Mostapha *	Traumatologie-orthopédie
Pr. CHTATA Hassan Toufik *	Chirurgie Vasculaire Périphérique
Pr. DOGHMI Kamal *	Hématologie clinique
Pr. EL MALKI Hadj Omar	Chirurgie Générale
Pr. EL OUENNASS Mostapha*	Microbiologie
Pr. ENNIBI Khalid *	Médecine interne
Pr. FATHI Khalid	Gynécologie obstétrique
Pr. HASSIKOU Hasna *	Rhumatologie
Pr. KABBAJ Nawal	Gastro-entérologie
Pr. KABIRI Meryem	Pédiatrie
Pr. KARBOUBI Lamya	Pédiatrie
Pr. LAMSAOURI Jamal *	Chimie Thérapeutique
Pr. MARMADE Lahcen	Chirurgie Cardio-vasculaire
Pr. MESKINI Toufik	Pédiatrie
Pr. MESSAOUDI Nezha *	Hématologie biologique
Pr. MSSROURI Rahal	Chirurgie Générale
Pr. NASSAR Ittimade	Radiologie
Pr. OUKERRAJ Latifa	Cardiologie
Pr. RHORFI Ismail Abderrahmani *	Pneumo-Phtisiologie

Octobre 2010

Pr. ALILOU Mustapha	Anesthésie réanimation
Pr. AMEZIANE Taoufiq*	Médecine Interne ***Directeur ERSSM***
Pr. BELAGUID Abdelaziz	Physiologie
Pr. CHADLI Mariama*	Microbiologie
Pr. CHEMSI Mohamed*	Médecine Aéronautique
Pr. DAMI Abdellah*	Biochimie- Chimie

Pr. DARBI Abdellatif*	Radiologie
Pr. DENDANE Mohammed Anouar	Chirurgie Pédiatrique
Pr. EL HAFIDI Naima	Pédiatrie
Pr. EL KHARRAS Abdennasser*	Radiologie
Pr. EL MAZOUZ Samir	Chirurgie Plastique et Réparatrice
Pr. EL SAYEGH Hachem	Urologie
Pr. ERRABIH Ikram	Gastro-Entérologie
Pr. LAMALMI Najat	Anatomie Pathologique
Pr. MOSADIK Ahlam	Anesthésie Réanimation
Pr. MOUJAHID Mountassir*	Chirurgie Générale
Pr. NAZIH Mouna*	Hématologie
Pr. ZOUAIDIA Fouad	Anatomie Pathologique

Decembre 2010

Pr. ZNATI Kaoutar	Anatomie Pathologique

Mai 2012

Pr. AMRANI Abdelouahed	Chirurgie pédiatrique
Pr. ABOUELALAA Khalil *	Anesthésie Réanimation
Pr. BENCHEBBA Driss *	Traumatologie-orthopédie
Pr. DRISSI Mohamed *	Anesthésie Réanimation
Pr. EL ALAOUI MHAMDI Mouna	Chirurgie Générale
Pr. EL OUAZZANI Hanane *	Pneumophtisiologie
Pr. ER-RAJI Mounir	Chirurgie Pédiatrique
Pr. JAHID Ahmed	Anatomie Pathologique
Pr. RAISSOUNI Maha *	Cardiologie

Février 2013

Pr. AHID Samir	Pharmacologie
Pr. AIT EL CADI Mina	Toxicologie
Pr. AMRANI HANCHI Laila	Gastro-Entérologie
Pr. AMOR Mourad	Anesthésie Réanimation
Pr. AWAB Almahdi	Anesthésie Réanimation
Pr. BELAYACHI Jihane	Réanimation Médicale
Pr. BELKHADIR Zakaria Houssain	Anesthésie Réanimation
Pr. BENCHEKROUN Laila	Biochimie-Chimie
Pr. BENKIRANE Souad	Hématologie
Pr. BENNANA Ahmed*	Informatique Pharmaceutique
Pr. BENSGHIR Mustapha *	Anesthésie Réanimation
Pr. BENYAHIA Mohammed *	Néphrologie
Pr. BOUATIA Mustapha	Chimie Analytique et Bromatologie

Pr. BOUABID Ahmed Salim*	Traumatologie orthopédie
Pr. BOUTARBOUCH Mahjouba	Anatomie
Pr. CHAIB Ali *	Cardiologie
Pr. DENDANE Tarek	Réanimation Médicale
Pr. DINI Nouzha *	Pédiatrie
Pr. ECH-CHERIF EL KETTANI	Mohamed Ali Anesthésie Réanimation
Pr. ECH-CHERIF EL KETTANI Najwa	Radiologie
Pr. ELFATEMI Nizare	Neuro-chirurgie
Pr. EL GUERROUJ Hasnae	Médecine Nucléaire
Pr. EL HARTI Jaouad	Chimie Thérapeutique
Pr. EL JAOUDI Rachid *	Toxicologie
Pr. EL KABABRI Maria	Pédiatrie
Pr. EL KHANNOUSSI Basma	Anatomie Pathologique
Pr. EL KHLOUFI Samir	Anatomie
Pr. EL KORAICHI Alae	Anesthésie Réanimation
Pr. EN-NOUALI Hassane *	Radiologie
Pr. ERRGUIG Laila	Physiologie
Pr. FIKRI Meryem	Radiologie
Pr. GHFIR Imade	Médecine Nucléaire
Pr. IMANE Zineb	Pédiatrie
Pr. IRAQI Hind	Endocrinologie et maladies métaboliques
Pr. KABBAJ Hakima	Microbiologie
Pr. KADIRI Mohamed *	Psychiatrie
Pr. LATIB Rachida	Radiologie
Pr. MAAMAR Mouna Fatima Zahra	Médecine Interne
Pr. MEDDAH Bouchra	Pharmacologie
Pr. MELHAOUI Adyl	Neuro-chirurgie
Pr. MRABTI Hind	Oncologie Médicale
Pr. NEJJARI Rachid	Pharmacognosie
Pr. OUBEJJA Houda	Chirugie Pédiatrique
Pr. OUKABLI Mohamed *	Anatomie Pathologique
Pr. RAHALI Younes	Pharmacie Galénique ***Vice-Doyen à la Pharmacie***
Pr. RATBI Ilham	Génétique
Pr. RAHMANI Mounia	Neurologie
Pr. REDA Karim *	Ophtalmologie
Pr. REGRAGUI Wafa	Neurologie
Pr. RKAIN Hanan	Physiologie
Pr. ROSTOM Samira	Rhumatologie
Pr. ROUAS Lamiaa	Anatomie Pathologique
Pr. ROUIBAA Fedoua *	Gastro-Entérologie
Pr SALIHOUN Mouna	Gastro-Entérologie

Pr. SAYAH Rochde	Chirurgie Cardio-Vasculaire
Pr. SEDDIK Hassan *	Gastro-Entérologie
Pr. ZERHOUNI Hicham	Chirurgie Pédiatrique
Pr. ZINE Ali *	Traumatologie Orthopédie

AVRIL 2013

Pr. EL KHATIB MOHAMED KARIM *	Stomatologie et Chirurgie Maxillo-faciale

MARS 2014

Pr. ACHIR Abdellah	Chirurgie Thoracique
Pr. BENCHAKROUN Mohammed *	Traumatologie- Orthopédie
Pr. BOUCHIKH Mohammed	Chirurgie Thoracique
Pr. EL KABBAJ Driss *	Néphrologie
Pr. EL MACHTANI IDRISSI Samira *	Biochimie-Chimie
Pr. HARDIZI Houyam	Histologie- Embryologie-Cytogénétique
Pr. HASSANI Amale *	Pédiatrie
Pr. HERRAK Laila	Pneumologie
Pr. JANANE Abdellah *	Urologie
Pr. JEAIDI Anass *	Hématologie Biologique
Pr. KOUACH Jaouad*	Génycologie-Obstétrique
Pr. LEMNOUER Abdelhay*	Microbiologie
Pr. MAKRAM Sanaa *	Pharmacologie
Pr. OULAHYANE Rachid*	Chirurgie Pédiatrique
Pr. RHISSASSI Mohamed Jaafar	CCV
Pr. SEKKACH Youssef*	Médecine Interne
Pr. TAZI MOUKHA Zakia	Génécologie-Obstétrique

DECEMBRE 2014

Pr. ABILKACEM Rachid*	Pédiatrie
Pr. AIT BOUGHIMA Fadila	Médecine Légale
Pr. BEKKALI Hicham *	Anesthésie-Réanimation
Pr. BENAZZOU Salma	Chirurgie Maxillo-Faciale
Pr. BOUABDELLAH Mounya	Biochimie-Chimie
Pr. BOUCHRIK Mourad*	Parasitologie
Pr. DERRAJI Soufiane*	Pharmacie Clinique
Pr. DOBLALI Taoufik	Microbiologie
Pr. EL AYOUBI EL IDRISSI Ali	Anatomie
Pr. EL GHADBANE Abdedaim Hatim*	Anesthésie-Réanimation
Pr. EL MARJANY Mohammed*	Radiothérapie
Pr. FEJJAL Nawfal	Chirurgie Réparatrice et Plastique
Pr. JAHIDI Mohamed*	O.R.L

Pr. LAKHAL Zouhair*	Cardiologie
Pr. OUDGHIRI NEZHA	Anesthésie-Réanimation
Pr. RAMI Mohamed	Chirurgie Pédiatrique
Pr. SABIR Maria	Psychiatrie
Pr. SBAI IDRISSI Karim*	Médecine préventive, santé publique et Hyg.

AOUT 2015

Pr. MEZIANE Meryem	Dermatologie
Pr. TAHIRI Latifa	Rhumatologie

PROFESSEURS AGREGES :

JANVIER 2016

Pr. BENKABBOU Amine	Chirurgie Générale
Pr. EL ASRI Fouad*	Ophtalmologie
Pr. ERRAMI Noureddine*	O.R.L
Pr. NITASSI Sophia	O.R.L

JUIN 2017

Pr. ABBI Rachid*	Microbiologie
Pr. ASFALOU Ilyasse*	Cardiologie
Pr. BOUAYTI El Arbi*	Médecine préventive, santé publique et Hyg.
Pr. BOUTAYEB Saber	Oncologie Médicale
Pr. EL GHISSASSI Ibrahim	Oncologie Médicale
Pr. HAFIDI Jawad	Anatomie
Pr. OURAINI Saloua*	O.R.L
Pr. RAZINE Rachid	Médecine préventive, santé publique et Hyg.
Pr. ZRARA Abdelhamid*	Immunologie

NOVEMBRE 2018

Pr. AMELLAL Mina	Anatomie
Pr. SOULY Karim	Microbiologie
Pr. TAHRI Rajae	Histologie-Embryologie-Cytogénétique

NOVEMBRE 2019

Pr. AATIF Taoufiq *	Néphrologie
Pr. ACHBOUK Abdelhafid *	Chirurgie Réparatrice et Plastique
Pr. ANDALOUSSI SAGHIR Khalid *	Radiothérapie
Pr. BABA HABIB Moulay Abdellah *	Gynécologie-obstétrique
Pr. BASSIR RIDA ALLAH	Anatomie
Pr. BOUATTAR TARIK	Néphrologie

Pr. BOUFETTAL MONSEF	Anatomie
Pr. BOUCHENTOUF Sidi Mohammed *	Chirurgie Générale
Pr. BOUZELMAT Hicham *	Cardiologie
Pr. BOUKHRIS Jalal *	Traumatologie-orthopédie
Pr. CHAFRY Bouchaib *	Traumatologie-orthopédie
Pr. CHAHDI Hafsa *	Anatolmie Pathologique
Pr. CHERIF EL ASRI Abad *	Neurochirugie
Pr. DAMIRI Amal *	Anatolmie Pathologique
Pr. DOGHMI Nawfal *	Anesthésie-réanimation
Pr. ELALAOUI Sidi-Yassir	Pharmacie Galénique
Pr. EL ANNAZ Hicham *	Virologie
Pr. EL HASSANI Moulay EL Mehdi *	Gynécologie-obstétrique
Pr. EL HJOUJI Aabderrahman *	Chirurgie Générale
Pr. EL KAOUI Hakim *	Chirurgie Générale
Pr. EL WALI Abderrahman *	Anesthésie-réanimation
Pr. EN-NAFAA Issam *	Radiologie
Pr. HAMAMA Jalal *	Stomatologie et Chirurgie Maxillo-faciale
Pr. HEMMAOUI Bouchaib *	O.R.L
Pr. HJIRA Naoufal *	Dermatologie
Pr. JIRA Mohamed *	Médecine Interne
Pr. JNIENE Asmaa	Physiologie
Pr. LARAQUI Hicham *	Chirurgie Générale
Pr. MAHFOUD Tarik *	Oncologie Médicale
Pr. MEZIANE Mohammed *	Anesthésie-réanimation
Pr. MOUTAKI ALLAH Younes *	Chirurgie Cardio-vasculaire
Pr. MOUZARI Yassine *	Ophtalmologie
Pr. NAOUI Hafida *	Parasitologie-Mycologie
Pr. OBTEL Majdouline	Médecine préventive, santé publique et Hyg.
Pr. OURRAI Abdelhakim *	Pédiatrie
Pr. SAOUAB Rachida *	Radiologie
Pr. SBITTI Yassir *	Oncologie Médicale
Pr. ZADDOUG Omar *	Traumatologie Orthopédie
Pr. ZIDOUH Saad *	Anesthésie-réanimation

2 - ENSEIGNANTS-CHERCHEURS SCIENTIFIQUES

PROFESSEURS/Prs. HABILITES

Pr. ABOUDRAR Saadia	Physiologie
Pr. ALAMI OUHABI Naima	Biochimie-chimie
Pr. ALAOUI KATIM	Pharmacologie

Pr. ALAOUI SLIMANI Lalla Naïma	Histologie-Embryologie
Pr. ANSAR M'hammed	Chimie Organique et Pharmacie Chimique
Pr .BARKIYOU Malika	Histologie-Embryologie
Pr. BOUHOUCHE Ahmed	Génétique Humaine
Pr. BOUKLOUZE Abdelaziz	Applications Pharmaceutiques
Pr. CHAHED OUAZZANI Lalla Chadia	Biochimie-chimie
Pr. DAKKA Taoufiq	Physiologie
Pr. FAOUZI Moulay El Abbes	Pharmacologie
Pr. IBRAHIMI Azeddine	Biologie moléculaire/Biotechnologie
Pr. KHANFRI Jamal Eddine	Biologie
Pr. OULAD BOUYAHYA IDRISSI Med	Chimie Organique
Pr. REDHA Ahlam	Chimie
Pr. TOUATI Driss	Pharmacognosie
Pr. YAGOUBI Maamar	Environnement, Eau et Hygiène
Pr. ZAHIDI Ahmed	Pharmacologie

Updated en 11/06/2020
KHALED Abdellah
Head of the human resources department
FMPR

Dedications

To

HIS LATE MAJESTY KING HASSAN II

May ALLAH have his soul in his Holy Mercy.

To

HIS MAJESTY THE KING MOHAMED VI

Supreme Chief and Chief of the state-major

May ALLAH glorify him and preserve His Kingdom.

To

HIS ROYAL HIGHNESS

CROWN PRINCE MOULAY EL HASSAN

May ALLAH protect him.

To

HIS ROYAL HIGHNESS
PRINCE MOULAY RACHID

May ALLAH protect him.

To

THE WHOLE ROYAL FAMILY

To

Mr. the General of the army-corps

Abdelfatah LOUARAK

General Inspector of the FAR and Commander of the South Zone

As a token of our great respect

Our deepest consideration and sincere admiration

To

Mr. Brigadier General Doctor

Mohammed ABBAR

Professor of Urology.

Inspector of the Health Service of the Royal Armed Forces.

As a token of our great respect,
And our deepest consideration

To

Mr. Doctor Colonel Major El Mehdi ZBIR.
Professor of Cardiology Director of HMIMV -Rabat.

As a token of our great respect

And our deepest consideration

To

Mr. Brigadier General Doctor
Abdelatif BOULAHYA
Professor of Cardiovascular Surgery Director of the Avicenne Military Hospital of Marrakech

As a token of our great respect
And our deepest consideration

To
Mr. Doctor Colonel Major Mohammed EL BAAJ
Professor of Internal Medicine, Director of the HMMI-Meknes.
As a token of our great respect
And our deepest consideration

To
Mr Doctor Colonel AMEZIANE Taoufiq
Professor of Internal Medicine
Director of the R.S.M.M.S
As a token of our great respect
And our deepest consideration
To
Mr. Doctor Colonel Abderrahmane ELMATAR Commander of the training and instruction group R.S.M.M.S
As a token of our great respect
And our deepest consideration

Special thanks

This work marks the end of an era, and the beginning of a hopefully better and brighter one.

I would like to thank the members of my small family; my mom Tourya, my dad Mostapha, and my little brother Zakaria who have always been there for me, and supported me greatly.

I would also like to thank a special group of my friends; the king of juicing, Rbiai, Sakri, Imzil, Ennaimi, Biyat, Zouhri, Khairi, Bijbij, Anouar, Adil, Salmane, Naoufal, Ilias, the godfather achraf, sgt Burdett, Fanoré, and the list goes on for sharing with me some of my best and worse memories and experiences during these last eight years of medical studies.

I want to also thank all of my teachers and friends from Misbah, Bismillah, and my entire medical class of 2012, and I want to share my deep appreciation and gratitude to Professor Abad sheriff el Assri and professor Miloud Gazzaz for providing me with this wonderful opportunity and support to make this humble peace of work possible.

Special thanks to one of my mightiest rivals of all time, you know who you are, without you I would've probably became strong anyways so don't flatter yourself too much :) but thanks for the little push, it has been a pleasure and an honor.

Abbreviations

LTL: left temporal lobe
RTL: right temporal lobe
TP53: tumor protein 53
NF1/2: neurofibromatosis 1/2
DNA: deoxyribonucleic acid
APC: argon plasma coagulation’
EGFR: epidermal growth factor receptor
TERT: telomerase reverse transcriptase
RTEL1: regulator of telomere length 1
T-reg cells: regulatory T cells
CMV : cytomegalovirus
TPO junction: temporo-parieto-occipital junction
MTG: middle temporal gyrus
STG: superior temporal gyrus
NSPC: neural stem and progenitor cell
GB: glioblastoma
SLF: superior longitudinal fasciculus
ILF: inferior longitudinal fasciculus
GBM: glioblastoma multiform
NADPH: nicotinamide adenine dinucleotide phosphate (reduced form)
MGMT: methylguanine methyltransferase
PCR: polymerase chain reaction
GFAP: glial fibrillary acidic protein

NOS: not otherwise specified
CT: computerized tomography
MRI: magnetic resonance imaging
fMRI: functional magnetic resonance imaging
ADC maps: apparent diffusion coefficient
FA maps: fractional anisotropy
DWI: diffusion-weighted imaging
VEGF: vascular endothelial growth factor
CRF: Corticotropin-releasing factor
DTI: diffusion tensor imaging
IFOF: inferior fronto occipital fasciculus
OD: oligodendroglioma
AED: antiepileptic drugs
MdLF: the middle longitudinal fascicle
GPS: global positioning system
iUS: intraoperative ultrasound
iMRI: intraoperative magnetic resonance imaging
PS: performance status
Gy: the gray: unit of absorbed dose of ionizing radiation
RT: radiotherapy

SUMMARY

INTRODUCTION

Gliomas comprise a heterogeneous group of neoplasms that differ in location within the central nervous system, in age and sex distribution, in growth potential, in extent of invasiveness, in morphological features, in tendency for progression, and in response to treatments. High grade Gliomas are by far the most common glial tumors and it represent over 60% of primary central nervous malignant tumors. In adults, It occur most often in the cerebral hemispheres, with the temporal lobe being the second most commonly affected region right behind the frontal lobe. The temporal lobe glioma is particularly interesting due to its unique accessible location, which may provide more valuable information about risk factors, anatomical relationships and the efficiency of the multidisciplinary treatments and approaches.

Indeed, we focus our study on the temporal lobe tumor because it seems to offer a good ability for extend tumor resection, without impairing a patient's functionality in the non-dominant side, compared to other central nervous parts.

We discuss the epidemiology, histology and imaging's advances for central nervous malignant gliomas, and specifically in the temporal lobe location which provide a better understanding of the extent of the lesion as well as its relationship with critical neuroanatomic function. We focus on the evolution of intraoperative imaging, functional brain mapping, and technology and how it participates to identify tumor from brain and how it has significantly improved the ability of surgeons for safer and more aggressive tumor removal.

In this study, we present some illustrative cases of temporal lobe malignant gliomas that were treated in our department. Therefore, we review the more recent literature's update in managing high grade gliomas in this location in the aim of addressing all the aspects of patient evaluation and care.

This study will be informative for surgeons, oncologists, neurologists, residents, and students who treat these patients and those who are training for a career in managing patients with these challenging tumors in such location.

EPIDEMIOLOGY

Incidence and prevalence[1]

The incidence of gliomas has increased worldwide since the late 1970s but it remains relatively low with 5 per 100,000. There are several possible causes for this increase, including improved diagnostic methods, such as modern radiologic imaging, and better access to neurosurgical services. Incidental findings of brain neoplasms increased with the introduction of CT and MRI technology in the 1980s. However, it has also been suggested that the overall increase in incidence is leveling off, whereas the increasing trend continues in the older age groups. There is no major geographical differences in the distribution of new diagnoses of glioblastoma.

.

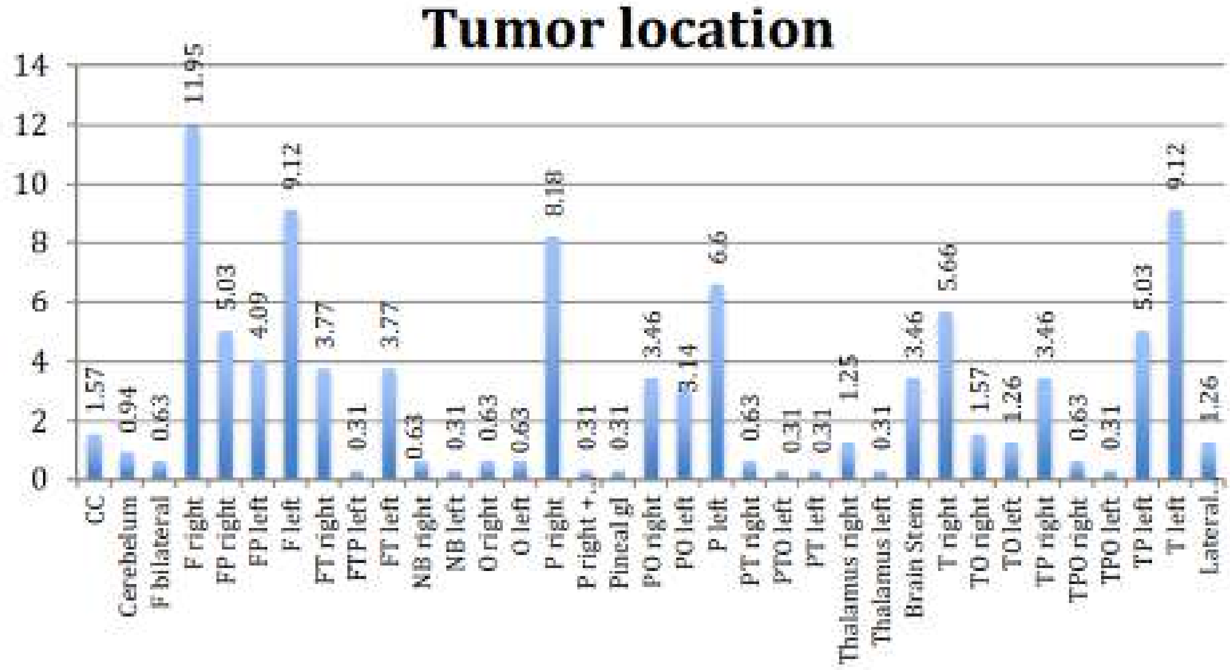


Figure1: Incidence by tumors location for high grade gliomas

A study conducted by Florian and associates in Romania showed that the most frequent locations of the high-grade gliomas were in the cerebral lobes, especially the right and the left frontal lobes, followed by the left temporal for single lobe location. For multiple lobe locations the left fronto-parietal and left temporo-parietal were the most frequent locations[2].

histologic type	Incidence (/100.000)
Glioblastoma	2.0
Diffuse astrocytoma	0.7
Anaplastic astrocytoma	0.5
Pilocytic astrocytoma	0.3
All other astrocytomas	0.03
Oligodendroglioma	0.5
Mixed glioma	0.5

Table1: Number and incidence of gliomas by histologic type[3]

In a study conducted in Finland by larjavaara and associates; a total of 331 incident gliomas were diagnosed during our study period. The majority were glioblastomas followed by Diffuse and anaplastic astrocytomas, pilocytic astrocytomas and Oligodendroglial tumors.

Temporal lobe region	% in LTL	% in RTL
Anterior	17.8	20.0
Posterior	30.1	23.3
Medial	37.0	46.7
Multi	15.1	10.0

Table2: comparison of percentage of gliomas between the regions of the right and the left temporal lobe[4].

a study conducted by Noll and associates in New York on 103 patients diagnosed between the years 2001 and 2010 with temporal lobe glioma showed a higher percentage of gliomas in the medial region of both the right and the left temporal lobe followed by the posterior and the anterior regions.

Age[1]

Glioblastoma is present at all ages, but the incidence is higher in the 5th and 6th decades of life. Age-adjusted annual incidence rates of glioblastoma peak in patients aged 75–84.

Sex ratio[1]

Males are affected 1.6 times more than females.

