ROYAUME DU MAROC
Université Mohammed V - Rabat
Faculté de Médecine et de Pharmacie
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MEMORY OF END STUDY

For obtaining

Speciality Diploma of **Medical Imaging**

entitled

COMPUTED TOMOGRAPHY OF CAUSTIC INGESTION : THE EXPEREINCE OF DEPARTEMENT OF RADIOLOGY EMERGENCY (ABOUT 28 CASES)

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THANKS

To my master and supervisor of

memory Pr Laamrani Fatima zahrae

Professor of Radiology in the Radiology Department of emergency

in CHU ibn Sina Rabat

You have done me a great honor by agreeing to guide

me in carrying out this work.

During my training, I had the privilege of benefiting from your knowledge,

and of immersing myself in your human qualities worthy of consideration.

Thank you for your patience, availability, and above all for your judicious

advice, which has helped fuel my reflection.

Please, dear master, find in this work the expression of my gratitude,

esteem, and deep respect.

To our master

Professor JROUNDI LAILA

Professor of Radiology in the Radiology Department of emergency

in CHU ibn Sina Rabat

We thank you for your unparalleled spontaneity
and kindness, as well as your precious help and guidance. You have
combined benevolence and kindness to inspire us and
be the subject of great admiration.

List of illustrations

LISTE OF FIGURE

LISTE OF TABLE

Summary

SUMMARY

Introduction

Caustic burns of the gastrointestinal tract are a frequent emergency condition in adults. In most cases, they are benign, but also can lead to serious damage and may cause death.

If the endoscopy is still the most important tool for diagnosing, treating, and monitoring caustic injuries, computed tomography is always an available option to assess the severity of lesions and to guide management and all that in a few minutes.

This condition calls for immediate and multidisciplinary management from emergency physicians, resuscitators, gastroenterologists, radiologists, visceral surgeons, and psychiatrists.

Multiple studies in the literature try to establish the position of imaging within the management of caustic ingestions are emerging.

We report a series of accumulated cases from the CHU IBN SINA to find the exact radiological endoscopic findings and to establish the right indications and advantages of computed tomography in the management of caustic ingestion victims

Materials and methods

I.DATA COLLECTION:

- The clinical, operative and endoscopic data of the patients were recollected within the archives of the visceral surgical emergencies and functional digestive exploration departments at the CHU IBN-SINA hospital.
- The radiological examinations, including images and interpretations, were obtained from the archives of the CHU's emergency radiology department using the PACS network.
- Some additional patient data was found on the GREENCUBE hospital computer network.
- • Documents consulted:
- Register of archives at the department of the visceral surgical emergencies: from 2018 to 2021.
- Records of archives at functional digestive exploration department: from 2018 to 2021.
- CHU-IBN SINA computerized database: GREENCUBE.
- Archives of the Emergency Radiology department of the CHU IBN-SINA and examinations on the PACS network.
- Medical records of patients with clinical observation, specialist opinions, and evolution.
- Operating reports and hospitalizations in intensive care.
- Endoscopic reports.
- Scan reports.
- X-ray pictures “Standard chest X-ray, abdomen without preparation, barium contrast”.
- biology report.

II.STATISTICAL MATERIAL:

- The qualitative variables will be expressed in persons and percentages.
- Quantitative variables will be expressed as mean standard deviation if the distribution is Gaussian or as median quartiles if the distribution is asymmetrical.
- Data processing was performed using SPSS STATISTICS 26 statistical software.
- The Khi-2 test for the qualitative variables was carried out in the form of cross tables. p-value was considered significant for a value less than 0.05.

III.SCANNOGRAPHIC EXPLORATION PROTOCOL:

- The scenographic examination was carried out as part of an initial lesion assessment or for monitoring lesions or complications:
- The scanner used in the Emergency Radiology department was a General Electric with 16 Barrettes.

• Preparation :

- Patient should be reassured and calm if conscious or sedated with conditioning for unstable cases.
- Questioning with the patient and/or the family: if there is any history of allergy to the contrast product or other substances, like oral antidiabetic drugs, kidney function, etc.

- Peripheral venous access with **a** good caliber of 16G/18G/20G depending on the quality of the venous system and the hemodynamic state.
- Hydration before and after examination if renal function is not good or consultation with the nephrologist.

• Protocol:

- Patient in s dorsal decubitus position.
- Connection of the patient to the mobile respirator circuit, if he is intubated with monitoring of vital constants on the mobile scope.
- Acquisition: Cervico-Thoraco-Abdominal or Thoraco-Abdominal.
- First acquisition before injection of contrast medium materiel, we can use digestive opacification using water-soluble iodine contrast product 'Gastrografin®': 100-150cc only if clinically we suspect fistulization.
- Injection of 2cc/Kg of ULTRAVIST 300 mg iodinated contrast medium for 3cc/second using an automatic injector.

  Arterial phase at 25 or 45 seconds: to explore the pancreas.

  Portal phase: at 75 seconds: to assess the enhancement of the esophagogastric wall and neighboring organs (Mediastinum, Liver, spleen, pancreas, mesentery, etc.)