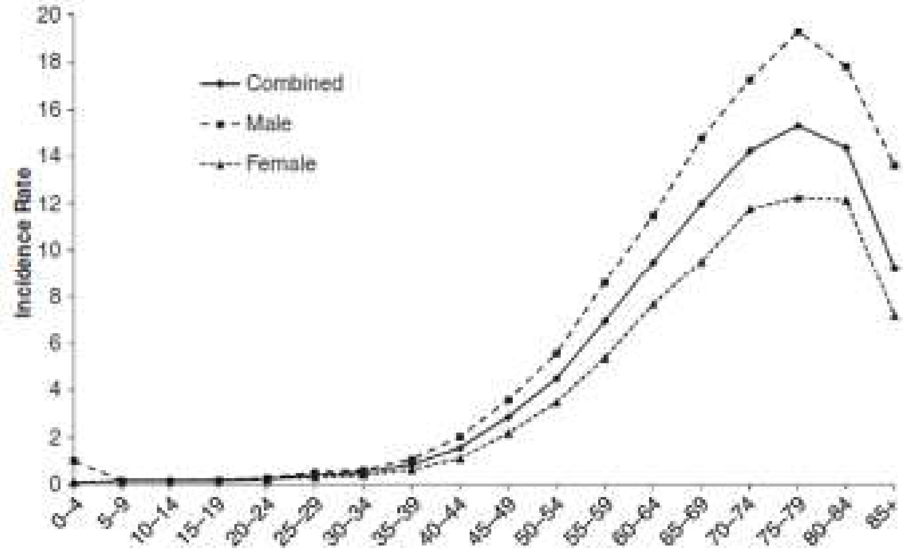


Figure2: age-adjusted and age-specific incidence rates for glioblastoma at diagnosis and gender between 2006–2010[5]

A study done in the United States over the course of 4 years (2006-2010) studying incidence rates per 100.000 showed that glioblastomas are interestingly frequent in males over females and concerns mostly the population over the age of 74 in both males and females while being fairly rare amongst younger population.

Risk factors

Endogenous Risk Factors[1]

Risk factors contributing to the malignant transformation of these cells remain elusive. Endogenous risk factors for glioblastoma other than age is observed in less than 1% of glioblastomas that arise in patients with hereditary cancer syndromes, like the Li-Fraumeni syndrome usually caused by mutations in the tumor suppressor gene TP53, neurofibromatosis both types I and II caused respectively by mutations in the NF1 and NF2 genes, or the Turcot syndrome resulting from mutations in the DNA repair gene APC.

These Glioblastomas in mostly young patients are usually preceded by the diagnosis of World Health Organization grade II or III.

Families with more than one member affected by glioblastoma is an exceptional situation, thus challenging the searching process for susceptibility gene loci.

Although a risk for glioma has been reported to be doubled in first-degree relatives of affected patients, the familial risk association is extremely low in consideration of the low overall glioma risk. As a result, no high penetrance gene variants linked with glioma risk have been identified among family members.

Population-based genome-wide association studies pinpointed five single nucleotide polymorphism risk alleles for gliomagenesis in the following four genes: TP53, EGFR(Epidermal growth factor receptor), TERT, and RTEL1.

telomerase-associated pathomechanism is associated with gliomagenesis in elderly patients. The low penetrance of risk alleles suggests that the pathophysiology of gliomas follows polygenic patterns and that in there turn follow distinct sequences of evolution.

Atopic disease is associated with reduced risk for gliomas by roughly 40%. The physiologic function of T-regs is to limit T-cell responses in order to prevent autoimmunity, and lower T-reg levels are observed in patients with atopic disease. In comparison, higher frequencies of T-regs decrease survival and contribute to the immunosuppressive microenvironment that stops immunologic antitumor responses.

Exogenous Risk Factors

Exogenous risk factors for the development of glioblastomas are very debatable and controversial. No association with exposure to cancerogenic agents or smoking has been reported[1].

Electromagnetic radiation: [6] [7][8] [9]

A link between gliomas and electromagnetic radiation from cell phones has not been conclusively proven. It was considered possible though several large studies have found no conclusive evidence, as summarized by the NIH's National Cancer Institute review of the topic .

However, further research is still being pursued to obtain more concrete evidence and verify that there is no relationship.

ionizing irradiation: [1]

Risk associations of gliomas with ionizing irradiation have been studied, but data on the more specific causality relationship with glioblastoma have not been reported.

Infection with cytomegalovirus[1] [10][11][12]

Viral oncogenesis role for cytomegalovirus in gliomagenesis have been postulated, because some of the CMV gene products interact with glioblastoma core signaling pathways, but experimental evidence to confirm this role for CMV is scarce.

However, most glioblastomas are infected with cytomegalovirus, which speeds the development of tumors.

ANATOMY OF THE TEMPORAL LOBE

Anatomy of the surface:

The **temporal lobe** is one of the six lobes of the brain that largely occupies the middle cranial fossa. it is the second largest lobe, after the larger frontal lobe, accounting 22% of the total neocortical volume. The lobe extends superiorly to the Sylvian fissure, and posteriorly to the lateral parietotemporal line, which separates the temporal lobe from the inferior parietal lobule of the occipital lobe inferiorly and the parietal lobe superiorly. The middle cranial fossa forms its anterior and inferior boundaries[13].

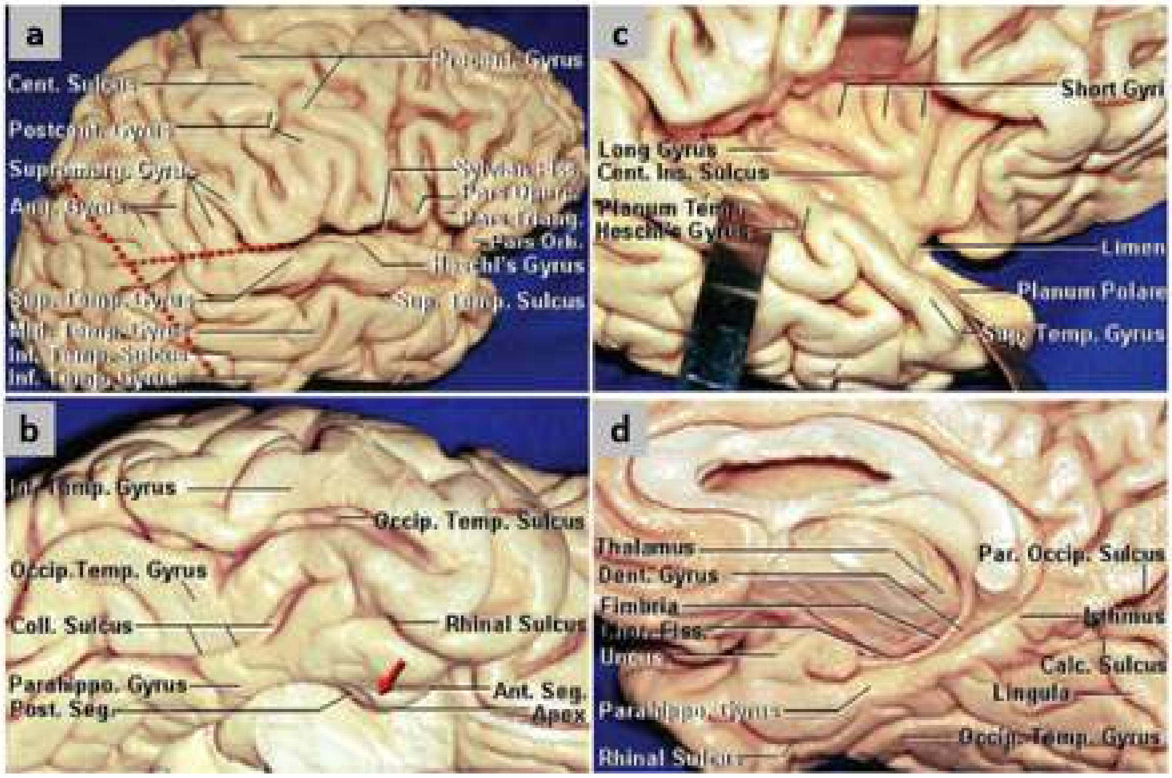


Figure 3: Anatomical landmarks of the cortex of the temporal lobe. Photographs are of the lateral (a), inferior (b), superior (c) surfaces and medial (d) on fixed cadaver[13].

Lateral surface[14]

The lateral temporal surface is made of three parallel gyri, the superior, middle, and inferior temporal gyri, divided by two sulci, the inferior and superior temporal sulci. Both the gyri and sulci parallel the sylvian fissure.

The superior temporal gyrus is located between the sylvian fissure and the superior temporal sulcus and is continuous around the lip of the fissure with the transverse temporal gyri, which extend obliquely backwards and medially toward the posterosuperior angle of the insula forming the lower wall of the posterior part of the floor of the sylvian fissure. **The middle temporal gyrus** is located between the superior and inferior temporal sulci. the ambient, the temporal horn and the crural cisterns are positioned deep to the middle temporal gyrus. **The inferior temporal gyrus** lies under the inferior temporal sulcus and continues around the inferior border of the hemisphere to form the lateral side of the basal surface. The angular gyrus, a parietal lobe structure, caps the upturned posterior end of the superior temporal sulcus. The inferior temporal gyrus is most of the time made of multiple fragmented gyri and may blend into the middle temporal gyrus without a clear sulcal demarcation.

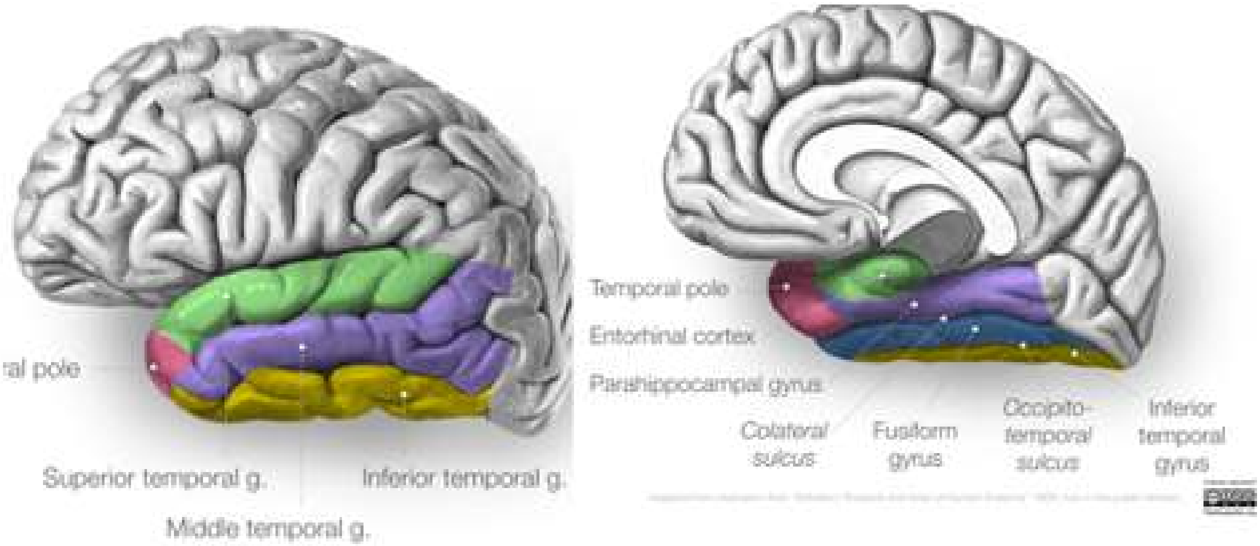


Figure 4: Lateral view of the left temporal lobe showing boundaries and limits with different gyrus and sulcus.

Medial surface[13]

The medial surface of the temporal lobe is very complex. It is formed predominantly by the rounded medial surfaces of the parahippocampal gyrus and uncus. This medial surface is the combination of three longitudinal strips of neural tissue, one located above the other, which are interlocked with the hippocampal formation. **The most inferior strip** is formed by the rounded medial edge of the parahippocampal gyrus, the site of the subicular zones; the middle strip is formed by the dentate gyrus, located on the medial surface of the hippocampal formation; and **the superior strip** is formed by the fimbria of the fornix, a white band comprised of the fibers emanating from the hippocampal formation and posteriorly directed into the crus of the fornix. **The parahippocampal and dentate gyri** are separated by the hippocampal sulcus, and the dentate gyrus and the fimbria are separated by the fimbriodentate sulcus. The amygdala and the hippocampal formation lie just beneath and are so intimately related to the mesial temporal cortex that they are considered in this section. The dentate gyrus blends posteriorly behind the splenium into the fasciolar gyrus, which is continuous with the indusium griseum.

The parahippocampal gyrus deviates medially at the site of the uncus that projects medially above the tentorial edge. The parahippocampal gyrus also extends around the lower border to form the medial part of the basal surface of the temporal lobe, where it is separated from the medially projecting uncus by the rhinal sulcus. Posteriorly, the part of the parahippocampal gyrus below the splenium of the corpus callosum is intersected by the anterior end of the calcarine sulcus, which divides the posterior portion of the parahippocampal gyrus into an upper part that is continuous above and posteriorly with the isthmus of the cingulate gyrus and continuous below and posteriorly with the lingual gyrus.

The uncus, the medially projecting anterior part of the parahippocampal gyrus has an angular shape with anterior and posterior segments that meet at a medially directed apex. The medial face of the anterior segment faces the proximal part of the sylvian, the carotid cistern, and the internal carotid and proximal middle cerebral arteries. The posterior segment faces the cerebral peduncle and, with the peduncle, forms the lateral and medial walls of the crural cistern through which the posterior cerebral, anterior choroidal, and medial posterior choroidal arteries pass.

The optic tract passes above the medial edge of the posterior segment in the roof of the crural cistern. The amygdaloid nucleus forms almost all of the interior and comes to the medial surface of the upper part of the anterior segment. The upper part of the posterior segment is formed largely by the medial aspect of the head of the hippocampus.

The apex, where the anterior and posterior segments meet, points medially toward the oculomotor nerve and posterior communicating artery. The head of the hippocampus reaches the medial surface in the upper part of the posterior segment at the anterior end of the dentate gyrus. Within the ventricle, a small medially projecting space, the uncal recess, situated between the ventricular surface of the amygdala and hippocampal head, is located lateral to the uncal apex.

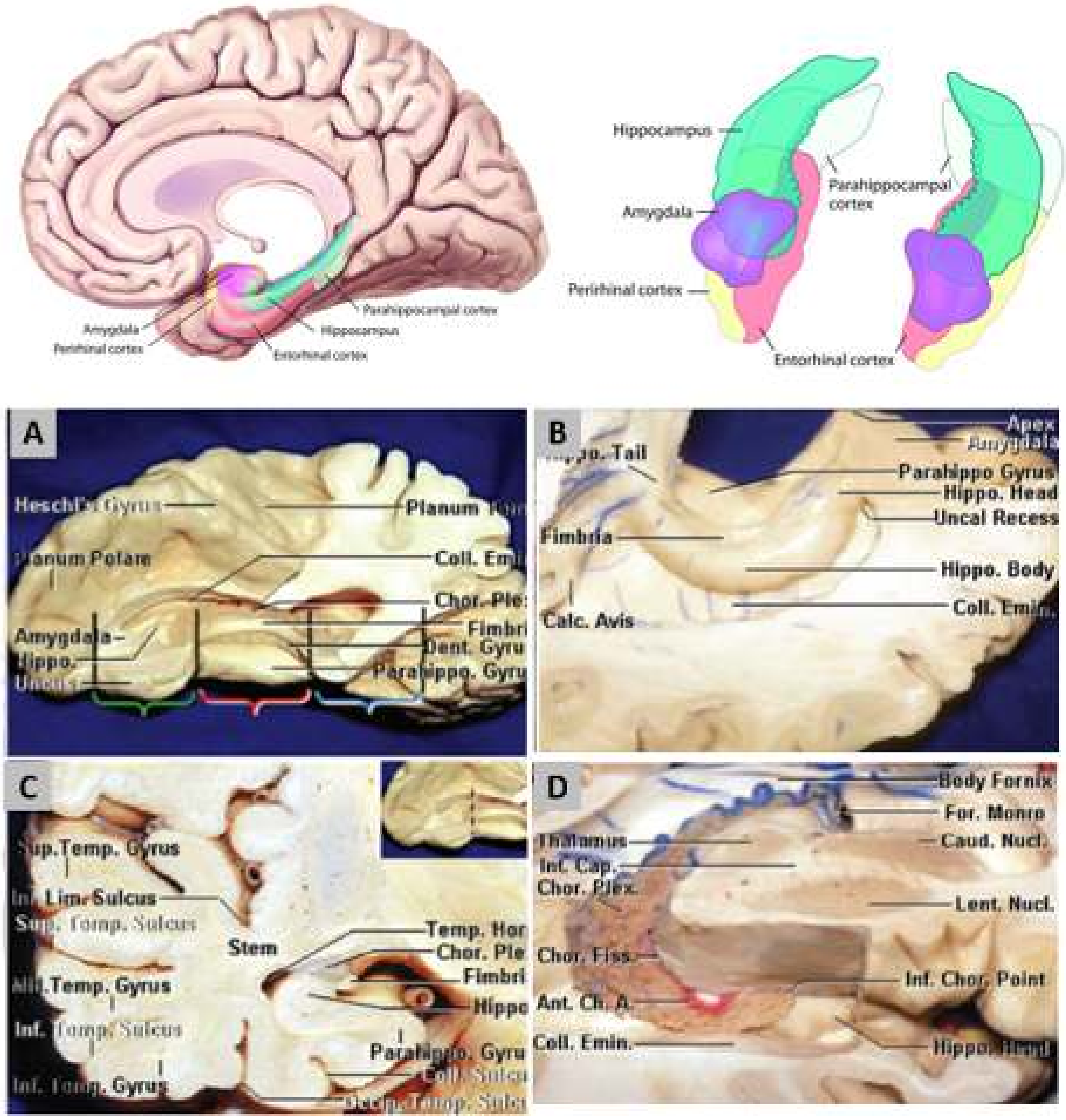
The inferior choroidal point, the lower end of the choroidal fissure along which the choroid plexus is attached, is located just behind the upper edge of the posterior uncal segment, immediately behind the head of the hippocampus, at the site where the anterior choroidal artery passes through the choroidal fissure to enter the temporal horn. The anterior choroidal artery arises near the midlevel of the anterior segment and hugs its surface, sloping gently upward, unless extremely tortuous. It continues to ascend as it courses posteriorly around the uncal apex and reaches the upper part of the posterior segment, where it passes through the fissure at the inferior choroidal point. The dentate gyrus, named for its characteristic tooth-like elevations, extends posteriorly from the upper part of the posterior segment and has the most prominent denticulations anteriorly.

The amygdala can be considered as being entirely located within the boundaries of the uncus. It forms the anterior wall of the temporal horn. Superiorly, the amygdale blends into the claustrum and Globus pallidus without any clear demarcation. The upper posterior portion of the amygdala tilts back above the hippocampal head and the uncal recess to form the anterior portion of the roof of the temporal horn. Medially, it is related to the anterior and posterior segments of the uncus. The amygdala gives rise to the stria terminalis, which courses between the thalamus and caudate nucleus deep to the thalamostriate vein.

The hippocampus, which blends into and forms the upper part of the posterior uncal segment, is a curved elevation, approximately 5 cm long, in the medial part of the entire length of the floor of the temporal horn. It has the dentate gyrus along its medial edge and a curved collection of gray matter in its interior that is referred to as Ammon’s horn. It sits above and is continuous below with the rounded medial surface of the parahippocampal gyrus referred to as the subicular surface. **Ammon’s horn** is characterized in transverse sections of the hippocampal formation by its reversed C- or comma-shaped orientation and by its tightly packed pyramidal cell layer.

The hippocampus is divided into three parts: head, body, and tail. The head of the hippocampus, the anterior and largest part, is directed anteriorly and medially, and forms the upper part of the posterior uncal segment. Superiorly, the head of the hippocampus faces the posterior portion of the amygdala. Anterior to the hippocampal head is the uncal recess, a cleft, located between the head of the hippocampus and the amygdala. The body of the hippocampus extends along the medial part of the floor of the temporal horn, narrowing into the tail that disappears as a ventricular structure at the anterior margin of the calcar avis.

The fimbria of the fornix arises on the ventricular surface of the hippocampus behind the head and just behind the choroidal fissure. The temporal horn extends into the medial part of the temporal lobe to just anterior to the hippocampal head and to just behind the amygdala. The temporal horn ends approximately 2.5 cm from the temporal pole. The inferior choroidal point, at the lower end of the choroidal fissure, is located just behind the head of the hippocampus and immediately lateral to the lateral geniculate body.



<u>Figure 5: Schematic and cadaveric specimen showing the medial part of the temporal lobe and its relationship to the adjacent structures.</u>

The superior surface, along with the insula, was exposed by removal of parts of the frontal and parietal lobes above the lateral sulcus (A). The hippocampus, dentate gyrus and fimbria as they appear after removal of the roof of the temporal horn of the lateral ventricle and of the choroid plexus.

Basal surface[13]

The basal surfaces of the temporal and occipital lobes are formed by the same gyri that continue from anterior to posterior across their uninterrupted border. They are traversed longitudinally by the longer collateral and occipitotemporal sulci and the shorter rhinal sulcus that divide the region from medial to lateral into the parahippocampal and occipitotemporal gyri and the lower surface of the inferior temporal gyrus. The basal surface of the parahippocampal gyrus forms the medial part of the inferior surface.

It extends backward from the temporal pole to the posterior margin of the corpus callosum. Its anterior end projects medially to form the uncus. It is continuous anteriorly with the uncus without a limiting border and continues posteriorly to blend into the isthmus of the cingulate gyrus and lingula.

The collateral sulcus, one of the most constant cerebral sulci, begins near the occipital pole and extends anteriorly, parallel and lateral to the calcarine sulcus. Posteriorly, it separates the lingula and occipitotemporal gyrus, and anteriorly, it courses between the parahippocampal and the occipitotemporal gyri.

The collateral sulcus may or may not be continuous anteriorly with the rhinal sulcus, the short sulcus extending along the lateral edge of the uncus. The collateral sulcus is located below the temporal horn and indents deeply into the basal surface producing a prominence, the collateral eminence, in the floor of the temporal horn on the lateral side of the hippocampus.

Posteriorly, in the area below the atrial floor, the collateral sulcus also indents deeply to produce a prominence in the triangular atrial floor called the collateral trigone. The temporal horn can be exposed from below by opening through the deep end of the collateral sulcus. The occipitotemporal sulcus courses parallel and lateral to the collateral sulcus and separates the occipitotemporal sulcus and basal surface of the inferior temporal gyrus.

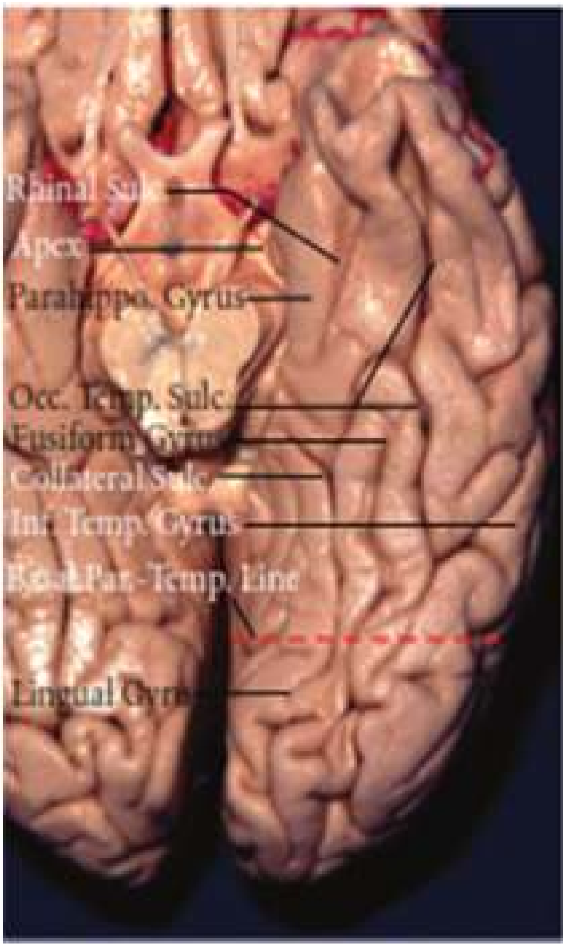


Figure 6: Inferior view of the left temporal lobe.

The basal surface the temporal lobe consists of, from lateral to medial, the inferior margin of the inferior temporal gyrus, the fusiform gyrus, and the parahippocampal gyrus. The fusiform gyrus is separated laterally from the inferior temporal gyrus by the occipitotemporal sulcus, and medially from the parahippocampal gyrus by the collateral posteriorly and rhinal sulci anteriorly, which are not continuous in every case. The basal parietotemporal line connecting the preoccipital notch and inferior end of parietooccipital sulcus separates the temporal and occipital lobes at the basal surface.

White Matter[15][16][17][18][19][20][21]:

Subcortical white matter comprises three populations of axons. **Association fibers** connect cortical areas within the same cerebral hemisphere. **Commissural fibers** connect mainly but not exclusively symmetrical cortical areas. **Projection fibers** connect cortical areas with subcortical nuclei of grey matter. The three types of fiber intersect extensively, but certain bundles can be demonstrated by dissection. The same bundles also can be imaged in the living brain by diffusion tensor imaging, a nuclear magnetic resonance technique.

The temporal cortex is connected by association fibers with all the other lobes of the forebrain.

The superior longitudinal fasciculus:

The largest named bundle is the arcuate fasciculus, whose anterior end is in the frontal lobe. The arcuate fasciculus passes above the insula and lentiform nucleus, where it is also named the superior longitudinal fasciculus and follows a curved course into the temporal lobe, thus providing two-way communication between frontal cortex, including Broca's expressive speech area, and Wernicke's receptive language area in the posterior part of the superior temporal gyrus. The condition of conduction aphasia is traditionally attributed to a destructive lesion that interrupts the arcuate fasciculus.

The uncinate fasciculus:

Another frontotemporal association bundle is the uncinate fasciculus, named for its hook-like shape, which passes around the stem of the lateral sulcus and connects the cortex of the temporal pole with the prefrontal cortex. The ventral amygdalofugal pathway is more dorsally and posteriorly located, above the anterior perforated substance.

Visual association cortex extends from the occipital lobe to the middle and inferior temporal and fusiform gyri.

The inferior longitudinal fasciculus:

is in the white matter inferolateral to the temporal horn connects these visual areas with one another and with the temporal polar cortex, an important source of fibers afferent to the amygdala.

The fornix and stria terminalis:

Already discussed in connection with the hippocampal formation and amygdala, respectively, can also be considered association fasciculi.

The corpus callosum:

The largest group of commissural fibers is the corpus callosum. Degeneration studies indicate that axons from the middle and posterior parts of the temporal cortex cross the midline in the central part of the body of the corpus callosum. The temporal poles, transverse temporal gyri, and amygdalae may be interconnected mainly by fibers of the anterior commissure.

Projection fibers afferent to the temporal cortex:

It includes those from the medial geniculate body to the primary auditory area of the transverse temporal gyri. These travel in the sublenticular limb of the internal capsule, where they probably are accompanied by fibers from the medioventral thalamic nucleus, which is connected with the amygdala, hypothalamus, hippocampal formation, and parahippocampal gyrus. For much of the temporal cortex, the sources of thalamic afferents have not been determined. All thalamocortical projections are accompanied by reciprocal corticothalamic fibers.

The thalamocortical projections:

An important thalamocortical pathway that passes through the temporal lobe is Meyer's loop of the geniculocalcarine tract, which is drawn into the anterior temporal white matter with the growth of the nearby temporal horn of the lateral ventricle. This loop carries signals derived from the upper quadrants of the contralateral visual fields to the corresponding primary visual cortex of the anterior half of the inferior bank of the calcarine sulcus.

Some efferent temporal cortical projection fibers go to the amygdala and hippocampus and are thus confined to the temporal lobe.

The temporopontine or temporoparietopontine tract:

Most textbooks of neuroanatomy show a large temporopontine or temporoparietopontine tract occupying the lateral quarter of the basis pedunculi in the midbrain. Degeneration studies following temporal lobe lesions in monkeys, however, show only a small temporopontine projection, originating in the superior temporal gyrus and ending in the most lateral of the pontine nuclei. In the absence of comparable information for the human brain, we must guess that the projection is similar to that of the monkey, and that the temporal cortex does not have a large, direct influence on the workings of the cerebellum.

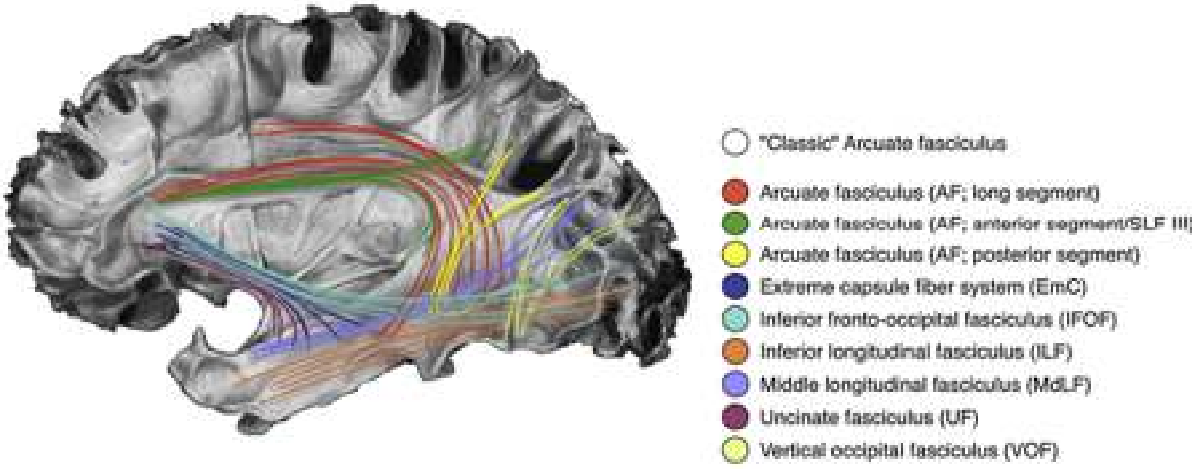


Figure 7: lateral view of the cerebrum, after dissection of the perisylvian region, showing a schematic representation of the Arcuate fasciculus and its relationship with the other white matter tracts.

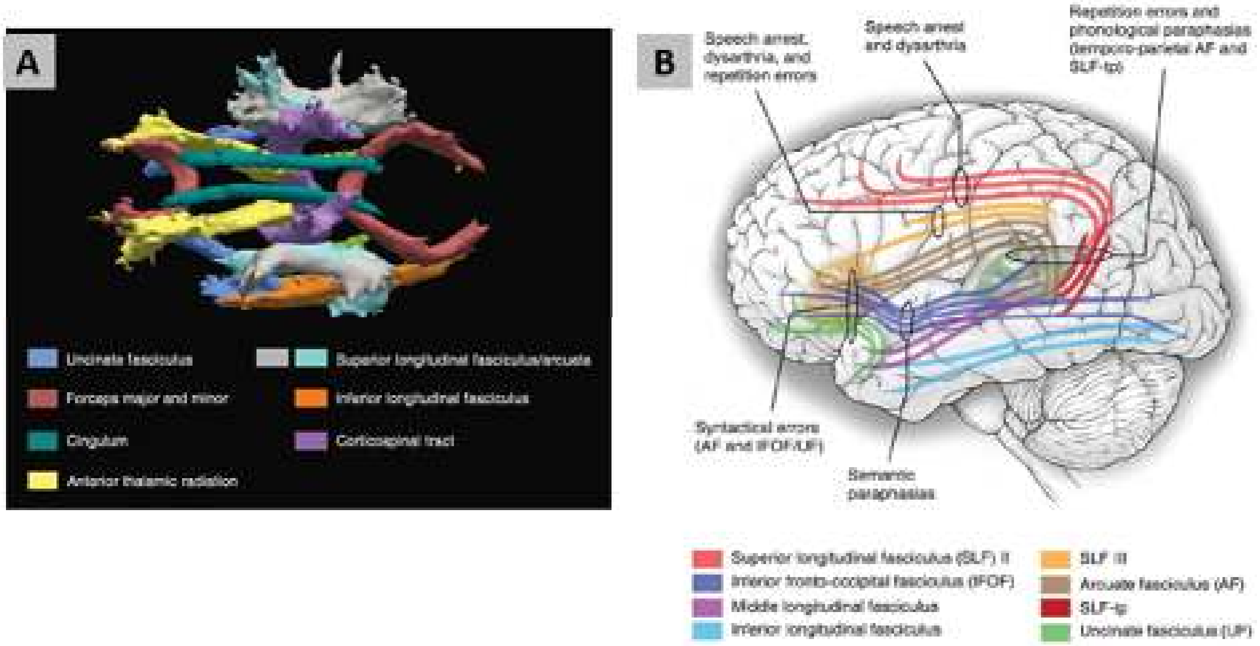


Figure 8: schematic reconstruction of the main White matter tracts, with superior (A) and Lateral (B) views, based on reported findings of decreased fractional anisotropy on diffusion tensor imaging.

Important Distinctions in Temporal Lobe Glioma Anatomy[22]:

Misinterpreting the exact anatomy of a glioma in the temporal lobe is one of the most common pitfalls in failed surgical efforts in glioma surgery. Specific areas of the temporal lobe can be greatly enlarged and distorted which can give the false impression that there is tumor in a part of the brain where there is not. Failure to persist in your resection until validated by the identification of clear anatomic boundaries is a common reason why a large amount of tumor is missed in some cases.

A common mistake is to misjudge the exact position of the Sylvian fissure in cases expanding the lateral temporal lobe. The fissure can be pushed upward by pure tumor bulk or the superior temporal gyrus can be distorted by tumor involvement. Careful attention to this detail, and observation of the position of the superficial middle cerebral vein can combat this common issue.

Involvement of the fusiform gyrus can be difficult to distinguish from hippocampal involvement, and they often occur together making this a moot point. However, fusiform gyrus tumors tend to extend preferentially posteriorly along the inferior longitudinal fasciculus, whereas hippocampal tumors follow the fornix or the cingulum.

Hippocampal gliomas are common and often misinterpreted. The hippocampus can be massive, and often compresses the descending motor fibers superiorly. A study of the coronal images is often helpful at determining the exact location of the deep structures. The tumor tends to extend along the fornix or parahippocampal gyrus or both. Some very enlarged fornices can give the untrained eye the impression that there is invasion of the thalamus or internal capsule, as seen in, but awareness of the tumor’s nature as a hippocampal glioma, and not a TPO junctional glioma makes this pattern more obvious.

Tumors at the TPO junction are far more complex. Because of the complex anatomy of the supramarginal gyrus, and the narrow depth of the insula at this posterior limit, it can be quite hard to tell which lobe these tumors arise from. This is discussed in more detail after discussing the insula and parietal lobe.

Much of the temporal lobe is well known to be removable without severe neurologic problems. The specific functions of the amygdala and hippocampus in memory and emotion are well known.

PHYSIOLOGY OF THE TEMPORAL LOBE

Visual memories[23]:

The temporal lobe communicates with the hippocampus and plays an important role in the formation of explicit long-term memory modulated by the amygdala.

Processing sensory input[23][24][25][26]:

Auditory

Adjacent areas in the superior, lateral, and posterior parts of the temporal lobes are involved in high-level auditory processing. The temporal lobe is involved in hearing, and holds the primary auditory cortex. The primary auditory cortex receives sensory information from the ears and secondary areas process the info into meaningful units like words and speech. The superior temporal gyrus includes an area where auditory signals from the cochlea reach the cerebral cortex first and then, are processed by the primary auditory cortex in the left temporal lobe.

Visual

The areas correlated with vision in the temporal lobe interpret the meaning of visual stimuli and establish object recognition. The ventral part of the temporal cortices appear to be involved in high-level visual processing of complex stimuli such as scenes (parahippocampal gyrus)and faces (fusiform gyrus).Anterior parts of this ventral stream for visual processing are involved in object recognition and perception.

Language recognition

The temporal lobe holds the primary auditory cortex, which is crucial for the processing of semantics in both vision and speech. Wernicke's area, which spans the region between parietal and temporal lobes, plays an important role in speech comprehension. The functions of the left temporal lobe are not limited to low-level perception but also include naming, verbal memory , and comprehension.

new memories

The medial temporal lobes are thought to be involved in encoding declarative memory. The medial temporal lobes include the hippocampi, which are essential for memory storage, therefore damage to this area can result in impairment in new memory formation leading to permanent or temporary anterograde amnesia.

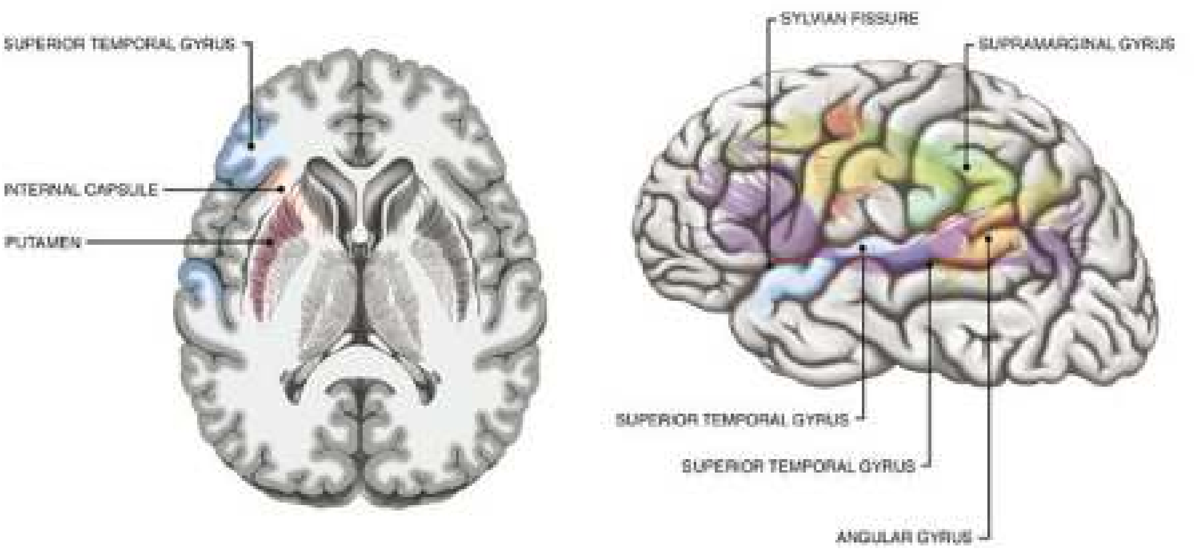


Figure 9: Schematic demonstrating the anatomic relationships of various structures within the temporo-parieto-occipital (TPO) junction.

the lateral surface of the posterior temporal lobe is like a semantic network. Semantic memory is a collection of known informations about the world, such as, the function of tools, the meaning of nonverbal expressions, the name of objects, and on the right side, probably the visual appearance of various objects as necessary to define one object as different from the other. Thus, the semantic network is as a library of varying extent which roughly extends outward from Heschl's gyrus, involving the MTG, the STG, and in most people into the supramarginal gyrus, and even the angular gyrus. This library which may cover a 1– 3 discrete areas of the superior temporal gyrus, or which may extend over much of the superior and middle temporal gyrus. Destruction of the entire semantic network, will cause problems with comprehension; however, anything short of this usually causes anomia, expressive speech problems or both. The idea that there is a small discrete area of cortex which can be damaged to cause pure receptive aphasia is a neurosurgical fantasy which will cause a wrong approach to brain mapping.

PATHOLOGY

Pathogenesis and topography[1][3]:

Glioblastoma originates from a cell that is thought to be a neural stem and progenitor cell. In adults, this type of cells have been identified in the hippocampi of the temporal lobes, in the subventricular zones lining the lateral ventricles and in the subcortical white matter.

In line with the hypothesis that NSPC are the cells from which originates gliomagenesis, the majority of adult gliomas arise in supratentorial brain areas, particularly the frontal (25.8%), temporal (19.7%), and parietal (12.2%) lobes.

The occurrence of bilateral gliomas was toward the frontal lobes, because most gliomas involving both hemispheres are bifrontal. Also, there is a more frequent involvement of the right hemisphere.

The non-uniform anatomic distribution of gliomas with frontal and temporal pre-dominance may reflect the participation of neurochemical, developmental, or functional factors in the pathogenesis of gliomas. It is also postulated that tumors in different parts of the brain arise from different precursor cells or that differences in the extracellular environment may account for the differences between lobes.

Location of glioma	Relative volume (cm^3)
Frontal lobe	3
Temporal lobe	2
Parietal lobe	2
Occipital lobe	1

Table 3: volume of tumor tissue depending on cerebral lobe

A study conducted in Finland concerning the incidence and the median volume of tumors in different lobes showed that the median volume of the tumor tends to be bigger in the frontal followed by the temporal lobe.

Macroscopic Features of GBs[27]:

Macroscopically GBs is heterogeneous, featuring cystic and gelatinous areas, multifocal hemorrhage and necrosis. A characteristic feature of GBs is the variation in gross appearance of the tumor from a region to another. Some of the regions as a result of tissue necrosis appear soft and yellow, whereas some of the tumor areas are firm and white and some regions show marked cystic degeneration and hemorrhage. The tumor is mostly represented by a single, relatively large, irregular lesion which usually arises in the white matter.

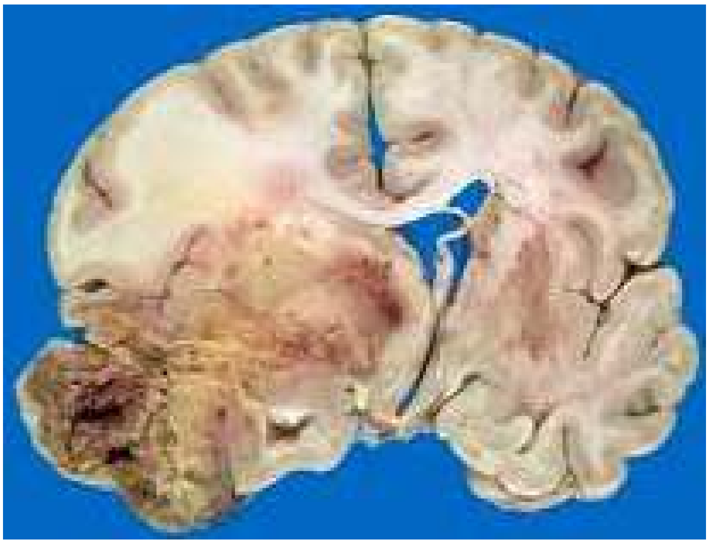


Figure 10: A coronal section of a post mortem brain harboring an infiltrative temporal malignant glioma which invade the insular and deep diencephalic structures[1].

Anatomic classification of Temporal lobe high grade gliomas[1]:

While there can be overlap of these subtypes, especially at more advanced stages of the disease, most temporal gliomas can be classified as one of six principle types.

a. Anterior: These tumors are the most common and are centered within the anterior temporal lobe without major invasion of the SLF. They can spread into the insula via the uncinate fasciculus or into the medial temporal structures.

b. Hippocampal: These are also common, and involve the medial hippocampal structures. Their preference is to follow the Papez circuitry and extend backwards along the fornix into the ventricle, or into the cingulum and isthmus. They can follow the diagonal band of Broca into the basal forebrain and contralateral amygdala, or the ventral amygdalofugal tract into the hypothalamus. They generally do not spread to the lateral system until late stages.

c. Inferior: There are uncommon and are based in the fusiform gyrus and preferentially spread along the ILF into the occipital lobe, though they may invade the medial structures as well.

d. Lateral: Thankfully, these tumors are relatively uncommon, but are usually bad news. They invade the lateral system, and preferentially follow the SLF. Many times, very little can be removed due to functional concerns. The hippocampus is generally spared (as we would expect with the lateral vs. medial tract distinction), and we generally try to leave the hippocampus in these cases if it is normal, as the recurrences do not commonly occur there.

e. Junctional: These are the worst of the bunch. They are fundamentally lateral temporal tumors, extend along the junctional anatomy of Heschl's and/or supramarginal gyrus, into the posterior insula and abut the internal capsule.

f. The right-sided ones can be resected with patients, but the left sided ones are often impossible to make much progress with if they are small and in the SLF or internal capsule. In essence, they combine all the bad parts of a lateral temporal case, with the risk of injuring the back of the internal capsule.

Microscopic features[1]:

Gliomas are the most common primary parenchymal central nervous system neoplasms. The classification, grading, and treatment of this group of tumors was essentially based on morphological criteria, which provided only suboptimal accuracy for the prediction of treatment outcomes. Glioma tumors are histologically separated into Grades I through IV according to the World Health Organization criteria. Grade I tumors typically have a good prognosis and more frequently occur in children, and Grade II tumors are characterized on histologic examination by hypercellularity. Grade III astrocytoma tumors (anaplastic astrocytoma tumors) are characterized on histologic examination according to hypercellularity, as well as nuclear atypia and mitotic figures. Grade IV gliomas, also known as GBMs, are characterized on histologic examination according to hypercellularity, nuclear atypia, mitotic figures, and evidence of angiogenesis and/or necrosis.

Molecular Profiles of Diffuse Astrocytic and Oligodendroglial Tumors[28] [29] [30] [31]:

Isocitrate Dehydrogenase (IDH)Mutations

The hallmark genetic modifications in diffuse gliomas are somatic mutations in the gene encoding human cytosolic NADPH-dependent isocitrate dehydrogenase 1 (IDH1). Less frequently involved are mutations of IDH2. The IDH1 enzyme normally catalyzes the oxidative carboxylation of isocitrate to alpha-ketoglutarate, resulting in the reduction of NADP to NADPH.

IDH mutations most likely represent an initiating event, but they are probably not sufficient to induce tumor growth on their own, instead they have to be accompanied by additional genetic. The combination of IDH1/p53/ATRX and IDH1/1p19q/TERT are respectively mutational signatures for astrocytic tumors and oligodendrogliomas.

Co-deletion of 1p/19q

1p/19q co-deletions has been estimated to be 80–90% in WHO grade II oligodendrogliomas and 50–70% in WHO grade III oligodendrogliomas. In spite of a strong association between 1p/19q loss and classic oligodendroglioma morphology, morphology alone cannot predict the co-deletion’s status. Although the genes on 1p/19q remain enigmatic, numerous correlations have been established demonstrating that many tumors with 1p/19q co-deletions also showcase

IDH1/IDH2 mutations; however, 1p/19q loss appears to be absent in cases with TP53mutations or EGFR amplifications.

Notably, the combined loss of 1p/19q is also found in mixed glial tumors (oligoastrocytomas), but extremely rare in non-glial malignancies.

MGMT Methylation Status

MGMT encoded by the gene at 10q26 is a suicide DNA repair enzyme that protects cells against damage from alkylating agents and ionizing radiation. It has become one of the most vastly studied molecular markers in neurooncology, because it has the potential to counteract the efficacy of chemotherapy with temozolomide.

MGMT methylation appears to be frequent in low grade and anaplastic gliomas (up to 90%), which show 1p/19q co-deletion. The MGMT status is mostly tested by methylation-specific PCR.

Role of Epidermal Growth Factor Receptor (EGFR) Pathway Aberrations

glioblastomas, have been found to upregulate several growth factors along with their receptors. The epidermal growth factor receptor gene at 7p12 has been described as the most frequently amplified gene in approximately 60% of glioblastomas. Further, about 50% of glioblastomas that overexpress wild-type EGFR also express EGFR mutant alleles, such as the EGFR variant III (EGFRvIII), which appears to be unique to glial cells.

The characterization of EGFR amplification in glioblastoma is based on the detection of double-minute chromosomes, which are small fragments of extrachromosomal DNA by fluorescent in situ hybridization.

The new classification of glioma WHO 2016[1]:

The uncovering of distinct genetic profiles for different glioma subtypes not only helped to improve the understanding of glioma pathogenesis, but also revealed that certain molecular changes are correlated to therapeutic response and prognosis.

The emergence of molecular signatures challenged the prognostic value of classic morphological grading. therefore, it became a major goal for contemporary glioma diagnostics to include molecular advances into routine tumor classification, which led to the International Society of Neuropathology Haarlem consensus guidelines and a revised World Health Organization classification for tumors of the central nervous system in 2016.

The classification is still largely based on histopathologic features centering around the 2007 World Health Organization classification. These cells resemble oligodendroglia and are called anaplastic if they have focal or diffuse characteristics which infer a worse prognosis.

The microscopic evaluation of gliomas begins the process of glioma classification and grading, according to 2016 World Health Organization standards. Including lineage-specific immunohistochemical stains such as GFAP. A second step involves molecular testing and biomarker detection for further subclassification and stratification. The results of both morphological and biomarker analysis are combined into a ‘final integrated diagnosis’. Certain mutational profiles appear to be mutually exclusive and define tumor lineages: The combinationIDH1/1p19q/TERT defines oligodendrogliomas, whereas the combination IDH1/p53/ATRX defines astrocytic tumors.

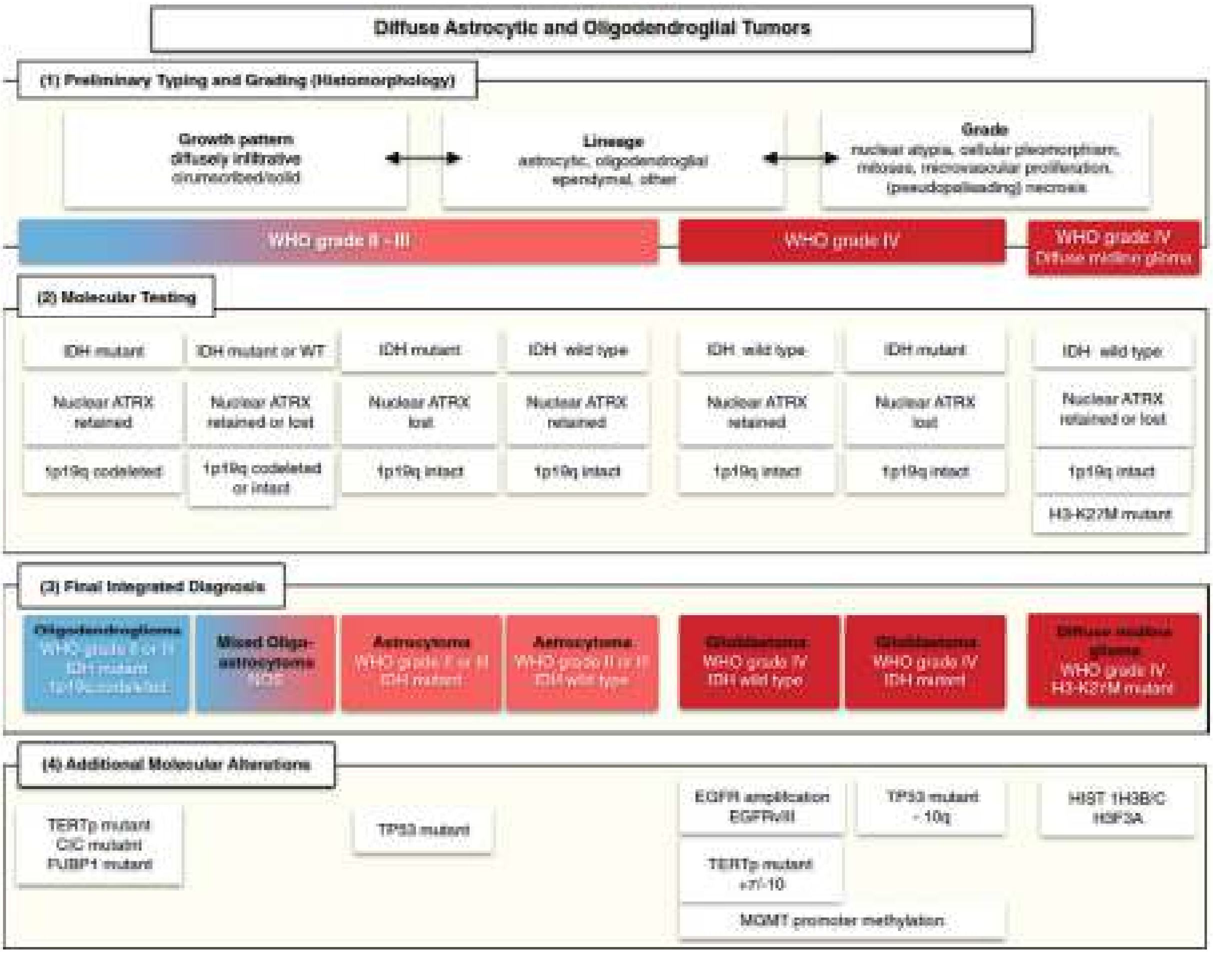


Figure 11: The inclusion of molecular markers led to important changes in the 2016 classification system of gliomas. In prior editions, astrocytic gliomas, oligodendrogliomas, and mixed oligoastrocytic gliomas each formed a separate group within the larger category of neuroepithelial neoplasms. this classification merges these gliomas into a single group: ‘Diffuse astrocytic and oligodendroglial tumors. Other than their infiltrative growth pattern, diffuse gliomas share frequent isocitrate dehydrogenase mutations, a hallmark genetic alteration, which plays an important role for the classification of gliomas.

Tumor entity/variant	WHO grade
Diffuse astrocytic and oligodendroglial tumors	
Diffuse astrocytic and oligodendroglial tumors Diffuse astrocytoma, IDH-mutant	II
Gemistocytic astrocytoma, IDH-mutant	II
Diffuse astrocytoma, IDH-wild type	II
Diffuse astrocytoma, NOS	II
Anaplastic astrocytoma, IDH-mutant	III
Anaplastic astrocytoma, IDH-wild type	III
Anaplastic astrocytoma, NOS	III
Glioblastoma, IDH-wild type	IV
Giant cell glioblastoma	IV
Gliosarcoma	IV
Epithelioid glioblastoma	IV
Glioblastoma, IDH-mutant	IV
Glioblastoma, NOS	IV
Diffuse midline glioma, H3K27M-mutant	IV
Oligodendroglioma, IDH-mutant and I1p/19q-co-deleted	II
Oligodendroglioma, NOS	II
Anaplastic oligodendroglioma, IDH-mutant and 1p/19q-co-deleted	III
Anaplastic oligodendroglioma, NOS	III
Oligoastrocytoma, NOS	II
Anaplastic oligoastrocytoma, NOS	III

Table 4: 2016 WHO Classification of Gliomas.

DIAGNOSIS

Clinical manifestations of temporal lobe gliomas[1] [4]

Symptoms of gliomas depend on which part of the central nervous system is affected. In our cases, most of temporal lobe gliomas are revealed by seizures(especially in someone without a history of seizures), Speech difficulties, headaches, vomiting, and cranial nerve disorders as a result of increased intracranial pressure, Confusion or a decline in brain function, Memory loss, Personality changes or irritability.

Epilepsy precedes the initial diagnosis in 24–68% of patients and develops in additional 19–38% later during the evolution of the disease.

Concerning neurocognitive performance, LTL glioblastoma patients exhibited significantly greater rates of impairment than RTL glioblastoma patients on tests of expressive and receptive language as well as attention and memory, Difficulties within these domains tend to worsen with involvement of multiple temporal lobe regions.

It's important to note that transient aphasia and transient sensorimotor deficits termed Todd's paresis may result from epilepsy, particularly from seizures evolving from the temporal lobe.

However, there is no typical clinical presentation of glioblastomas in general. And Despite its fatal prognosis, quality of life and cognitive functioning may be preserved or improved by the standard treatment options, even in the frail elderly population, but once first-line treatments have failed, decline of quality of life and cognition is often rapid and may also be severe.

Imaging techniques

Morphological imaging: CT AND MRI[1]

imaging provides important preoperative information regarding surgical options and the optimal site for biopsy to obtain tumor tissues that most accurately reflect the actual tumor grade. Contrast enhancement is a well-recognized imaging feature of high-grade glioma and is associated with histological attributes of aggressiveness and cellularity, although some low-grade gliomas can also manifest enhancement, but additional imaging findings of lesions crossing the midline, hemorrhage, heterogeneity, ill-defined borders, necrosis, and mass effect, as well as clinical factors such as the patient's age, can help in the differential diagnosis.

CT and MRI have been used for imaging the anatomic abnormality associated with glioblastomas. CT may be performed when MRI is not possible, because of metallic implants (cardiac pacemaker), or when acute hemorrhage is suspected, but the sensitivity of CT to detect glioblastoma-specific changes is inferior to MRI. The lesions are usually hypointense compared to the adjacent brain matter on CT images and the presence of a moderate to severe edema causes mass effect commonly resulting in midline shift.

On MRI, the lesions are often hypointense on T1-weighted images, hyperintense on proton density weighted images and hyperintense on T2-weighted images. Improved definition of tumor heterogeneity may be possible using the relatively new, FLAIR sequence that eliminates signal from cerebrospinal fluid and shows the lesion with a relatively high intensity compared with normal grey and white matter.

lesions are quite variable in appearance on contrast enhanced T1-weighted MR images and may include irregular and nodular rim enhancement, central hypodensity that is interpreted as necrosis, ill-defined margins and extensive surrounding hyperintensity corresponding to edema regions of abnormal contrast enhancement are present for the majority of GBs and typically represent macroscopic tumor.

DWI is particularly helpful in differentiating necrotic glioblastoma from Brain abscesses, in that pyogenic abscesses tend to be bright on DWI and dark on ADC maps, indicating restricted diffusivity, whereas tumoral cysts and necrosis are dark on DWI and bright on ADC maps, consistent with facilitated diffusivity. It is important to remember that intratumoral hemorrhage can result in low diffusivity within necrotic-looking tumor but that the presence of blood products is unusual in abscesses and can be readily confirmed by standard sequences.

Metabolic imaging

Magnetic Resonance Spectroscopy[1]:

Magnetic resonance spectroscopy can noninvasively measure concentrations of metabolites thus improving accuracy in predicting tumor grade.

Chemical shift imaging (CSI) displays regional variation in specific metabolites.

Commonly analyzed metabolites include N-acetyl aspartate (NAA), choline (Ch), creatine (Cr), myoinositol (mI), lactate (lac), and lipid.

NAA decreases in concentration in most known brain pathologic conditions that damage neurons (tumor, inflammation, and infection). And it can be absent when brain parenchyma is completely replaced by non-neuronal tissues (metastasis, cyst, and abscess…).

Choline is typically elevated in neoplastic processes particularly glioblastomas.

Elevation of the Ch:NAA ratio (greater than 2) in the peritumoral region can be a sign of tumor infiltration can also help differentiate areas of tumor with the highest cell density from edema in order to facilitate biopsy target selection, a Ch:NAA ratio of more than 2.1 at MRS has been used to detect regions of aggressive tumor phenotype within glioblastoma.

A high myoinositol concentration has been observed in low-grade astrocytomas.

Lipid and lactate levels typically increase with higher tumor grade as a result of hypoxia and necrosis and are correlated with poor survival.

Lactate/NAA ratio greater than 0.25, and the presence of lipid allow the distinction of brain parenchyma infiltrated by tumor.

MRS in conjunction with perfusion imaging has a sensitivity of 72% and a specificity of 92% in the differentiation of tumors from nonneoplastic lesions.

For infiltrative gliomas, MRS can detect distant tumor infiltration as showcased by increased myoinositol and glutamine levels in the contralateral hemisphere of patients with untreated glioblastomas.

Positron Emission Tomography[32]:

Tumors can be detected by the increased uptake of fluorodeoxy- glucose F 18 that correlates with both tumor grade and patient’s survival. Increased 18F-FDG uptake is not specific to tumor, however, being found in infectious and inflammatory processes.

In malignant brain tumors, higher proliferative activities in neoplastic cells result in increased amino acid transport, providing a basis for using radiolabeled amino acids as targets for brain tumor in PET.

Functional imaging in preoperative planning

Diffusion Tractography[33] :

diffusion tractography (DTI) uses water diffusion to estimate white matter tract directionality. Water diffuses parallel to axonal fibers and is restricted in the perpendicular direction termed anisotropy. With six or more MR gradient measurements, a diffusion tensor can be calculated.

From the diffusion tensor, the fractional anisotropy ranging from zero to one represents the degree of directionality of a tract. FA maps are color-coded 2D visual representations of directional water diffusion, while tensor maps are their 3D counterparts. Some of the most common tracts mapped in the treatment of temporal lobe gliomas in general is the arcuate fasciculus (language). DTI provides more sensitivity to tract disruption than routine MRI where white matter tracts might appear normal.

Arcuate Fasciculus[34]:

The Arcuate Fasciculus (AF) is a bidirectional white matter tract that connects the temporal (Wernicke's area), frontal (Broca's area), and parietal lobes. Because it spans much of the language dominant hemisphere, a tumor can interact with it causing language symptoms ranging from conduction aphasias to comprehension deficits depending on the affected region of the AF.

High angular resolution diffusion-weighted imaging (HARDI), a higher order DTI algorithm was used in brain tumor patients to map the AF to successfully predict long-term language dysfunction. They found that a patient whose language was intact had the preservation of the AF in common as well as the temporo-parietal component of the superior longitudinal fasciculus. As DTI becomes more refined, the subcomponents of the AF may become an interesting application for the DTI to predict even more subtle iatrogenic speech deficits.

Cortico-Spinal Tract[35]:

Connecting the regions of the motor gyrus to the spinal cord, these upper motor neurons set up a large descending bundle that goes from the medial and lateral cortex through the anterior limb of the internal capsule, into the medulla and into the spinal cord. Tumors that impinge upon the cortico-spinal tract can cause paresis or weakness at various levels in the descending pathway. DTI may be helpful in revealing displacement relative to a tumor.

Visual Projections[36]:

DTI has been used to find out the distance between optic tract fibers that can predict visual outcomes.

It is also used to localize the optic radiations in an effort to predict or spare function but this is a more common approach during epilepsy rather than tumor resection.

Limitations[1]:

Anything that affects water diffusion will affect the DTI measurement. Accordingly, edema is a common obstacle to accurate measures of diffusivity

Technical attempts are being made to better distinguish between gray matter, white matter, tumor, and surrounding edema.

Functional MRI Task

Based fMRI[37]

Task-based blood oxygen-dependent (BOLD) fMRI measures changes in blood oxygenation in a given region during a behavioral task. Increases in net neuronal activity lead to increased regional blood flow through specific metabolic signals that increases the diameter of local arterioles and drains local veins.

task-based fMRI scan demands timed participation from the patient in behavioral and cognitive tasks that can range from simple finger tapping to picture naming or sentence completion.

The most common applications of task-based functional neuroimaging for presurgical planning of glioma surgery are essentially language mapping and sensorimotor mapping.

Resting State fMRI[38]

resting state fMRI (RS-fMRI) measures spontaneous low-frequency fluctuations in the BOLD signal at rest to be able to discern patterns of cortical participation in spontaneous brain activity. Many intrinsic networks have been described using this technique including a somatosensory, a visual, an auditory, and a language network. RS-fMRI localizes members of a network, and measure the degree of correlation which may be related to the degree of network participation or “connectedness.” This measure allows to study the effects of a tumor on intrinsic connectivity.

the clinical utility of RS-fMRI is still in the process of being validated.

Language fMRI[1]:

Applications of fMRI language mapping in temporal lobe malignant glioblastoma patients can be divided into evaluations of language laterality and most importantly localization.

Language Localization[39]:

Localizing language may help the neurosurgeon in planning a trajectory to the temporal lobe tumors in general. This can be helpful information because both intra-axial and extra-axial tumors can displace function.

fMRI activations are thought to represent local field potentials that include inhibitory and excitatory components.

Related, a language fMRI map involves both essential and supportive activations. An essential activation is defined as a region critical for a task and cannot be resected without significant and major deficit. A supportive function, if resected, may impart a minor deficit. fMRI maps of language cannot distinguish between the two, and as a result, direct cortical stimulation is often performed in order to confirm which fMRI localizations are critical and which are expendable in the service of the most complete resection possible.

Language Lateralization[40]

Measurement of language lateralization helps identifying the hemisphere with greater involvement in language functions, which proves valuable for preoperative decision making for temporal lobe tumor patients.

Using fMRI to lateralize language was historically seen as a welcome alternative to the intracarotid amobarbital procedure (IAP or “Wada” test) due to its invasive nature.

Technical Considerations and limits of the fMRI in high grade glioma[1]

There are many technical considerations in the use of fMRI in malignant glioma patients. Of these, abnormal neovasculature that is inherent to many glial tumors is one of the most significant. High-grade gliomas in particular have been shown to decouple the BOLD fMRI signal from behavior through dysautoregulation. In such cases, the measured BOLD response lags the expected response when compared to the timing of the stimulus presentation. If not carefully identified, such an error can lead to false negatives.

TREATMENT

Goals

The major therapeutic goals for patients with temporal lobe gliomas in general revolves around getting a higher life expectancy with the preservation of the best quality of life that we can possibly obtain by minimizing language, cognitive, and memory deficits while also avoiding if not minimizing the recurrence of complications linked both to the tumor itself and to the therapeutic approaches (surgery, chemotherapy…).

Treatment for brain gliomas depends on the location, the cell type, and the grade of malignancy. Often, treatment is a combined approach, using surgery(maximum safe resection), radiation therapy, and chemotherapy.

Means

Medical treatment:

Intracranial hypertension[41]:

Due to the rigid skull, once compensatory mechanisms are exhausted, ICP rises due to the augmentation in volume of the brain tissue.

First level of treatment intensity: correct factors that may increase ICP

- Head-up positioning (maximum 30°)
- Maintain cerebral perfusion pressure 50–70 mmHg
- $PaO_2 > 8$ kPa (60 mmHg), preferably > 10 (75 mmHg) or even 12 kPa (90 mmHg)
- Sedation (propofol, fentanyl, neuromuscular blockers where required)
- Temperature: normothermia (36–37.5°C)
- Keep $PaCO_2$ normal (4.5–5.0 kPa; 34–38 mmHg)

If ICP >20 mmHg, without surgical options go to second level.

Second level of treatment intensity: increased intensity of medical treatment

- Mannitol 20% (e.g., 2 mL/kg up to three doses; caution if osmolality >or 320 mosmol/L)
- Hypertonic saline (e.g., 5% NaCl 2 ml/kg (do not repeat if Na >155 mmol/L)

- Consider reducing PaCO2 (3.5–4.5 kPa; 30–34 mmHg) and establishing ischemia monitoring (PbtiO2 or SjO2)
- Consider electroencephalogram and anticonvulsants if indicated
- Consider lowering body temperature to 35°C

If ICP >20–25 mmHg despite these measures, go to third level.

Third level of treatment intensity: therapies with controversial impact on outcome

- Consider deeper hypothermia (target 33–34°C)
- Consider barbiturate coma (maintain cerebral perfusion pressure).

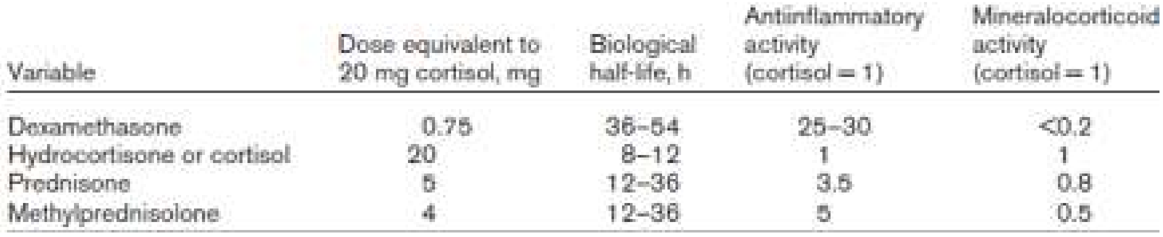
Management of perilesional edema[42]:

Corticosteroids:

Corticosteroids are the centerpiece of treatment for brain oedema. Side effects are tolerable when using low doses of steroids.

Dexamethasone is the most commonly used. It is approximately six times as potent as prednisone (20 mg dexamethasone is equivalent to 130 mg prednisone) and reaches full effect within 24 to 72 hours. Dosages of dexamethasone vary between 4 to 100 mg/day. Dexamethasone has lower mineralocorticoid effects than other corticosteroids. The latter effect may lead to increased risk of hyponatremia, which enhances the generation of edema.

The Preferable dexamethasone dose is 2×2mg/day orally. In acute situations, a bolus of 10 mg dexamethasone intravenously may be needed, followed by 16mg dexamethasone in divided daily doses.



Variable	Dose equivalent to 20 mg cortisol, mg	Biological half-life, h	Antiinflammatory activity (cortisol = 1)	Mineralocorticoid activity (cortisol = 1)
Dexamethasone	0.75	36–54	25–30	<0.2
Hydrocortisone or cortisol	20	8–12	1	1
Prednisone	5	12–36	3.5	0.8
Methylprednisolone	4	12–36	5	0.5

Table 5: Dexamethasone compared with other corticosteroids.

Osmotherapy:

Osmotherapy with mannitol, glycerol, or hypertonic saline is often used in patients with severe brain edema after stroke or neurotrauma. A typical dose of, for example, mannitol is 1 g/kg (250 mL of a 20% solution in an average adult), resulting in a reduction in intracranial pressure of 30 to 60% for 2 to 4 hours. Osmotherapy results in massive osmotic diuresis, so fluid and electrolyte balance should be monitored carefully.

Novel treatments:

Experimental treatment with VEGF, CRF, and cyclooxygenase inhibitors seems promising, but should undergo more clinical testing before it can be recommended for use in routine practice.

Management of epilepsy[43] [44] [45]:

It is crucial to note that epilepsy in this context is secondary, thus the definitive treatment of seizures in patients with glioblastoma in general is going to be mainly surgery unless, of course, it’s contraindicated.

The goals in managing patients with epilepsy are straightforward:

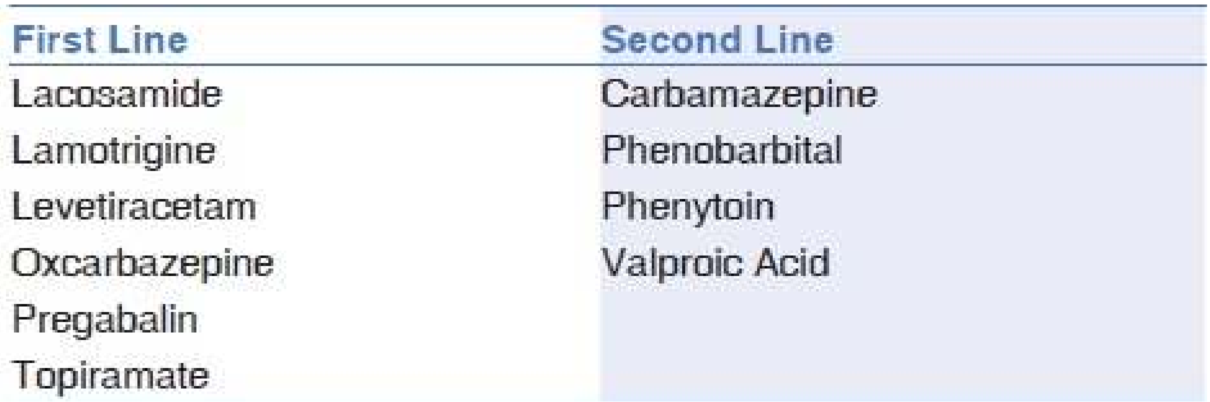
(a) eliminate seizures completely
(b) avoid medication side effects
(c) restore patients to physical, mental, and social health.

AEDs are always administered systemically, most often orally, but sometimes intravenously, intramuscularly, or rectally. Although generally convenient, such routes also increase the chances of systemic adverse effects that can limit the amount of drug that is given.

Antiepileptic drugs (AEDs) are commonly used prophylactically in patients with brain tumors after craniotomy, but evidence supporting this practice is very limited. a comprehensive systematic review and meta-analysis in 2019 showed that there was no significant effect in preventing de novo brain tumor–related epilepsy after craniotomy with AED treatment.

Following a tumor related seizure, patients should be offered AEDs. Although there is a lack of level 1 evidence to support the choice of AEDs for the treatment of brain tumor related epilepsy, there have been a number of studies over the past decade considering the efficacy of a range of AEDs. AEDs that are frequently selected as first-line therapy include second-generation non-enzyme-inducing AEDs such as lamotrigine and levetiracetam. Sodium

Valproate is frequently used as adjunctive therapy. Levetiracetam and sodium valproate have both been suggested as monotherapies with good seizure control profile in patients with glioblastoma, sodium valproate has been suggested to be associated with an improvement in survival of up to three months as reported in The European Organization for Research and Treatment of Cancer (EORTC) study. Initiation of a treatment should be considered when the risk for prolonged seizures or chronic epilepsy is high and the risk of toxicity is acceptable.



First Line	Second Line
Lacosamide	Carbamazepine
Lamotrigine	Phenobarbital
Levetiracetam	Phenytoin
Oxcarbazepine	Valproic Acid
Pregabalin	
Topiramate	

Table 6: recommendation for the choice of anti-epileptic drugs (these agents are presented in alphabetic order).

Surgical approach of Temporal high-grade gliomas[46] [47]

Principles of surgery in Temporal Lobe Gliomas:

Glioma becomes an area of the brain, and not a discrete lesion, and it is foolish to ignore the fundamental nature of this disease and allow the tumor to recur in easily expendable brain. In fact, surgery has different goals, and trying to not be aggressive on the tumor, often is not doing the patient a favor.

Temporal lobe glioma surgery is the basis upon which excellence in glioma surgery elsewhere is built. More saliently, learning the temporal lobe and how to not only stay safe, but also how to ensure an aggressive and complete resection allows us to learn a great deal about relationships with the rest of the cerebrum.

Preoperative Planning:

Temporal lobe glioma surgery is mainly built around the temporal lobectomy.

Temporal lobectomy is the best tolerated lobectomy and is very effective, guaranteeing an excellent resection. Most cuts on the back of the temporal lobe disconnect the majority of the temporal lobe circuits from the rest of the brain, so in most cases it is best to maximize the amount of tumor cells being removed.

Awake or asleep surgery?

Awake glioma surgery has traditionally been undertaken when the tumor is very close to the speech areas. Most of authors suggest that almost all glioma patients should undergo surgery while awake, with the only exceptions being patients who cannot handle awake surgery, and patients undergoing repeat surgery for a small recurrence. A rationale to keep essentially all patients awake:

a. a glioma that can be done better asleep than awake is essentially nonexistent. Being awake creates the possibility of protecting other functions, or taking a margin around the tumor. When you do awake surgery all the time, everyone (Neurosurgeon and anesthesiologist) becomes automatically better at it.

b. If the patient is asleep, you are not monitoring any of the speech and motor areas, making it easier to wreak havoc as you cut into deep brain areas with complex anatomy.
c. No anesthesia=no anesthesia related problems=quick recovery.
d. No anesthesia=no interferences with your mapping.
e. Patients with poor preoperative function (who many people don't map because they are hard to map) are the patients who need whatever little function they have left the most, and the ones who have their tumor closest to horrible brain areas.
f. Subcortical mapping requires continuous testing throughout the disconnection phase and continues until the entire cut is well clear of anything at risk in all directions.

Surgical stages for removing temporal lobe gliomas: the 3Ds Aids

The 3 "Ds" (Define; Divide; Destroy) represent the three stages of a glioma operation, and the transition between phases calls for changes in surgical technique.

The "Define" Stage

This is a non-destructive phase of the operation where the main goals are cognitive, and which begins prior to skin incision and ends when the brain begins to be cut. The sulcal patterns are studied thoroughly to precisely determine what anatomy is exposed and pinpoint the tumor's location. Image guidance and DTI are used to tentatively plan a cut and determine the goals of the cortical mapping. Cortical mapping is then done to determine the cortical termini of white matter, and safe entry points.

The Approach

The decision is relatively simple as there are two major tracts at risk, the IFOF, and the temporal ramus of the SLF. Further, with the exception of junctional and lateral cases (which are a "surrounded on all sides" type cut), the division will be either an anterior occipital or a posterior temporal cut, depending on the tumor's relationship to the temporal ramus of the SLF.

The craniotomy will be located over the planned portion of the MTG and STG where anticipating the SLF and its termini to be based on the DTI. The bone flap is made large enough to be movable a bit if we find essential parts of the speech network (this movement is most of the time done anteriorly). We must make sure we can reach the “corner of the temporal lobe”. We also make sure we can reach the temporal floor under the bone flap.

There is no need to elevate the entire temporalis muscle and take the bone down to the middle fossa floor.

In, inferior, anterior or hippocampal temporal lobe gliomas, it is almost always required to drop the tip of the head downwards as the difficult angles are typically looking from inferior to superior over the patient’s shoulder under the lateral system or the Insula, or upward into the atrium.

Cortical Mapping

How to Map the Cortex

Stimulation of the cortex is often executed with a bipolar electrode. The stimulation is used as a method to turning off the area we want to cut, and seeing if the patient is able to function without that area. This is why awake surgery is superior to most existing extra operative functional testing, like task based functional MRI, as most other methods tell you what brain areas are active during as task, but cannot accurately tell how a system would work without the part in question.

As always, we start the mapping with motor, motor planning, and speech arrest, but we should keep in mind that these functions are not usually found in this part of the brain. We generally don’t expose the supra-Sylvian opercula in these cases. reading and Naming are critical here, especially on the left side. We also perform neglect tasks, on either side, but it usually is mostly right-sided and localizes to the SLF.

How High Do You Set the Current?

The current setting is balanced between the desires to make sure negative sites are truly shut off during the testing against the risk of creating an intraoperative seizure. The best rule of thumb is starting low and escalating early in the mapping process (start at 2 mA and increase until we find a site or reach 4.5 mA).

The “Divide” Stage

This phase is the most critical, and the most challenging portion of the resection. In the division phase, the brain is divided in way that the bulk of the tumor specimens is separated from the eloquent brain regions which will be left intact. This is achieved with ongoing functional mapping of the subcortical white matter and is continued until the cut extends unequivocally past the critical regions.

Posterior Temporal Division

The posterior temporal disconnection is the longest and hardest portion of the temporal lobe surgery. The cut is an upside-down L-shaped cut, with its principle limbs paralleling the STG, and splitting the back of the temporal lobe from superior to inferior. In general, the posterior cut is angled posteromedially under the lateral white matter system into the temporal horn. The challenge with putting the cut very posteriorly is encountering the semantic networks, which on the right are neglect sites and the left are naming sites.

The first order of business with performing this cut is to find the Sylvian fissure and insula by taking down the STG from front to back. It is crucial to identify the “artery of death” exiting the posteroinferior fissure and try manipulating this as little as humanly possible. A stroke of this artery is devastating to language on the left side and will often injure the entire semantic network.

Once the STG has been removed, we make the posterior cut. It is best to try to find the temporal horn as soon as possible as this tells you where the IFOF is running (always above the horn), and the horn is the anchor that the rest of the cut is based around. We then continue the cut inferiorly until we are close to the temporal floor. At that point, we work in the temporal horn from inside out to extend this cut anteriorly below the insula. Once we are roughly anterior to the limen insula, the temporal lobe is disconnected, and the patient is put to sleep as the work on the middle fossa dura is fairly painful and does not require mapping.

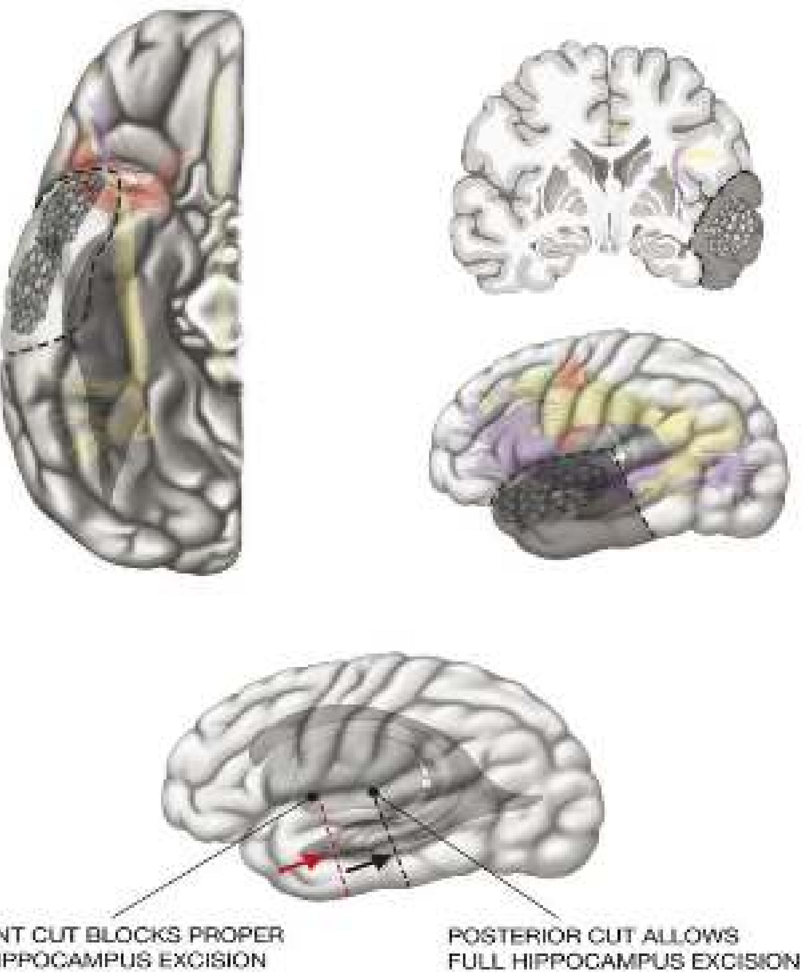


Figure 12: The main landing site for these cuts are the middle fossa floor and the temporal horn. First, the STG is removed subapically from front to back to identify the insula, to identify and free the arteries, and to get an idea of the orientation of the temporal pole. This subapicalization continues into the "corner," i.e., the anterior superior part of the STG which lies in front of the limen insula. Following this, the temporal horn is located by cutting the MTG and beginning the posterior limb just inferior to the location of the posteroinferior insula. Once this is opened, it is continued anterior to posterior along its length to clear the tumor from the IFOF from back to front. Finally, the posterior cut is continued until it roughly reaches the temporal floor and is down to the depth of the temporal horn.
At this point, the SLF is clear, and the disconnection is achieved.

Lateral Temporal and Junctional Disconnections

In lateral cases the disconnection is the entire case, as you are most likely disconnecting on the inferior, posterior, anterior and medial surfaces, with the sole safe boundary being the pia of the Sylvian fissure. Here, we stimulate often, work slow, stay subpial as much as possible, and use sulcal limits as thoughtfully as possible. At some point, the subcortical mapping, or concern for a tract on DTI will cause you to stop dissecting deeper, and the lower boundary is arbitrarily defined. you Occasionally make it to the ventricle in these cases.

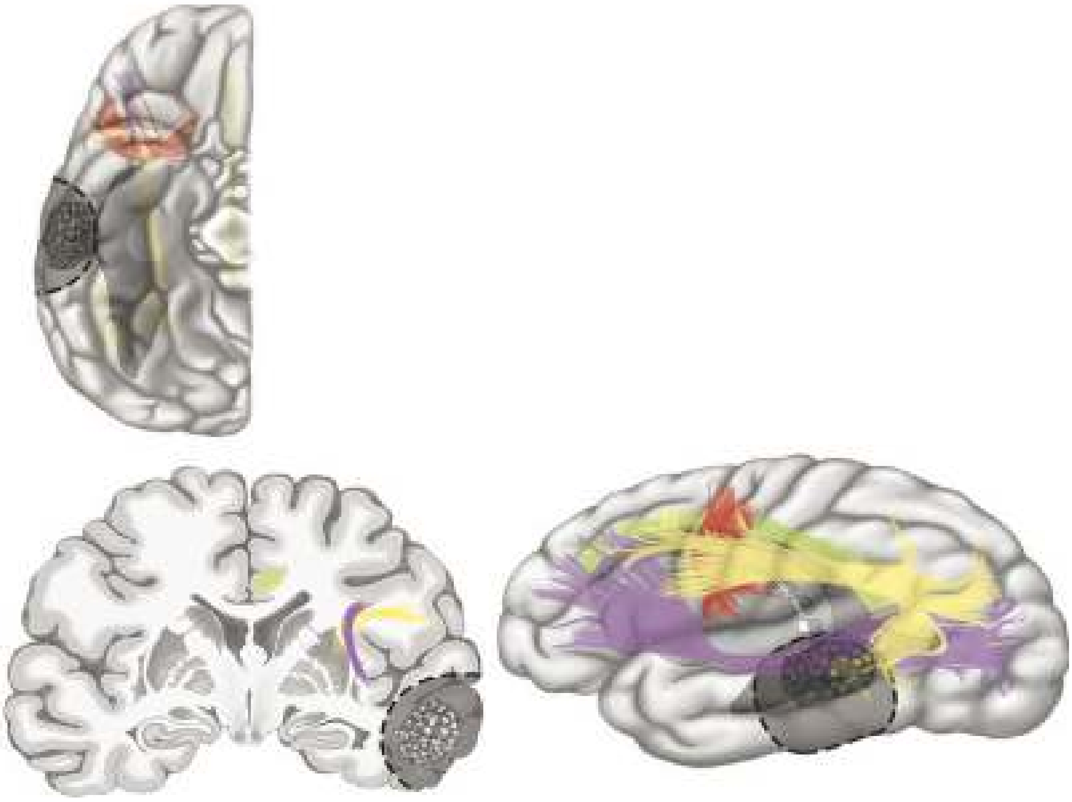


Figure 13: Schematic demonstrating a lateral temporal disconnection.
Tumors of the lateral system involve finding the sematic network and the SLF, and trimming the tumor from the front or back of the networks as tolerated by mapping.

Junctional tumors of the temporal lobe present the hard challenge of not only involving the lateral networks, but also following the fibers of the SMG into the deeper structures which puts the basal ganglia and the posterior internal capsule at risk.

The disconnection proceeds in two steps, similar to an insular glioma. In the first step a lateral disconnection is executed to remove as much of the SMG and STG as tolerated by the mapping. Note that the subpialization in this area is very complex. The second step involves, following the tumor into the back of the insula and resecting it as tolerated.

Once you have made the best possible window into the tumor, you should locate the surface of the insula, which may be small this far posteriorly, and if attainable enter the atrium, which provides excellent orientation to this area. Once you reach the deep part of the work, it is wise to switch your mapping to monitor the leg, as this is now what is at a higher risk.

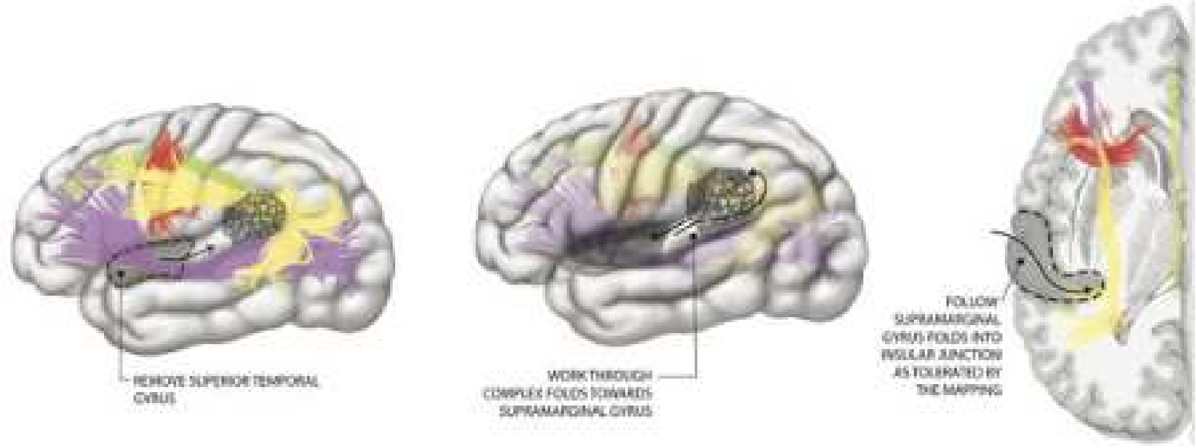


Figure 14: Schematic showing a junctional temporal disconnection.

The “Destroy” Stage

After the division phase, the remaining tumor volume is basically a non-eloquent brain tumor and can be taken care of by using standard anatomic techniques, like temporal lobectomy.

The en Bloc Anterior Temporal Lobectomy

The majority of temporal gliomas of the type which have an asleep portion (the hippocampal, anterior, and inferior), can be well managed with a temporal lobectomy. Usually an amygdalohippocampectomy is also indicated as these patients usually have some medial temporal lobe participation, often have seizures, and medial temporal lobe involvement gives the tumor access to an effective path to distant locations including the other side of the brain.

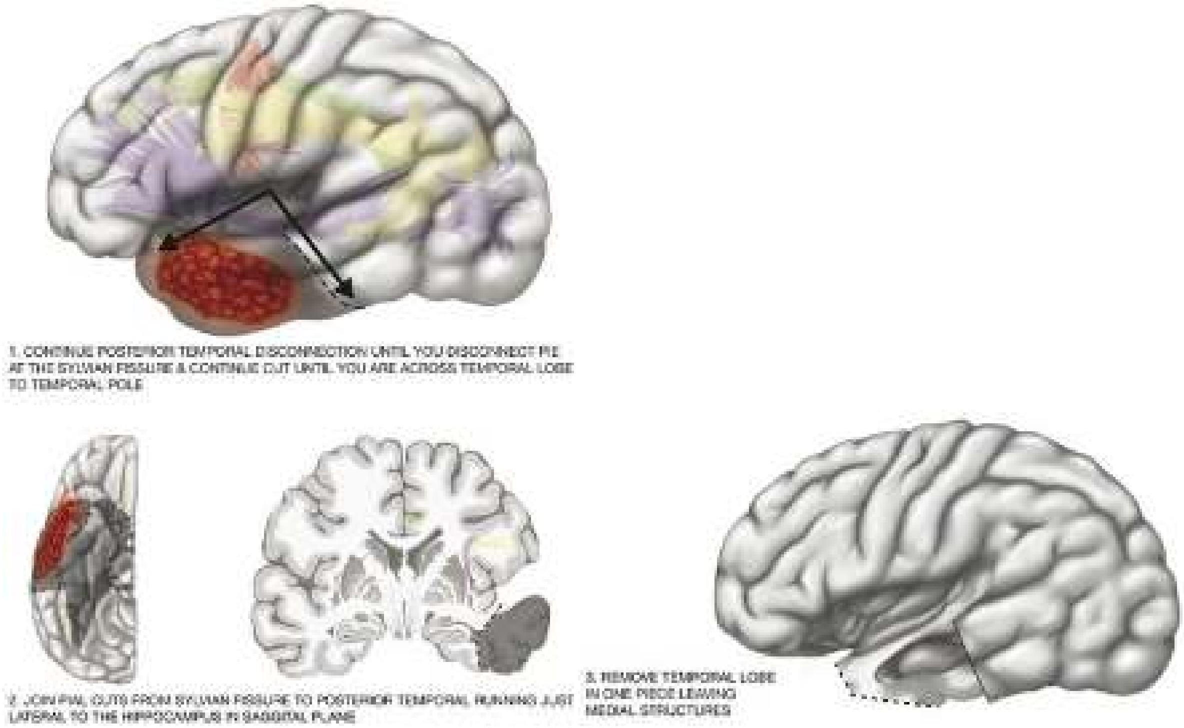


Figure 15: phases of an “en-bloc” temporal lobectomy.

The rationale for an en-bloc resection of the temporal lobe has nothing to do with oncologic benefit (as with en-bloc resections for other cancers), but instead its benefit is that it is more complete, faster, and would be considered safer, than the traditional method of trying to subpialize your way from lateral to medial until you find the uncus. Once the disconnection is made, the brain is subpialized from the anterior-superior corner, and the pia is cut and divided just below the sphenoid wing. The pial cut continues from anterior to posterolateral passing just lateral to the hippocampus until it curves laterally to meet the posterior limb of the disconnection. The temporal pole is then folded inward and the bridging veins are sacrificed and the lobe is taken out in one piece.

If you have performed the cuts described above, then the rest of the temporal lobectomy can be finalized in a couple minutes. This involves a “c” shaped cut along the inferior temporal lobe and pia, which extends from the part of STG abutting the anterior Sylvian fissure, continues anterior to posteriorly along the temporal floor running just lateral to the hippocampus then hooking laterally parallel to the superficial parts of the posterior temporal cut until it meets the inferior portion of the cut, after which the bridging veins are cut, and the lobe is extracted in one large piece.

The Medial Temporal Structures

In hippocampal tumors especially, the hippocampus and the uncus can be quite large, and this requires you to keep going with the resection until you have clearly seen the limits of the structure.

Subpialization is a crucial aspect of safe glioma surgery, especially in the medial temporal lobe. It involves gentle repeated suctioning on the surface to pull the brain out from under the pia. It is not recommended to bipolar extensively on the pia as the heat can conduct through and injure nerves and arteries below the pia, but occasional light bipolar bursts can loosen up the brain and allow it to suck away from the pia. You should not be using the bipolar to stop artery bleeds in this area.

Finally, it is important to preserve arteries deep to the tentorial incisura, but there are many arteries crossing the incisura at right angles to supply the hippocampus, PHG etc. These are always sacrificable once they are clearly identified, as they are supplying the portion of the brain you are removing. It is best to deal with these arteries before they avulse off their more important parent arteries in the cistern.

Steps of medial structures removal

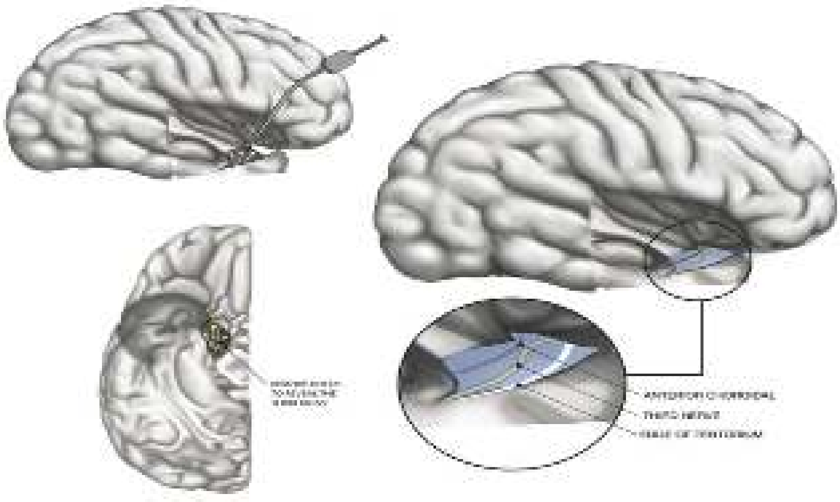


Figure 16: Removal of the uncus. The uncus is identified as the tissue located just superior anterior, and medial to the hippocampus, which extruded over the tentorial incisura. The uncus should be removed subpially, and this should continue until you visualize all four sulcal limits of the uncus, and until the third nerve and carotid are visualized through the pial bank.

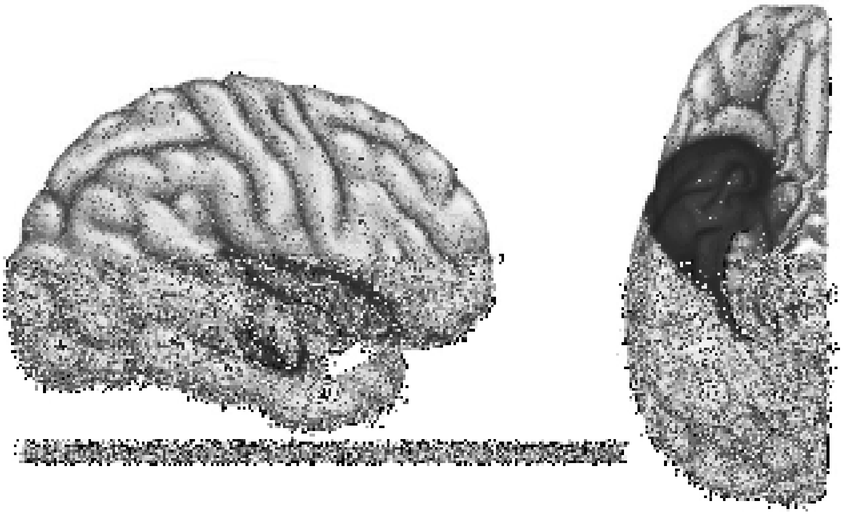


Figure 17: Removal of the hippocampus.

This step is always the last, as the hippocampus is a wonderful landmark. It is mandatory to continue to work until you clearly see the ependymal boundaries of the hippocampus within the temporal horn, as the hippocampus can be very large in these cases. After removing the hippocampus, further subpialization over the edge of the tentorium will remove the parahippocampal gyrus as far back as needed, until the edge of the brainstem is visible through the pia.

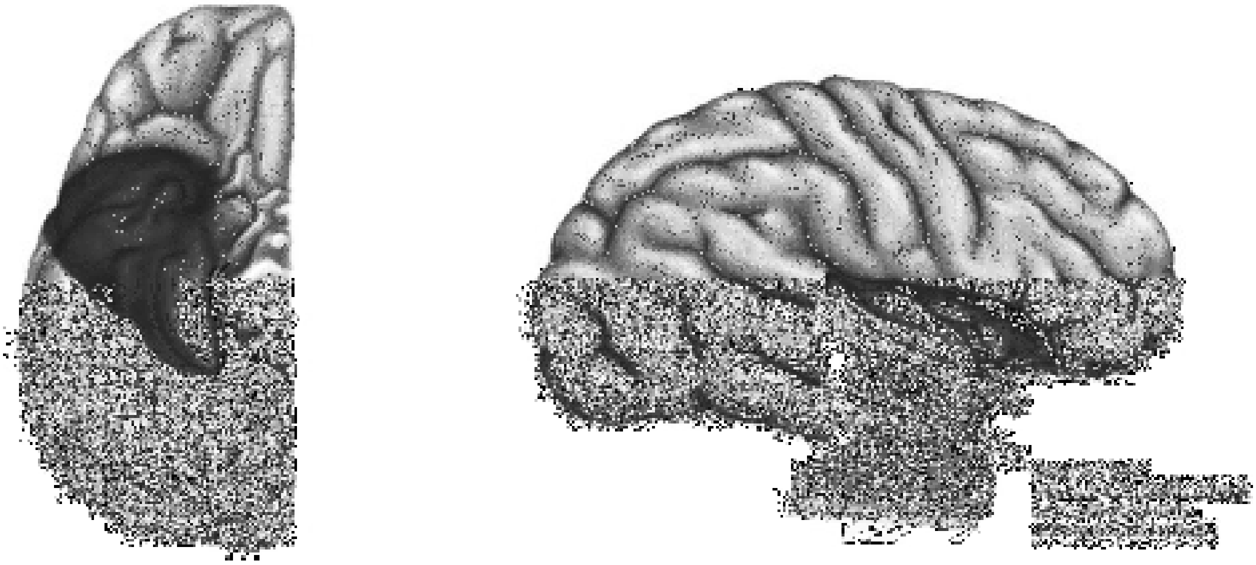


Figure 18: The removal of the parahippocampal gyrus.
Once the hippocampus is removed, everything remaining directly medial to the resection cavity is PHG. This can be removed by following the pia backwards from the uncus until the side of the brainstem is visible though the ambient cistern.

TPO Junction Gliomas

These tumors compel you to operate in a part of the brain that makes us particularly human. Not only are the gyri eloquent (MTG, STG, SMG, angular), but they are complex anatomically. The SMG and angular gyri are c-shaped and fold in a conical fashion with their white matter going down in a spiral hydra like fashion to enter the IFOF and SLF. The relationship with the insula is complex: while these gyri are opercula, the Sylvian fissure is shallow at this posterior section, and the unexperienced eye can easily miss the limit between opercula and insula. Finally, nearly every major association fiber tract of the lateral system (optic radiations, IFOF, SLF, posterior thalamic peduncle, MdLF, not to mention the basal ganglia and the leg portion of the internal capsule) passes under this area, making it easy to sabotage numerous functional systems in a small area of work. This goes without mentioning the fact that most of the arteries in this region are en passage to one critical brain area or another, including the artery of death. These tumors are never easy, and you are generally happy with a lesionectomy that doesn't cause too much damage, but a few tips follow:

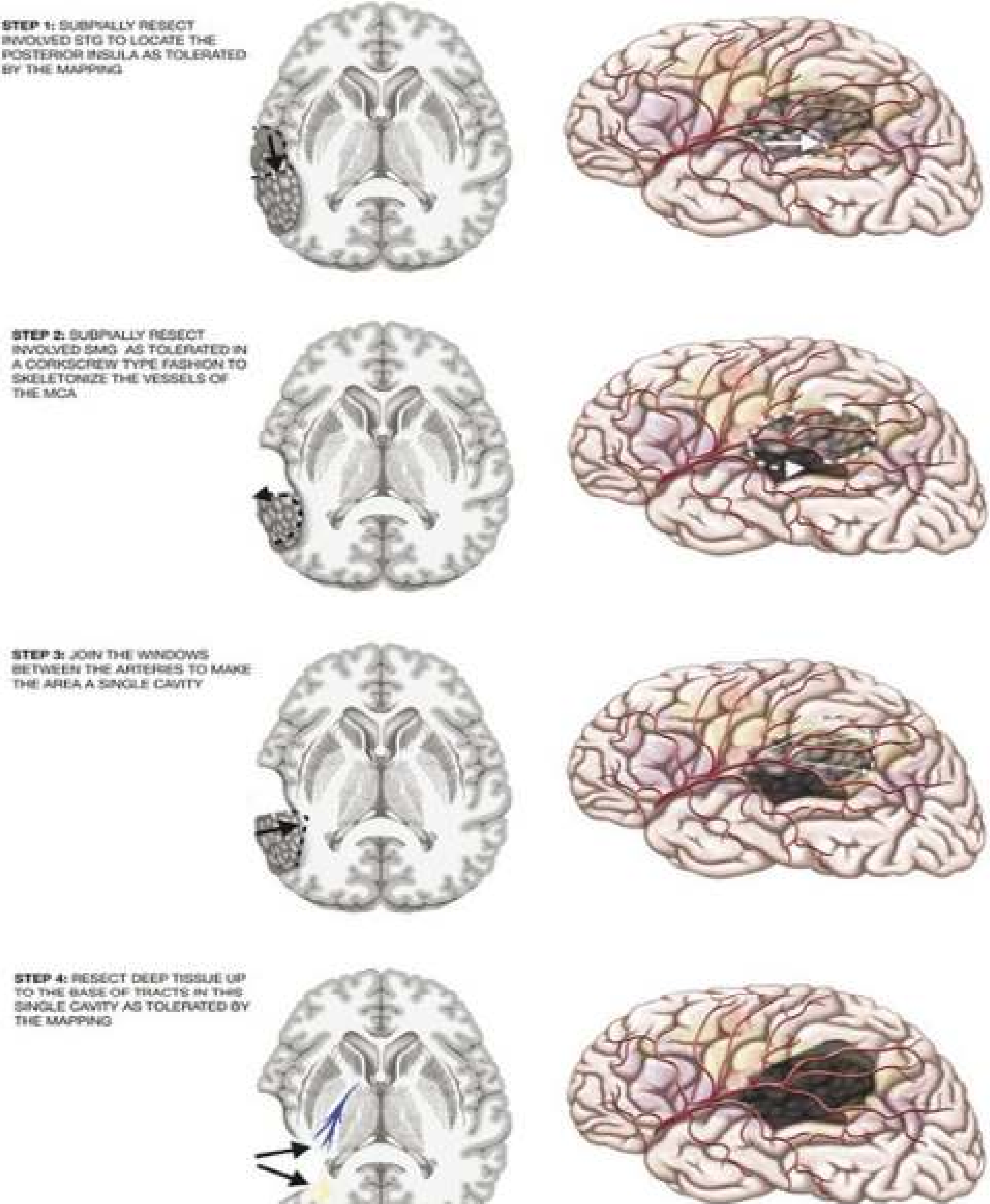


Figure 19: This figure demonstrates the phases of a TPO junction tumor.

temporal lobectomy may be considered in patients presenting high grade astrocytomas of the temporal lobe alone, and whose tumors can technically be totally resected via temporal lobectomy.

This procedure has the potential to lower local recurrences when compared to a standard wide local excision, and also improves the ability of the radiation oncologist to properly treat the necessary postoperative target to full dose while at the same time sparing critical normal structures.

Fluorescence-Guided Resection of Malignant Gliomas[1]:

During surgery, it’s sometimes extremely difficult to distinguish gross tumor and a more or less broad region of infiltrated and functionally intact tissue. neurosurgeons strongly tend to overestimate resection based on their subjective impression.

limitations of traditional methods for adequately identifying tumor tissue during the process of surgery have spawned new concepts, among which fluorescence, based on passive or active accumulation of fluorophores in tumor tissue.

Utilizing special wavelengths of light for excitation and filter combinations help to selectively detect fluorophores because they emit light at a wavelength differing and exceeding the excitation wave- length. The obvious advantage is real-time detection of tumor during surgery based on the difference of contrast between tissues with and without fluorophores. With fluorescence information available using modified surgical microscopes and light conditions, the surgeon can resect using the fluorescence mode. Tumor detection by this method is independent of neuronavigation and brain shift.

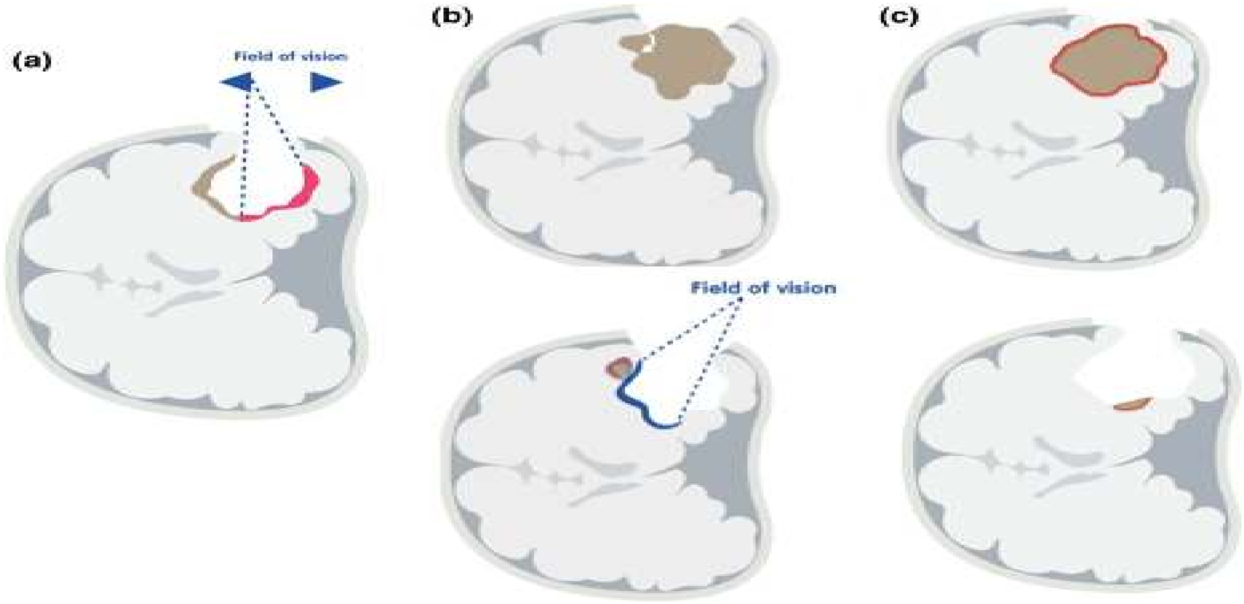


Figure 20: Possible pitfalls in fluorescence-guided resections potentially resulting in inadvertent residual tumor on post-op MRI.
A Residual tumor is hidden under over-hanging edges and is outside the direct field of vision; B cystic regions that collapse. Fluorescing tumor is not visible on the surface; C non-fluorescent necrotic tumor obscures residual enhancing tumor. Surgeons should take to resect following the margin of fluorescence

Intraoperative imaging:

Stereotactic Neuronavigation[48]

Stereotactic neuronavigation systems use 3D digitizers to register anatomical landmarks with preoperative imaging. The result is a GPS helping to localize the tumor through the use of a tracking system that links to a detector with a probe. modern, frameless neuronavigation became standard at major tumor centers in the last two decades.

With regard to surgical resection, more focused, smaller craniotomies can be performed and reliably centered over the tumor epicenter when guided by stereotactic neuronavigation

the combination of using multimodal neuronavigation with fMRI- and DTI-integrated systems, coupled with cortical and subcortical mapping techniques, enhances surgical safety by facilitating maximal surgical resection, identifying tracts for stimulation, reducing surgery time and preserving function.

The major limitations of the use of stereotactic neuronavigation include patient-to-image registration inaccuracies and brain shift, resulting in a decrease of anatomic accuracy.

numerous routine steps of an operation have been shown to negatively impact the accuracy of frameless stereotactic neuronavigation (placement of surgical drapes, the placement of skin retractors, and the process of performing a craniotomy...) along with the duration of the operation itself. These factors may add up to an overall spatial registration error of 5 mm.

State-of-the-art operating rooms with iUS and iRMI capabilities can help overcome this with the re-registration of intraoperative ultrasound and MRI images. allowing for the ability to navigate from a more precise representation of the current anatomical state.

Intraoperative Ultrasound (iUS)[49] [50]:

iUS is commonly used as a noninvasive adjunct in brain tumor surgery and can help evaluate normal anatomical relationships, providing information about location and size of brain tumors. Additionally, iUS with Doppler can provide information on the vascularity of a tumor, location of the main feeders, as well as the placement of normal cerebral vasculature. the transducer probe of the ultrasound is wrapped in a sterile sheath with sterile lubrication on the tip and thus can be readily available to the surgeon on the operative field throughout the entirety of the operation. The use of saline is important when using the iUS on the brain in an effort to improve acoustic coupling, and the operator has the ability to increase or reduce the intonation depth to provide more anatomical detail.

In general, hyperechoic lesions have less water content, a stromal components and rich capillary network.

Brain tumors often appearing hyperechoic to normal brain tissue. However, the variable appearance of the surrounding brain due to tumor infiltration and edema can make this interpretation difficult.

The most important advantage of iUS is the real-time information, including characterization and localization of a lesion, compensation for brain shift (stereotactic neuronavigation systems), and detection of unforeseen surgical sequelae.

iUS can define critical tumor margins and decrease the likelihood of leaving residual tumor

it can be readily available with a low cost when used routinely in an established tumor practice.

The time it takes to perform an intraoperative ultrasound is short and can be performed multiple times, without additional personnel if the surgeon has sufficient experience with its use and interpretation.

Intraoperative MRI[51] [52][53]

In the recent years, MRI became an important intraoperative tool due to its ability to image the brain during surgery thus facilitating superior neuronavigation and overcoming the limitations of stereotactic neuronavigation systems, rendering them more effective throughout the whole surgery.

it also has the outstanding benefit of verifying the extent of tumor resection and location of residual tumor after resection has taken place, Plus the capability to confirm the correct positioning of a biopsy needle in the case of stereotactic biopsies and the demonstration of early complications (ischemia, hemorrhage…) that can be addressed intraoperatively consequently, proving itself to be an effective tool in improving neurosurgical results while minimizing complications.

Despite these advantages, an iMRI system presents a lot of logistical and safety challenges, the first among these is the design and construction of a suitable operating room.

It is important to note that the iMRI system can be limited by metallic implants in the patient.

Drawbacks also include the significant upfront expense of the MRI systems and the requirement of additional personnel.

the benefits of obtaining intraoperative imaging must always be weighed against its limitations and also the risk of additional operating and anesthesia time.

The role of intraoperative stimulation mapping[1]:

ISM is acknowledged as the gold standard technique for brain mapping. It has proven effective in preserving the patient's functional integrity, providing him with a greater survival and functional outcomes.

An increased rate of early postoperative deficits is present in series using ISM, usually fully resolving within a period ranging from a few weeks to 3 months, which is probably due transient post- operative sequelae (perilesional contusion, oedema and hypoperfusion…).

In addition, the integration of the different stimulation modalities (the 60-Hz bipolar and the multipulse train-of-five monopolar stimulation) was proved to lower the rate of permanent motor deficits, resulting in the expansion of the population of patients who could benefit from the surgical treatment.

The Role of Laser-Induced Thermal Therapy in the Management of Malignant Gliomas[1]:

MR-guided LITT is a rapidly evolving and minimally invasive surgical technique that induces rapid cytoreduction in tumors not surgically accessible without a significantly high morbidity rate.

Treatment of glial tumors and brain metastases failing radiation and surgery remain one of the major indications for the use of LITT.

LITT procedures can be performed both in the operating room and in a diagnostic MRI suite. Using a neuro-navigation planning software, it is estimated that each laser pass will allow us to cover a cylinder of tissue of up to 25–30 mm in diameter. By overlaying each trajectory onto the lesion, one can plan the number of trajectories required to cover the lesion and the direction from which the laser is best introduced.

LITT can be performed under conscious sedation along with local anesthetics or general anesthesia.

best results are obtained if the imaging for the procedure is performed the day of surgery.

Cell kill coverage with LITT can range from 100% of lesions usually in those less than 3.5 cm in diameter, to frequently quoted ranges of 78–98% and can go as low as 28% in larger and more complex lesions.

While in some patients LITT can result in meaningful Progression Free Survival and Overall Survival, a neurological deterioration manifested by one third of the patients renders the role of its use in High Grade Gliomas ill-defined.

Ideally multicentered studies comparing LITT to best alternative management should be performed to determine its true efficacy.

Adjuvant therapy

Chemoradiotherapy[54]

The standard postoperative therapy protocol for patients with newly diagnosed glioblastoma in good general condition is radiotherapy (30 × 2 Gy = 60 Gy of the involved field) plus daily concomitant temozolomide at 75 mg/m2, followed by an interval of approximately 4 weeks without any therapy and up to 6 cycles of temozolomide at 150–200 mg/m2 on 5 out of 28 days. The cooperative effort of the European Organization for Research and Treatment of Cancer and the National Cancer Institute of Canada demonstrated improvement of median overall survival from the adjunct of temozolomide to radiotherapy alone by 2.5, but benefit from temozolomide in patients without MGMT promoter hypermethylation was only marginal.

Furthermore, patients aged 66–70 years appeared not to benefit from the association of temozolomide to radiotherapy.

Chemotherapy[1][55]:

In recent years with increasing recognition of the chemosensitivity associated with ODs and 1p/19q, chemotherapy has played a significant role in the management of this disease. The association of 1p/19q with chemosensitivity was showcased to be significant in oligoastrocytomas and anaplastic ODs. While chemotherapy didn't affect median survival in combination with radiation in non-deleted tumors (2.6 vs. 2.7 years), there was a significant improvement if, procarbazine, vincristine and lomustine (PCV) were associated with RT (14.7 vs. 7.3 years). Given the increasing role of IDH status in gliomas, there was an overall survival benefit to combined radiotherapy and chemotherapy in IDH-mutated tumors but not in IDH wild-type tumors.

studies have shown improved response to chemotherapy in tumors with 1p/19q codeletion, and showcased an association of IDH mutation with an improved overall survival and chemosensitivity with temozolomide.

Therapeutic Approach in Elderly Patients

postoperative chemotherapy with temozolomide is now used instead of radiotherapy in elderly patients with hypermethylation of the MGMT promoter. temozolomide at 150–200 mg/m2 on 5 of 28 days is the standard dosing regimen that should be followed in patients with both newly diagnosed and recurrent glioblastoma.

Radiation Therapy[1] [56]:

Historically, radiotherapy comes after surgery within weeks from completion or delayed and utilized in case of progression or recurrence.

Postoperative conventional daily radiotherapy improves survival for adults with good functional well-being and high-grade glioma compared to no postoperative radiotherapy. Hypo fractionated radiation therapy has similar effect for survival compared to conventional radiotherapy, particularly for patients aged 60 and older with glioblastoma.

Radiations act by causing DNA damage resulting in the apoptosis of active tumor cells while also damaging normal surrounding brain tissue. Short term side effects (fatigue, dizziness, headaches, vomiting, nausea, seizures…) can resolve; however, the concern comes with long-term side effects like reduced quality of life, neurocognitive changes, and radiation necrosis.

Treatment of gliomas, specifically ODs, is usually a dose to 45–54 Gy in 1.8– 2.0 Gy/fractions.

Role for Stereotactic Radiosurgery and Stereotactic Radiation Therapy

SRS and SRT are techniques using an external stereotactic localization system to accurately aim at small targets typically within the cranium. Because of the combination of high accuracy targeting a small volume, high doses of radiation can usually be safely administered.

the term SRS is used When delivering the dose in one treatment (or up to five).

the term SRT is used When the stereotactic planning and delivery system is employed with more traditional low dose per day fractionation.

SRT and SRS allow for limited dose to the adjoining regular structures by utilizing multiple radiation beams, usually with arcs, accurate localization of the lesion.

treatment precision and accuracy are within 1– 1.5 mm with frame-based or frameless approaches. the target volume with SRS is defined by residual or recurrent T1-enhancing tumor in comparison to conventionally fractionated treatment.

Given that glioblastoma usually recurs within the original treatment field after conventional doses of RT, radiosurgery proved itself to bea useful adjunct to other treatment modalities.

Hypo fractionated stereotactic radiotherapy (hSRT) incorporates SRT with intermediate dose per fraction and a limited number of fractions.

Radiosurgery and proton irradiation

Radiosurgery and proton irradiation are rarely used in glioblastoma in general, because the high precision of dose application which is the signature of these techniques does not match the requirements of a diffusely infiltrating disease process.

Management at Recurrence[1]:

treatment options are overall limited, and therapeutic options depend not only on previously administered therapies, but are substantially influenced by tumor characteristics, availability, and local preferences. The efficacy of any established treatment for recurrent glioblastoma is modest. Therefore, best supportive care focusing on the amelioration of symptoms including psycho-oncological interventions may be adequate in many patients already at first progression.

Disease monitoring:

MRI, typically performed every 2–3 months, is the standard method for the diagnosis of recurrent disease. Unimodal response assessment relying on contrast enhancement was the standard approximately two decades, but pseudo response, rapid normalization of contrast enhancement under anti-angiogenic treatment, and pseudo progression, an increase in size of contrast-enhancing lesions after radiotherapy, challenge MRI-based response evaluation in glioblastoma. therefore, multidimensional criteria defined by the Response Assessment in Neuro-Oncology working group, which takes into consideration type and time of treatment, corticosteroid use, T2/FLAIR, and clinical characteristics, have been widely adopted and highlight the requirement of interdisciplinary boards for treatment decisions in glioblastoma patients in general.

Role of repeat surgery:

There is No association of repeat surgery with overall survival. However, post-recurrence survival was almost twofold longer in patients with complete resection of contrast-enhancing tumor thus indicating that repeat surgery should only be offered to patients where total resection of contrast-enhancing tumor seems possible.

Role of radiotherapy:

repeat radiotherapy in glioblastoma, particularly in patients with smaller tumor volumes, better general condition, younger age, and a period of at least 6–12 months after first radiotherapy. schedules that have been used are in the range of 30– 36 Gy in 2–3.5 Gy fractions, often used as stereotactic radiotherapy.

hypo fractionated radiotherapy is the first-choice treatment at recurrence in elderly patients with MGMT methylated glioblastoma.

Role of chemotherapy:

The majority of patients are treated with single agent systemic treatments at recurrence of glioblastoma. Options that are usually well tolerated include CCNU/lomustine, temozolomide, and bevacizumab.

alkylating agent lomustine is commonly utilized for recurrent glioblastoma. Oral administration at longer time intervals (90–110 mg/m2 every 6 weeks) is advantageous in patients with altered general condition, but progression-free survival rates at 6 months do not exceed 19–25%.

temozolomide is paired with longer post-recurrence survival, particularly in patients with apparent benefit from first-line temozolomide after a temozolomide-free interval. Such patients bare mostly tumors with hypermethylation of the MGMT promoter.

Guidelines summary[57] [58]:

The treatment of glioblastomas remains difficult in that no contemporary treatments are curative. While overall mortality rates remain high, recent work leading to an understanding of the molecular mechanisms and gene mutations combined with clinical trials are leading to more promising and tailored therapeutic approaches. Patients with glioblastomas should be evaluated by a team of specialists, including a neurologist, neurosurgeon, neurooncologist, and radiation oncologist, in order to develop a coordinated treatment strategy.

Upon initial diagnosis of glioblastoma multiforme (GBM), standard treatment consists of maximal surgical resection, radiotherapy, and concomitant and adjuvant chemotherapy with temozolomide.

Concerning the temporal malignant glioma, Roh al. advanced that gross-total resection plus an additional lobectomy, when performed on the nondominant side, can improve survival without impairing a patient's functionality. Since the extent of resection is the only modifiable prognostic factor demonstrated for GBM to date, the application of this surgical method could improve the overall survival of GBM patients.

Because these tumors cannot be cured with surgery, the surgical goals are to establish a pathological diagnosis, relieve mass effect, and, if possible, achieve a gross total resection to facilitate adjuvant therapy.

Special consideration should be given to some important factors, including age of the patient, location and size of the tumor, neurological status, functional impairment (quantified by Karnofsky Performance Status Scale), significant comorbidity and patient and family preferences. Patients with GBM should have **surgery for maximal tumor removal** whenever safe, to facilitate adjuvant therapy. **Biopsy** is only indicated in cases of "inoperability" of the tumor, because of the associated risks are minimal. The image-guided stereotactic techniques are preferred over open biopsy.

The National Comprehensive Cancer Network (NCCN) has released guidelines on central nervous system cancers which includes recommendations for the diagnosis and treatment of glioblastomas (grade IV gliomas). The goals of surgery are to obtain a diagnosis, alleviate symptoms of increased intracranial pressure or compression, increase survival, and decrease the need for corticosteroids. Adjuvant treatment options depend on the patient performance status, age and MGMT promoter methylation status.

Category 1 recommendations for first-line treatment:

- Patients 70 years or younger, with good PS, regardless of the tumor's MGMT methylation status should receive fractionated standard brain RT plus concurrent and adjuvant temozolomide (TMZ) with or without alternating electric field therapy.
- Patients older than 70 years, with good PS and MGMT promoter-methylated tumors should receive hypo fractionated brain RT plus concurrent and adjuvant TMZ or standard brain RT plus concurrent and adjuvant TMZ and alternating electric field therapy.
- Patients older than 70 years, with good PS and MGMT unmethylated or indeterminant tumors should receive standard brain RT plus concurrent and adjuvant TMZ and alternating electric field therapy.

Most glioblastomas recur in and around the original tumor bed, but contralateral and distant recurrences are not uncommon, especially with lesions near the corpus callosum. The indications for reoperation of malignant astrocytomas after initial treatment with surgery, radiation therapy, and chemotherapy are not firmly established.

OUTCOMES AND FOLLOW-UP

Recurrence:

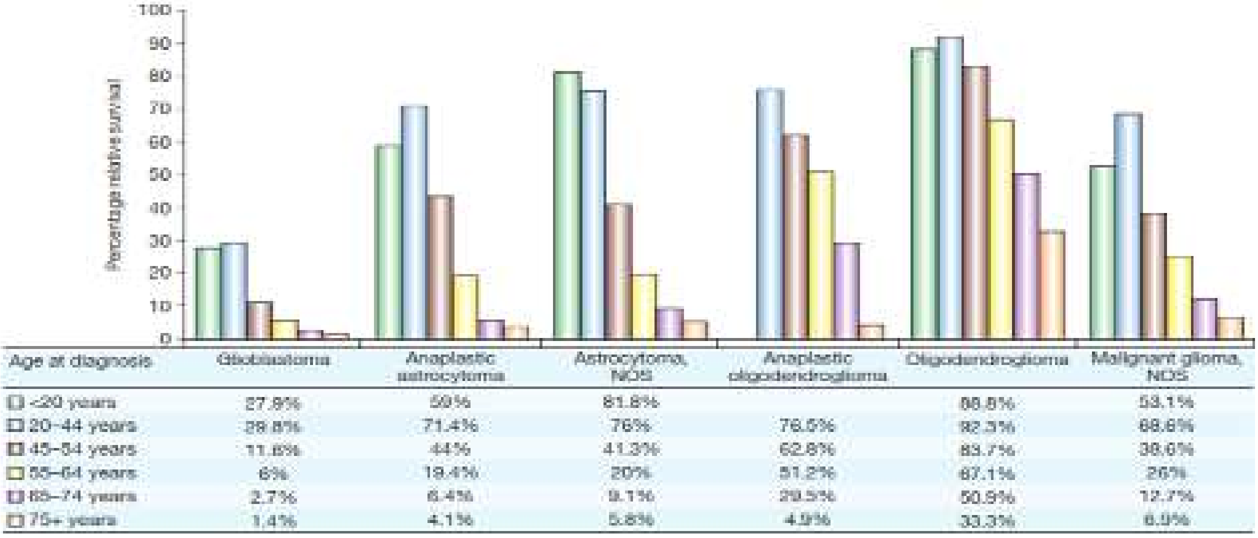
Recurrence inevitably occurs, most patients experiencing it after 7–8 months of primary treatment.

Mortality:

Despite the synergistic multimodal strategies and individualized therapies, the available treatment is of limited utility, and patients have a poor prognosis, with a progression-free survival (PFS) of 7–8 months, a median survival of 14–16 months and 5-year overall survival (OS) of 9.8%[1].

Age:

The median diagnosis age is 64 and survival rates decrease with age. Approximately 33.3% of children and adolescents with glioblastoma survive for two years, as opposed to only 3.3% of patients aged 75 or more[1]. Despite the better prognosis of younger glioblastoma patients compared to the older ones, no curative therapies exist for either of them[1].



Age at diagnosis	Glioblastoma	Anaplastic astrocytoma	Astrocytoma, NOS	Anaplastic oligodendroglioma	Oligodendroglioma	Malignant glioma, NOS
☐ <20 years	27.8%	59%	81.8%		88.8%	53.1%
☐ 20–44 years	29.8%	71.4%	76%	76.5%	92.3%	68.6%
☐ 45–54 years	11.6%	44%	41.3%	62.8%	83.7%	38.6%
☐ 55–64 years	6%	19.4%	20%	51.2%	67.1%	26%
☐ 65–74 years	2.7%	6.4%	9.1%	29.5%	50.9%	12.7%
☐ 75+ years	1.4%	4.1%	5.8%	4.9%	33.3%	6.9%

Figure 21: Glioma 2-year relative survival probabilities by age at diagnosis and histologic subtype[59]

A study conducted in the US between the years of 1973-2002 showed a lower survival rate amongst the 75+ years population affected with glioblastoma compared to the higher survival rate amongst younger population especially those who were diagnosed with oligodendroglioma[59]

WHO grade[60][61] [62]:

Typically, any tumor presenting as above WHO grade I will have a prognosis resulting in eventual death, varying from years (WHO grade II/III) to months (WHO grade IV).

the median overall survival of anaplastic grade III gliomas is approximately 3 years, glioblastoma multiforme has a poor overall survival of 15 months.

Surgery[63] [64]:

In spite of aggressive local treatment with maximal surgical resection and adjuvant radiotherapy, a lot of temporal lobe GBMs and glioblastomas in general fail locally, study supports the theory of neural pathway infiltration as one of the most important patterns of failure.

achieving a >98% resection was significantly associated with improved overall survival compared to resection of <98% of the tumor (median survival 14 months vs. 9 months)

Type of resection	Local failure	Regional failure	Distant failure	Isolated regional or distant failure
Subtotal resection or biopsy	80%	63%	43%	20%
Total or near total resection	86%	33%	18%	14%

Table 7: Patterns of failure depending on the extent of resection.

A study conducted in Australia showed that overall failures are fairly frequent and that except local failure, total or near total resection of the tumor had a lower percentage of failure compared to the subtotal resection.

Prognostic value of molecular profiles[1]:

IDH prognosis

IDH 1 and IDH 2 mutations are found to be correlated with a favorable prognosis and overall prolonged survival time independent of treatment. Interestingly, patients with IDH1-mutated glioblastomas (grade IV) show better survival than patients with wild-type anaplastic astrocytomas (grade III). The IDH status, however, does not predict treatment-specific responses of patients with glioma.

1p/19q prognosis

patients with 1p/19q deleted tumors, who undergo tumor resection alone without receiving any adjuvant treatment, do not show longer progression-free survival, suggesting that 1p/19q loss characterizes a tumor with higher sensitivity to genotoxic agents. Cases of anaplastic oligodendrogliomas with IDH mutation and 1p/19q co-deletion had significantly prolonged median survival time when treated upfront with vincristine and radiotherapy compared to radiotherapy alone.

The absence of 1p/19q co-deletion in anaplastic oligodendrogliomas led to a significantly shorter survival time and showed no difference between radio-chemotherapy and radiotherapy-only.

MGMT prognosis:

The MGMT promoter methylation status is considered at the moment to be one of the most significant predictors of clinical outcome and response to treatment with temozolomide[1].

glioblastoma cells with MGMT promoter that are hyper-methylated respond better to temozolomide, as they lack the ability to efficiently repair the damage introduced by alkylation. In the presence of MGMT hyper-methylation, radiation alone had no significant extension of survival times.

Also, MGMT promoter methylation patterns can change between initial tumor diagnosis and later recurrence, particularly in MGMT-methylated cases implying that MGMT methylation is only of prognostic value for the initial assessment, and it is not predictive of outcome for recurrences.

ILLUSTRATIVE CASES

Case 1:

A 49-year-old patient, without medical history, complained about a worsening headache associated with intermittent vomiting and concentration problems for the past two months.

The evolution was made towards the aggravation of the signs of intracranial hypertension with the occurrence of seizures evolving in a context of asthenia without fever.

the day before his admission, the patient presented a rapid decline of consciousness with a Glasgow score fluctuating between 12 and 14.

In the clinical exam, the patient is afebrile, pupils are equal and reactive, there is a positive Cushing's reflex, brusinski was positive on the left and indifferent on the right.

Preoperative cerebral MRI showed a voluminous right intra-axial temporal process. There is also the presence of a significant perilesional edema exerting a mass effect on the medial line.

After the introduction of an anti-edematous treatment, the patient was operated for a subtotal resection of the tumoral process.

Anatomopathological examination showed that it was a glioblastoma.

The post-operative evolution was favorable without the occurrence of any deficits.

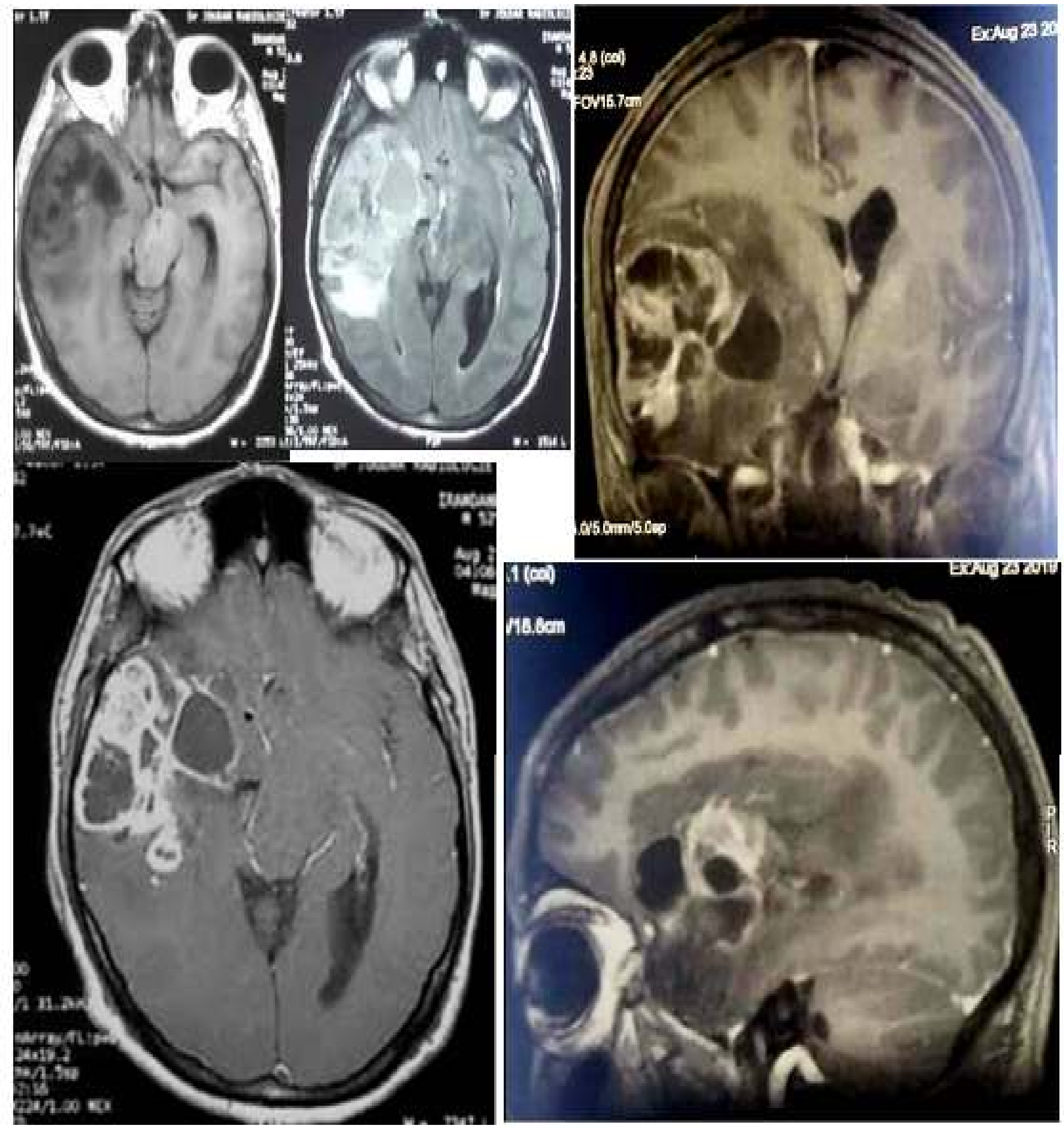


Figure 22: preoperative MRI on T1WI without and after gadolinium, FLAIR and T2WI showing the huge right-side temporal glioblastoma.

Figure 23: postoperative imaging of a 49 years old patient diagnosed with a temporal lobe glioblastoma

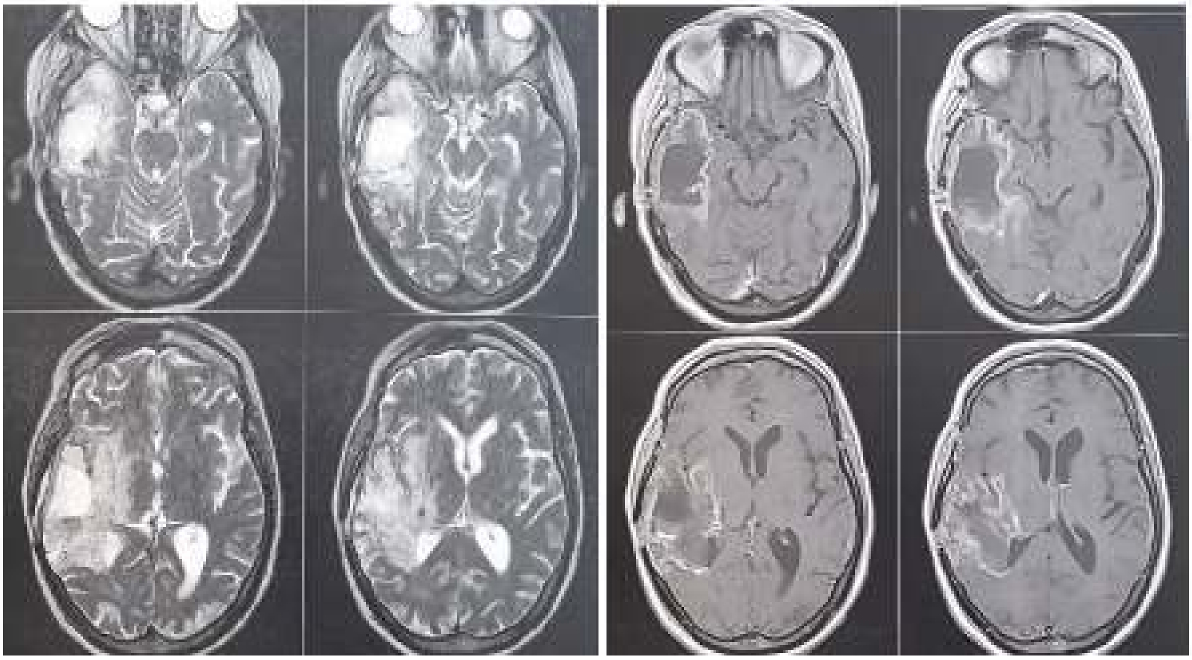


Figure 24: control MRI after 10 months of the 49 years old patient diagnosed with temporal lobe glioblastoma.

Case 2:

This is a 60-year-old patient without medical history who suffered of progressively worsening headaches during the previous two months, associated with a visual disturbance that has evolved towards a weakness of the right side without seizure or consciousness disturbance.

On examination, the patient was conscious aphasic and presented a discrete right hemiparesis.

Cerebral MRI showed a left expansive temporal-insular process with an associated sub-falcorial engagement

The patient was operated, under general anesthesia, with a subtotal resection of the mainly temporal component of the tumor. There was no remarkable incident in the post-operative courses.

The pathology revealed that it was a glioblastoma.

An adjuvant radio-chemotherapy was undertaken. The evolution was marked by a recurrence of the tumor 10 months after the first operation revealed by the reappearance of headaches throughout 1 month without any other associated sign.

The MRI control showed the progression of the remnant tumor with huge peritumoral edema.

The patient is now scheduled for a second tumor reduction surgery.

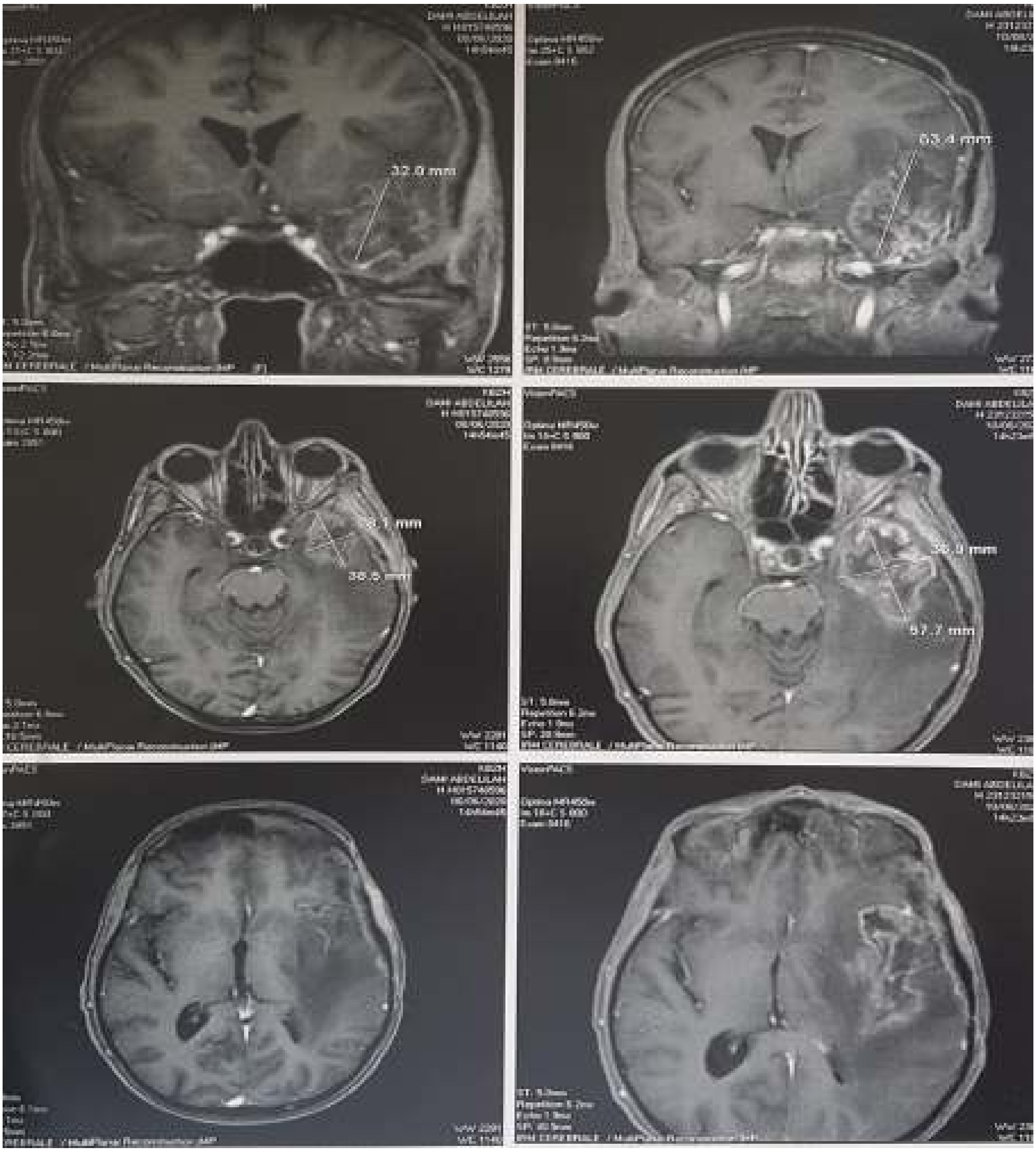


Figure 25: preoperative MRI of a 60 years old patient suffering from a left temporal glioblastoma.

Figure 26: postoperative CT scan imaging of a 60 years old patient diagnosed with temporal lobe glioblastoma

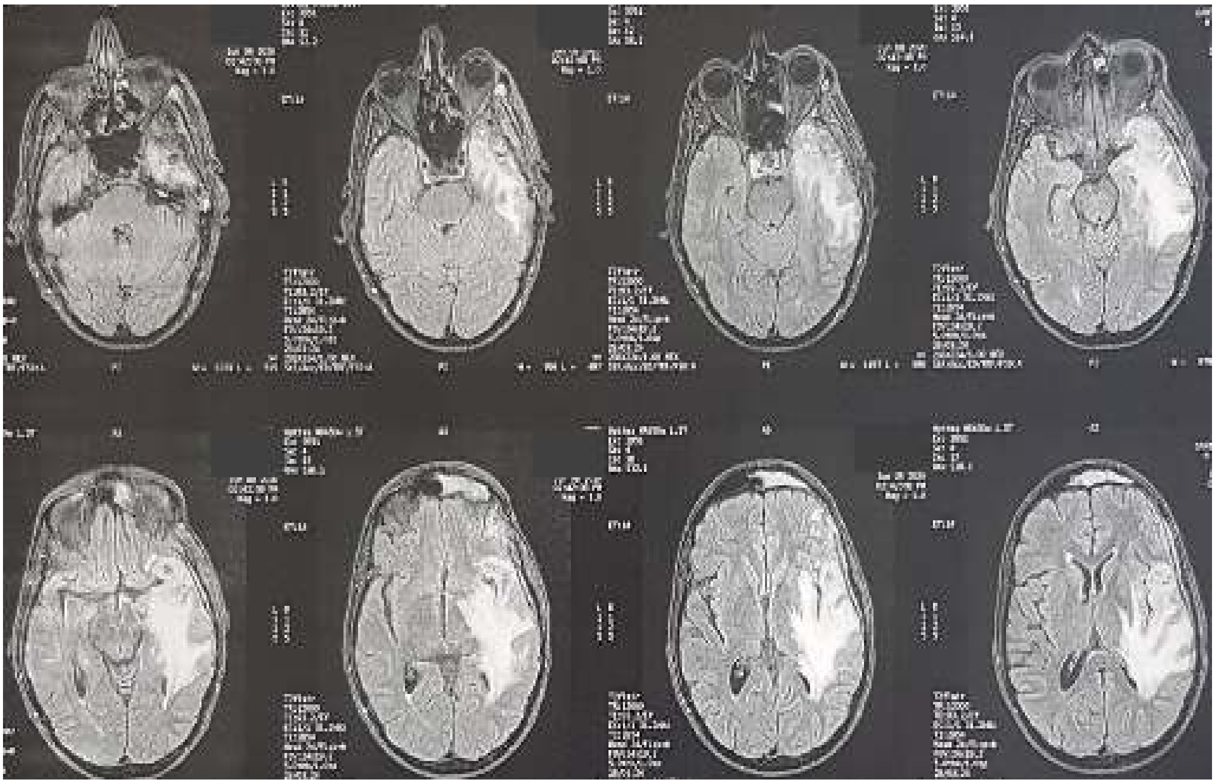


Figure 27: MRI FLAIR control of the follow up at 10 months of the patient previously diagnosed for left temporal lobe glioblastoma and treated by subtotal resection followed by radiotherapy and chemotherapy, presenting a worsening course of headaches without neurological deficit. We find out a severe edema involving the adjacent white matter tracts.

Case 3:

This is a 78-year-old patient treated for prostate adenoma in 2014 who presented over the course of 3 weeks a weakness of the left side with visual disturbance and general asthenia.

On exam the patient was confused presented a bradypsychie, aphasia and a predominantly brachial left hemiparesis.

Cerebral MRI reveals a 6 cm right temporo-insular process with features of malignancy. Due to the poor Karnofsky score of the patient a minimal therapeutic approach has been proposed. Indeed, a CT-guided stereotactic biopsy was performed under local anesthesia. Pathology demonstrated that it was a glioblastoma.

The patient was placed on concomitant radio-chemotherapy with TEMODAL for 6 weeks.

Faced with the no improvement of the patient's condition, it was decided to abstain from therapy during a neuro-oncology staff meeting. The patient died 2 months later.

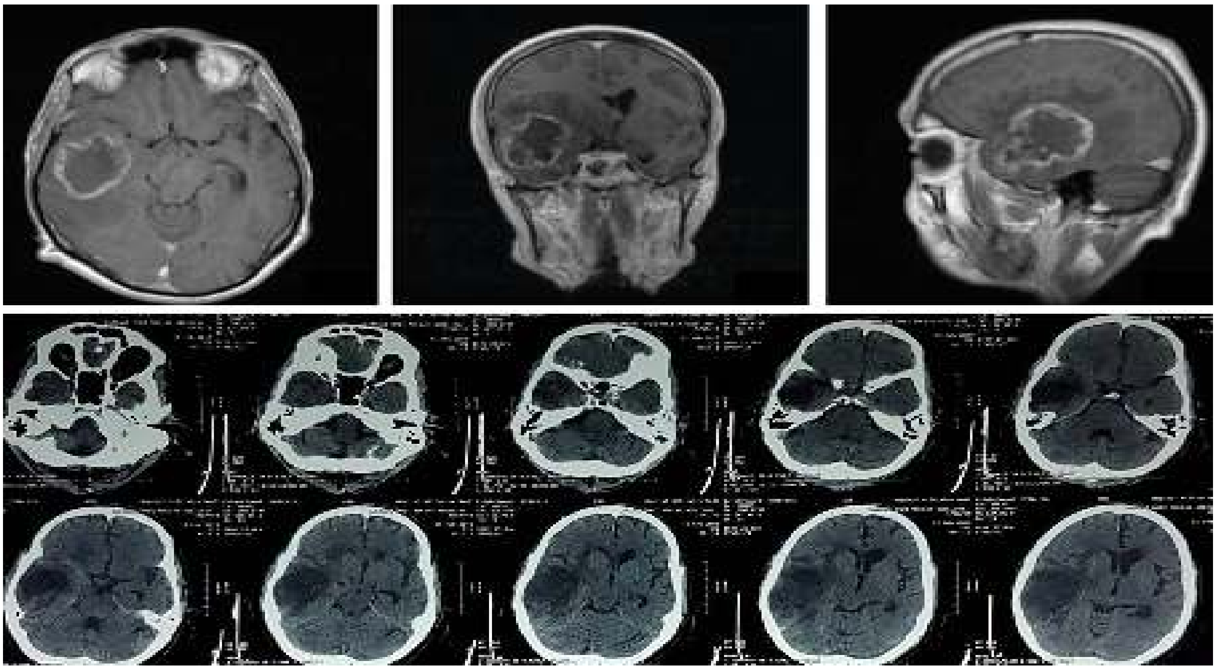


Figure 28: T1WI with gadolinium in axial, coronal and sagittal plan showing the large temporal tumor with cystic component and enhancing rim. Below is the axial CT scan imaging without contrast performed 1 week after the stereotactic biopsy showing the enlargement of the cyst and the surrounding edema.

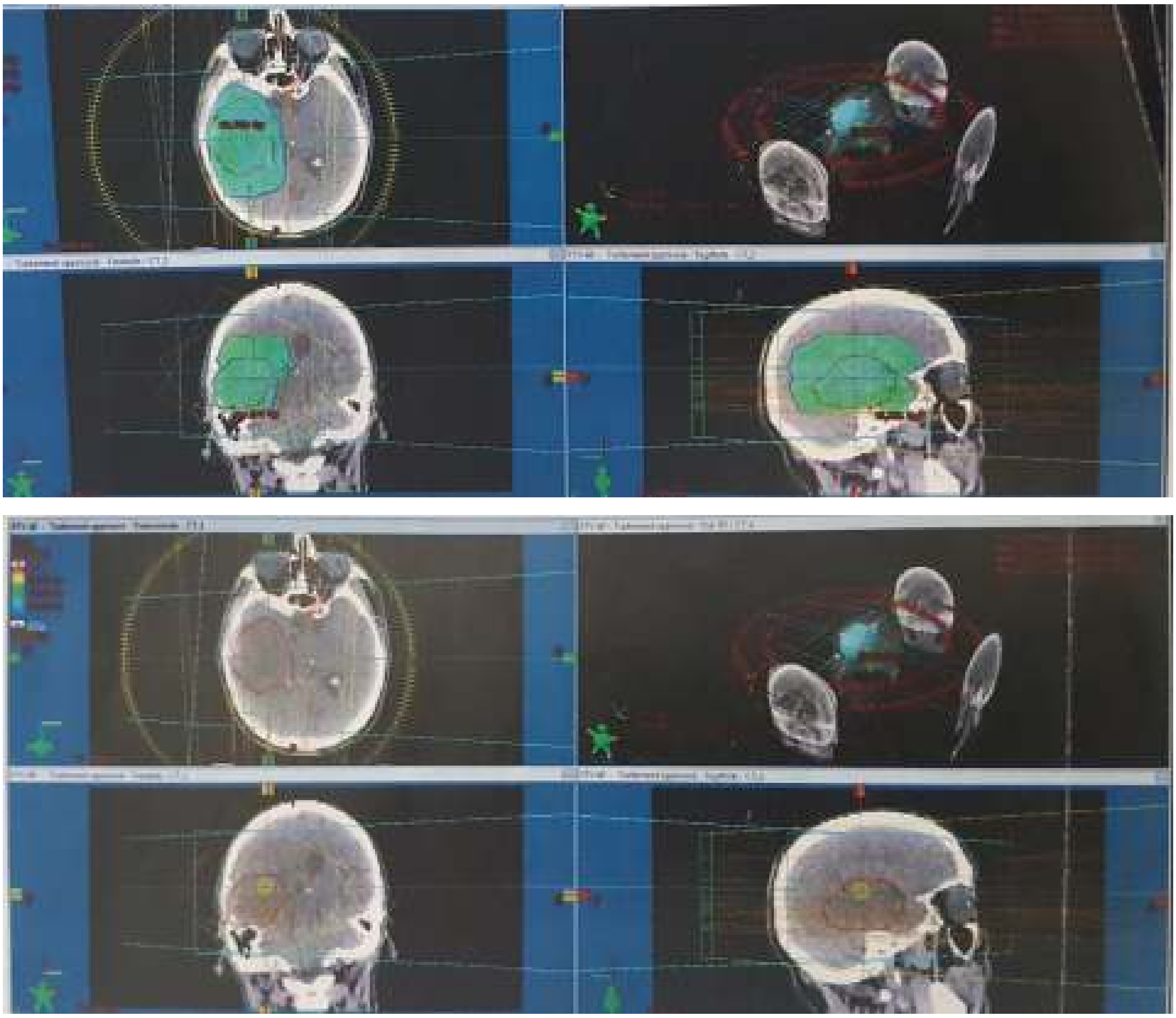


Figure 29-30: radiotherapy planification and target for a 78 years old patient diagnosed with temporal lobe glioblastoma.

CONCLUSION

Effective treatment in GBM remains one of the most formidable challenges in neuro-oncology. Treatment is multimodal and despite significant advances in diagnostic technology, surgical technique, radiation, chemotherapy and targeted therapy, the prognosis remains poor.

The extent of resection is a powerful prognostic factor and is currently considered the only modifiable factor that may be associated with increased survival. The temporal lobe beside the frontal lobe offer more ability for lobectomy, defined as a supratotal resection, which results in superior Progression-free survival and overall survival without negatively impacting patient performance.

Indeed, this review of the most recent research in the field outpoints the need of prospective study for standardization of the surgical techniques and indications in the management of temporal lobe malignant gliomas. Large-scale research efforts are required to understand the molecular biology of brain tumors and to discover novel therapies. Synergistic multimodal strategies and individualized treatments are likely to be the best approach of these complex tumors to finally improve survival and quality of life of the patients.

References

[1] J. Moliterno Gunel, J. M. Piepmeier, and J. M. Baehring, Eds., *Malignant Brain Tumors*. Cham: Springer International Publishing, 2017.

[2] A. Oslobanu and St. I. Florian, “Anatomic locations in high grade glioma,” *Romanian Neurosurg.*, vol. 29, no. 3, pp. 271–277, Sep. 2015, doi: 10.1515/romneu-2015-0036.

[3] S. Larjavaara *et al.*, “Incidence of gliomas by anatomic location,” *Neuro-Oncol.*, vol. 9, no. 3, pp. 319–325, Jul. 2007, doi: 10.1215/15228517-2007-016.

[4] K. R. Noll, M. Ziu, J. S. Weinberg, and J. S. Wefel, “Neurocognitive functioning in patients with glioma of the left and right temporal lobes,” *J. Neurooncol.*, vol. 128, no. 2, pp. 323–331, Jun. 2016, doi: 10.1007/s11060-016-2114-0.

[5] Department of Neurosurgery, Jordan University Hospital and Medical School, University of Jordan, Amman, Jordan, A. F. Tamimi, M. Juweid, and Department of Radiology and Nuclear Medicine, Jordan University Hospital and Medical School, University of Jordan, Amman, Jordan, “Epidemiology and Outcome of Glioblastoma,” in *Glioblastoma*, Department of Neurosurgery, University Hospitals Leuven, Leuven, Belgium and S. De Vleeschouwer, Eds. Codon Publications, 2017, pp. 143–153.

[6] F. Söderqvist, M. Carlberg, K. Hansson Mild, and L. Hardell, “Childhood brain tumour risk and its association with wireless phones: a commentary,” *Environ. Health*, vol. 10, no. 1, p. 106, Dec. 2011, doi: 10.1186/1476-069X-10-106.

[7] Ll. Morgan, S. Kesari, and D. Davis, “Why children absorb more microwave radiation than adults: The consequences,” *J. Microsc. Ultrastruct.*, vol. 2, no. 4, p. 197, 2014, doi: 10.1016/j.jmau.2014.06.005.

[8] “IARC classifies Radiofrequency Electromagnetic Fields as possibly carcinogenic to humans,” p. 6, 2011.

[9] “Cell Phones and Cancer Risk Fact Sheet - National Cancer Institute.” https://www.cancer.gov/about-cancer/causes-prevention/risk/radiation/cell-phones-fact-sheet (accessed Jul. 25, 2020).

[10] M. Michaelis, P. Baumgarten, M. Mittelbronn, P. H. Driever, H. W. Doerr, and J. Cinatl, “Oncomodulation by human cytomegalovirus: novel clinical findings open new roads,” *Med. Microbiol. Immunol. (Berl.)*, vol. 200, no. 1, pp. 1–5, Feb. 2011, doi: 10.1007/s00430-010-0177-7.

[11] K. Barami, “Oncomodulatory mechanisms of human cytomegalovirus in gliomas,” *J. Clin. Neurosci.*, vol. 17, no. 7, pp. 819–823, Jul. 2010, doi: 10.1016/j.jocn.2009.10.040.

[12] K. Dziurzynski *et al.*, “Consensus on the role of human cytomegalovirus in glioblastoma,” *Neuro-Oncol.*, vol. 14, no. 3, pp. 246–255, Mar. 2012, doi: 10.1093/neuonc/nor227.

[13] B. Kucukyuruk, R. M. Richardson, H. T. Wen, J. C. Fernandez-Miranda, and A. L. Rhoton, “Microsurgical Anatomy of the Temporal Lobe and Its Implications on Temporal Lobe Epilepsy Surgery,” *Epilepsy Res. Treat.*, vol. 2012, pp. 1–17, 2012, doi: 10.1155/2012/769825.

[14] H. McE. Knower, “Atlas and text-book of human anatomy. Volumes I, II and III. By Professor J. Sobotta, of Wurzburg. Edited, with additions, by J. Playfair McMurrich, A.M., Ph.D., Professor of Anatomy at the University of Toronto, Canada. Quarto volumes of a total of 794 pages, containing 850 illustrations, mostly in colors. Philadelphia and London: W. B. Saunders Company, 1906.” *Anat. Rec.*, vol. 2, no. 7, pp. 268–273, Oct. 1908, doi: 10.1002/ar.1090020705.

[15] D. N. P. ANt and L. A. Vignolo, “INTRA- AND INTERHEMISPHERIC PROJECTIONS OF THE PRECENTRAL, PREMOTOR AND ARCUATE AREAS IN THE RHESUS MONKEY,” *Brain Res.*, p. 17, 1971.

[16] M. Catani, D. K. Jones, and D. H. ffytche, “Perisylvian language networks of the human brain,” *Ann. Neurol.*, vol. 57, no. 1, pp. 8–16, Jan. 2005, doi: 10.1002/ana.20319.

[17] M. C. De Lacoste, J. B. Kirkpatrick, and E. D. Ross, “Topography of the Human Corpus Callosum:,” *J. Neuropathol. Exp. Neurol.*, vol. 44, no. 6, pp. 578–591, Nov. 1985, doi: 10.1097/00005072-198511000-00004.

[18] J. A. Kiernan, “Anatomy of the Temporal Lobe,” *Epilepsy Res. Treat.*, vol. 2012, pp. 1–12, 2012, doi: 10.1155/2012/176157.

[19] J. A. Kiernan and M. L. Barr, *Barr’s the human nervous system: an anatomical viewpoint*, 9th ed. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2009.

[20] E. F. Chang, K. P. Raygor, and M. S. Berger, “Contemporary model of language organization: an overview for neurosurgeons,” *J. Neurosurg.*, vol. 122, no. 2, pp. 250–261, Feb. 2015, doi: 10.3171/2014.10.JNS132647.

[21] J. A. Duarte, J. Q. de Araújo e Silva, A. A. Goldani, R. Massuda, and C. S. Gama, “Neurobiological underpinnings of bipolar disorder focusing on findings of diffusion tensor imaging: a systematic review,” *Rev. Bras. Psiquiatr.*, vol. 38, no. 2, pp. 167–175, Mar. 2016, doi: 10.1590/1516-4446-2015-1793.

[22] “Michael E. Sughrue.The Glioma Book. Edition 2019. Thieme. ISPN: 9781626234444.,” .

[23] E. E. Smith and S. M. Kosslyn, *Cognitive psychology: mind and brain*, First edition, Pearson new international edition. Harlow: Pearson, 2014.

[24] “Schacter, Daniel L.; Gilbert, Daniel T.; Wegner, Daniel M. (2010). Psychology (2nd ed.). New York: Worth Publishers. ISBN 9781429237192.”

[25] “Hickok, Gregory; Poeppel, David (May 2007). ‘The Cortical Organization of Speech Processing’. Nature Reviews Neuroscience. 8 (5): 393–402. doi:10.1038/nrn2113.” .

[26] “Ruiz Mitjana, Laura. ‘Lóbulo temporal: anatomía, funciones y características’ [Temporal lobe: anatomy, functions and characteristics]. MedSalud (in Spanish).” .

[27] F. Hanif, K. Muzaffar, kahkashan Perveen, S. Malhi, and S. Simjee, “Glioblastoma Multiforme: A Review of its Epidemiology and Pathogenesis through Clinical Presentation and Treatment,” *Asian Pac. J. Cancer Prev.*, vol. 18, no. 1, Jan. 2017, doi: 10.22034/APJCP.2017.18.1.3.

[28] “Xu W, et al. Oncometabolite 2-hydroxyglutarate is a competitive inhibitor of alpha-ketoglutarate- dependent dioxygenases. Cancer Cell. 2011;19 (1):17–30.” .

[29] “Jenkins RB, et al. A t(1;19)(q10;p10) mediates the combined deletions of 1p and 19q and predicts a better prognosis of patients with oligodendroglioma. Cancer Res. 2006;66(20):9852–61.” .

[30] “Esteller M, et al. Inactivation of the DNA-repair gene MGMT and the clinical response of gliomas to alkylating agents. N Engl J Med. 2000;343 (19):1350–4.” .

[31] “Pelloski CE, et al. Epidermal growth factor receptor variant III status defines clinically distinct subtypes of glioblastoma. J Clin Oncol. 2007;25(16):2288–94.” .

[32] “Albert NL, Weller M, Suchorska B, Galldiks N, Soffietti R, Kim MM, et al. Response Assessment in Neuro-Oncology (RANO) Working Group and European Association for Neuro-Oncology (EANO) recommendations for the clinical use of PET imaging in gliomas. Neuro Oncol. 2016.” .

[33] “Breier JI, et al. Language dysfunction after stroke and damage to white matter tracts evaluated using diffusion tensor imaging. AJNR Am J Neuroradiol. 2008;29(3):483–7.” .

[34] “Catani M, Thiebaut de Schotten M. A diffusion tensor imaging tractography atlas for virtual in vivo dissections. Cortex. 2008;44(8):1105–32.” .

[35] “Romano A, et al. Pre- and intraoperative tracto- graphic evaluation of corticospinal tract shift. Neu- rosurgery. 2011;69(3):696–704; discussion 704–5.” .

[36] “Hajiabadi M, Samii M, Fahlbusch R. A preliminary study of the clinical application of optic pathway diffusion tensor tractography in suprasellar tumor surgery: preoperative, intraoperative, and postopera- tive assessment. J Neurosurg. 2016;125(3):759–65.” .

[37] “Biswal BB, Kannurpatti SS, Rypma B. Hemody- namic scaling of fMRI-BOLD signal: validation of low-frequency spectral amplitude as a scalability factor. Magn Reson Imaging. 2007;25(10):1358–69.” .

[38] “Biswal B, et al. Functional connectivity in the motor cortex of resting human brain using echo-planar MRI. Magn Reson Med. 1995;34(4):537–41.” .

[39] “Einevoll GT, et al. Modelling and analysis of local field potentials for studying the function of cortical circuits. Nat Rev Neurosci. 2013;14(11):770–85.” .

[40] “Dym RJ, et al. Is functional MR imaging assessment of hemispheric language dominance as good as the Wada test? A meta-analysis. Radiology. 2011;261 (2):446–55.” .

[41] M. Czosnyka, J. D. Pickard, and L. A. Steiner, “Principles of intracranial pressure monitoring and treatment,” in *Handbook of Clinical Neurology*, vol. 140, Elsevier, 2017, pp. 67–89.

[42] E. C. A. Kaal and C. J. Vecht, “The management of brain edema in brain tumors:,” *Curr. Opin. Oncol.*, vol. 16, no. 6, pp. 593–600, Nov. 2004, doi: 10.1097/01.cco.0000142076.52721.b3.

[43] C. W. Bazil, MD, PhD and T. A. Pedley, MD, “ADVANCES IN THE MEDICAL TREATMENT OF EPILEPSY,” *Annu. Rev. Med.*, vol. 49, no. 1, pp. 135–162, Feb. 1998, doi: 10.1146/annurev.med.49.1.135.

[44] C. Mirian, M. Møller Pedersen, A. Sabers, and T. Mathiesen, “Antiepileptic drugs as prophylaxis for de novo brain tumour-related epilepsy after craniotomy: a systematic review and meta-analysis of harm and benefits,” *J. Neurol. Neurosurg. Psychiatry*, vol. 90, no. 5, pp. 599–607, May 2019, doi: 10.1136/jnnp-2018-319609.

[45] “ET Sayegh, S Fakurnejad, Oh T, O Bloch, AT Parsa: Anticonvulsant prophylaxis for brain tumor surgery: determining the current best available evidence. J Neurosurg 121:1139–1147, 2014.” .

[46] “11-temporal-lobe-gliomas-2019.pdf.” .

[47] J. C. Marsh, J. V. Turian, A. M. Herskovic, J. A. Wendt, R. Godbole, and R. W. Byrne, “Temporal Lobectomy for High Grade Gliomas: Impact on Outcomes and Implications for Postoperative Radiation Treatment Field Design,” *Cancer Clin. Oncol.*, vol. 2, no. 1, p. p5, Nov. 2012, doi: 10.5539/cco.v2n1p5.

[48] “Orringer DADA. Neuronavigation in the surgical management of brain tumors: current and future trends. Expert Rev Med Devices. 2012;9(5):491– 500.” .

[49] “Catalano O, Nunziata A, Siani A. Fundamentals in oncologic ultrasound: sonographic imaging and intervention in the cancer patient. Italy: Springer-Verlag; 2009.” .

[50] “Ivanov MM. Intraoperative ultrasound in neuro- surgery—a practical guide. Br J Neurosurg. 2010;24(5):510–7.” .

[51] “Nimsky CC. Intraoperative high-field-strength MR imaging: implementation and experience in 200 patients. Radiology. 2004;233(1):67–78.” .

[52] R. B. Schwartz *et al.*, “Intraoperative MR Imaging Guidance for Intracranial Neurosurgery: Experience with the First 200 Cases,” *Radiology*, vol. 211, no. 2, pp. 477–488, May 1999, doi: 10.1148/radiology.211.2.r99ma26477.

[53] C. Senft, A. Bink, K. Franz, H. Vatter, T. Gasser, and V. Seifert, “Intraoperative MRI guidance and extent of resection in glioma surgery: a randomised, controlled trial,” *Lancet Oncol.*, vol. 12, no. 11, pp. 997–1003, Oct. 2011, doi: 10.1016/S1470-2045(11)70196-6.

[54] “Weller M, van den Bent M, Hopkins K, Tonn JC, Stupp R, Falini A, et al. EANO guideline for the diagnosis and treatment of anaplastic gliomas and glioblastoma. Lancet Oncol. 2014;15(9):e395–403.” .

[55] “Wick W, Platten M, Meisner C, Felsberg J, Tabatabai G, Simon M, et al. Temozolomide chemotherapy alone versus radiotherapy alone for malignant astrocytoma in the elderly: the NOA-08 randomised, phase 3 trial. Lancet Oncol. 2012;13 (7):707–15.” .

[56] L. Khan, H. Soliman, A. Sahgal, J. Perry, W. Xu, and M. N. Tsao, “External beam radiation dose escalation for high grade glioma,” *Cochrane Database Syst. Rev.*, Aug. 2016, doi: 10.1002/14651858.CD011475.pub2.

[57] “NCCN Clinical Practice Guidelines in Oncology: Central Nervous System Cancers--V. 1.2019. National Comprehensive Cancer Network. Available at https://www.nccn.org/professionals/physician_gls/pdf/cns.pdf. March 5, 2019; Accessed: June 15, 2019.,” .

[58] “TH Roh, S-G Kang,JH Moon, KS Sung, HH Park, SH Kim, EH Kim, JH Chang. Survival benefit of lobectomy over gross-total resection without lobectomy in cases of glioblastoma in the noneloquent area: a retrospective study. doi.org/10.3171/2018.12.JNS182558.”

[59] J. A. Schwartzbaum, J. L. Fisher, K. D. Aldape, and M. Wrensch, “Epidemiology and molecular pathology of glioma,” *Nat. Clin. Pract. Neurol.*, vol. 2, no. 9, pp. 494–503, Sep. 2006, doi: 10.1038/ncpneuro0289.

[60] N. Sanai, S. Chang, and M. S. Berger, “Low-grade gliomas in adults: A review,” *J. Neurosurg.*, pp. 1–18, Aug. 2011, doi: 10.3171/2011.7.JNS10238.

[61] E. A. Maher, “Malignant glioma: genetics and biology of a grave matter,” *Genes Dev.*, vol. 15, no. 11, pp. 1311–1333, Jun. 2001, doi: 10.1101/gad.891601.

[62] F. E. Bleeker, R. J. Molenaar, and S. Leenstra, “Recent advances in the molecular understanding of glioblastoma,” *J. Neurooncol.*, vol. 108, no. 1, pp. 11–27, May 2012, doi: 10.1007/s11060-011-0793-0.

[63] D. Jayamanne *et al.*, “Predicting patterns of failure in temporal lobe GBMs: possible implications on radiotherapy treatment portals,” *Radiat. Oncol.*, vol. 13, no. 1, p. 133, Dec. 2018, doi: 10.1186/s13014-018-1078-y.

[64] D. Kuhnt, A. Becker, O. Ganslandt, M. Bauer, M. Buchfelder, and C. Nimsky, “Correlation of the extent of tumor volume resection and patient survival in surgery of glioblastoma multiforme with high-field intraoperative MRI guidance,” *Neuro-Oncol.*, vol. 13, no. 12, pp. 1339–1348, Dec. 2011, doi: 10.1093/neuonc/nor133.

ABSTRACT

Title: malignant temporal lobe gliomas
Author: Talouni Aymene
Thesis director: Pr Abad Cherif el Assri
Keywords: Malignant gliomas, temporal lobe, surgery, radiotherapy.

Introduction: Gliomas are the most frequent intrinsic brain tumor type in adults and are characterized by diffuse and invasive growth. Malignant gliomas of the histological grades 3 and 4 according to the current WHO classification consist of highly proliferative and migratory tumor cells with a variety of genetic alterations. In adults, It occur most often in the cerebral hemispheres, with the temporal lobe being the second most commonly affected region right behind the frontal lobe. The temporal lobe glioma is particularly interesting due to its unique accessible location, which may provide more valuable information about risk factors, anatomical relationships and the efficiency of the multidisciplinary treatments and approaches.

Methods: In this work, we present some illustrative cases of temporal lobe malignant gliomas that were treated in our department. Therefore, we review the more recent literature's update in managing high grade gliomas in this location in the aim of addressing all the aspects of patient evaluation and care.

Discussion: We discuss the epidemiology, histology and imaging's advances for central nervous malignant gliomas, and specifically in the temporal lobe location which provide a better understanding of the extent of the lesion as well as its relationship with critical neuroanatomic function. We focus on the evolution of intraoperative imaging, functional brain mapping, and technology and how it participate to identify tumor from brain and how it has significantly improved the ability of surgeons for safer and more aggressive tumor removal.

Conclusion: a comprehensive overview of the state-of-the-art treatment for temporal lobe malignant gliomas will prove useful by updating physicians on new therapeutic paradigms and what is on the horizon for the near future. This study will be informative for surgeons, oncologists, neurologists, residents, and students who treat these patients and those who are training for a career in managing patients with these challenging tumors in such location.

RESUME

Titre : les gliomes malins du lobe temporal
Auteur : Talouni Aymene
Directeur de la thèse : Pr Abad Cherif El Assri
Mots clés : gliomes malins, lobe temporal, chirurgie, radiothérapie.

Introduction: Les gliomes sont les tumeurs cérébrales intrinsèques les plus fréquents chez l'adulte et se caractérisent par une croissance diffuse et invasive. Les gliomes malins de grades histologiques 3 et 4 selon la classification actuelle de l'OMS se composent de cellules tumorales hautement prolifératives et migratrices avec diverses altérations génétiques. Chez l'adulte, elle survient le plus souvent dans les hémisphères cérébraux, le lobe temporal étant la deuxième région la plus fréquemment touchée juste derrière le lobe frontal. Le gliome du lobe temporal est particulièrement intéressant en raison de sa localisation unique accessible, qui peut fournir des informations plus précieuses sur les facteurs de risque, les relations anatomiques et l'efficacité des traitements et des approches multidisciplinaires.

Méthodes : Dans ce travail, nous présentons quelques cas illustratifs de gliomes malins du lobe temporal qui ont été traités dans notre service. Nous réalisons une revue de la mise à jour la plus récente de la littérature sur la prise en charge des gliomes de haut grade dans cette localisation. Le but serait de traiter tous les aspects de l'évaluation et des soins des patients présentant un gliome temporal de haut grade.

Discussion: Nous discutons les progrès de l'épidémiologie, de l'histologie et de l'imagerie des gliomes malins cérébraux, et plus particulièrement dans la localisation du lobe temporal. Nous focalisant notre discussion sur l'évolution de l'imagerie peropératoire, de la cartographie fonctionnelle du cerveau, de la chirurgie éveillée et sur la façon dont elle a considérablement amélioré la capacité des chirurgiens à réaliser des exérèses tumorales de manière plus sûre et plus agressive afin de garantir un meilleur pronostic.

Conclusion: un aperçu complet de l'état de l'art du traitement des gliomes malins du lobe temporal s'avérera utile en informant les médecins des nouveaux paradigmes thérapeutiques et de ce qui se profile à l'horizon dans un proche avenir. Cette étude sera informative pour les chirurgiens, les oncologues, les neurologues, les résidents et les étudiants qui traitent ces patients et ceux qui se forment pour une carrière dans la gestion des patients atteints de ces tumeurs difficiles dans un tel endroit.

ملخص

العنوان: الأورام الدبقية الخبيثة في الفص الصدغي.

الكاتب: تالوني أيمن

المشرف: الأستاذ عباد شريف العسري

الكلمات الأساسية: الأورام الدبقية الخبيثة ، الفص الصدغي ، الجراحة ، العلاج الإشعاعي

مقدمة: الأورام الدبقية هي أكثر أنواع أورام الدماغ الجوهرية شيوعًا عند البالغين وتتميز بقدرتها الكبيرة على النمو السريع و الانتشار. تتكون الأورام الدبقية الخبيثة من الدرجتين 3 و 4 وفقًا لتصنيف منظمة الصحة العالمية الحالي من خلايا ورمية عالية التكاثر ومهاجرة مع مجموعة متنوعة من التعديلات الجينية. عند البالغين، تحدث في أغلب الأحيان في نصفي الكرة المخية ، حيث يكون الفص الصدغي ثاني أكثر المناطق المصابة شيوعًا خلف الفص الأمامي مباشرةً. يعتبر الورم الدبقي الصدغي مثيرًا للاهتمام بشكل خاص نظرًا لموقعه الفريد الذي يمكن الوصول إليه، والذي قد يوفر معلومات أكثر قيمة حول العوامل المساهمة في تطوره والعلاقات التشريحية وكفاءة العلاجات والأساليب متعددة التخصصات.

الطريقة: في هذا العمل، نقدم بعض الحالات التوضيحية للأورام الدبقية الخبيثة في الفص الصدغي التي تم علاجها في قسمنا. وبذلك، نقوم بمراجعة أحدث الأساليب للتعامل مع الأورام الدبقية عالية الدرجة في هذا الموقع بهدف تقييم المريض ورعايته.

المناقشة: نناقش تطورات الإحصائيات المرضية و علم الأنسجة والتصوير للأورام الدبقية الخبيثة العصبية المركزية ، وتحديداً في موقع الفص الصدغي الذي يوفر فهمًا أفضل لمدى الآفة بالإضافة إلى علاقتها بالوظيفة التشريحية العصبية الحرجة. نحن نركز على تطور التصوير أثناء الجراحة ، ورسم الخرائط الوظيفية للدماغ ، وكيفية مشاركتها في تحديد الورم من الدماغ وكيف أنها حسنت بشكل كبير قدرة الجراحين على إزالة الورم بشكل أكثر أمانًا وأكثر فاعلية.

الخلاصة: إن نظرة عامة شاملة على أحدث طرق العلاج للأورام الدبقية الخبيثة في الفص الصدغي ستثبت أنها مفيدة من خلال إطلاع الأطباء على النماذج العلاجية الجديدة وما هو في الأفق في المستقبل القريب. ستكون هذه الدراسة مفيدة للجراحين وأطباء الأورام وأطباء الأعصاب والمقيمين والطلاب الذين يعالجون هؤلاء المرضى.

Serment d'Hippocrate

Au moment d'être admis à devenir membre de la profession médicale, je m'engage solennellement à consacrer ma vie au service de l'humanité.

- *Je traiterai mes maîtres avec le respect et la reconnaissance qui leur sont dus.*
- *Je pratiquerai ma profession avec conscience et dignité. La santé de mes malades sera mon premier but.*
- *Je ne trahirai pas les secrets qui me seront confiés.*
- *Je maintiendrai par tous les moyens en mon pouvoir l'honneur et les nobles traditions de la profession médicale.*
- *Les médecins seront mes frères.*
- *Aucune considération de religion, de nationalité, de race, aucune considération politique et sociale ne s'interposera entre mon devoir et mon patient.*
- *Je maintiendrai le respect de la vie humaine dès la conception.*
- *Même sous la menace, je n'userai pas de mes connaissances médicales d'une façon contraire aux lois de l'humanité.*

Je m'y engage librement et sur mon honneur.

قسم ابقراط

بسم الله الرحمان الرحيم

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

المملكة المغربية
جامعة محمد الخامس بالرباط
كلية الطب والصيدلة
الرباط

سنة: 2020 **أطروحة رقم: 313**

أطروحة: *الأوراق الدبقية الخبيثة في الفص الصدغي*

قدمت ونوقشت علانية يوم..

من طرف

السيد (ة): تالوني أيمن

المزداد (ة): 26/08/1994

من المدرسة الملكية لمصلحة الصحة العسكرية – الرباط

لنيل شهادة دكتور في الطب

الكلمات الأساسية : الأورام الدبقية الخبيثة ، الفص الصدغي ، الجراحة ، العلاج الإشعاعي

تحت إشراف اللجنة المكونة من الأساتذة:

السيد: ميلود كزاز
أستاذ في جراحة الأعصاب **رئيس**
السيد: شريف العسري عباد
أستاذ في جراحة الأعصاب **مشرف**
السيد: عادل ملحاوي
أستاذ في جراحة الأعصاب **عضو**
السيد: حسن سفات
أستاذ في العلاج الإشعاعي **عضو**
السيد: حسن النوالي
أستاذ في الأشعة **عضو**