- Monitoring of complications during the examination: Vomiting, skin rash, facial-cervical edema, respiratory distress, extravasation, etc.

Reading :

The analysis was performed by the duty radiologist. A reinterpretation of the images was required by two radiologists, including a senior, based on the score established by MIRCEA CHIRICO modified according to the needs of our study.

IV.ETHICS :

Respect for confidentiality and medical secrecy was essential rules during our study with encryption of personal data by assigning a number to each patient.

V.OPERATING SHEET AND OBSERVATIONS :

FICHE D'EXPLOITATION

IDENTITE :

- NOM :
- AGE :
- SEXE : ☐ FEMININ ☐ MASCULIN
- ADRESSE :
- NUM TEL :
- NUM D'ENTREE : NUM ARCHIVES :

SYMPTOMATOLOGIE :

☐ DOULEUR THORACIQUE ☐ DETRESSE RESPIRATOIRE

☐ DOULEUR ABDOMINALE ☐ ASYMPTOMATIQUE

☐ VOMISSEMENTS ☐ HEMORRAGIE DIGESTIVE

AUTRE :

HISTOIRE DE LA MALADIE : INGESTION DE CAUSTIQUE :

☐ ACCIDENTELLE ☐ VOLONTAIRE

- **HEURES AVANT CONSULTATION :**
- **NATURE :**

FAIBLEMENT CAUSTIQUE : ☐ MOUSSANT ☐ IRRITANT ☐ TRES IRRITANT

CAUSTIQUE MAJEUR : ☐ AC FORT ☐ BASE FORTE ☐ OXYDANT

- **FORME :** ☐ LIQUIDE ☐ SOLIDE ☐ GEL ☐ POUDRE
- **QUANTITE :** « 50ml = 2 gorgées + 150ML= 1 verre »

☐ SUPERIEURE A 150ML ☐ INFERIEURE A 150ML

- **ASSOCIATION :**
- **VOMISSEMENT :**

EXAMEN PARACLINIQUE :

- **RADIO THORACIQUE STANDARD:** ☐ NORMALE ☐ PNEUMOTHORAX

☐ PNEUMOMEDIASTIN ☐ FOYER D'INHALATION ☐ AUTRE :

- **ABDOMEN SANS PREAPARTION :** ☐ NORMALE ☐ CROISSANT GAZEUX

☐ NIVEAUX HYDRO-AERIQUES ☐ AUTRES

- **TOMODENSITOMETRIE :**

☐ Cervico-Thoracique ☐ ABDOMINO-PELVIEN

☐ AVEC INJECTION PDC ☐ SANS INJECTION PDC

RESULTAT :

☐ EPAISSISSEMENT PARIETAL ☐ OEDEME SOUS-MUQUEUX

☐ PNEMATOSE PARIETALE ☐ INFILTRATION DE LA GRAISSE PERI-DIGESTIVE

☐ DEFAUT DE REHAUSSEMENT PAROI DIGESTIVE ☐ PNEUMOPERITOINE

☐ AEROPORTIE ☐ Dilatation œsophagienne ☐ EPANCHEMENT INTRA-PERITONEAL

☐ AMINCISSEMENT DE LA PAROI ☐ PANCREATITE

☐ Autre :

- **BIOLOGIQUE :**

NA			ASAT			HB		RA	
K			ALAT			GB		fibrin	
CL			TROPO			PNN			
Mg			CK			LY			
Ca			LDH			PLT			
PH			LIPASE			TP			
Urée			GLY			TCA			
Créat			CRP			D-D			

- **FIBROSCOPIE OESO-GASTRIQUE :**

☐ OEDEME ☐ EROSION ☐ ULCERE SUPERFICIEL ☐ ULCERE PROFOND

☐ NECROSE FOCALE ☐ NECROSE DIFFUSE ☐ PERFORATION

☐ STADE de ZARGAR :

Œsophage : Fundus : Antre : Corps : Bulbe : Duodénum :

ANTECEDANTS :

PERSONNELS :

- **MEDICAUX :** ☐ DIABETE ☐ HTA ☐ CARDIOPATHIE
  ☐ PATHOLOGIE RESPIRATOIRE ☐ MALADIE DIGESTIVE
  ☐ TROUBLES PSYCHIATRIQUE ☐ AUTRES
- **CHIRURGICAUX :** ☐ CHIRURGIE THORACIQUE ☐ CHIRURGIE CARDIAQUE
  ☐ CHIRURGIE ABDOMINALE ☐ AUTRES
  TYPE : DATE :
- **TOXICO/MEDICAMENTEUX :**

FAMILIAUX :

EXAMEN CLINIQUE A L'ADMISSION :

- **EXAMEN RESPIRATOIRE : :**

☐ INSTABLE ☐ STABLE

- **EXAMEN CARDIOVACULAIRE :**
  ☐ INSTABLE ☐ STABLE

- **EXAMEN NEUROLOGIQUE :**

☐ INCONSCIENT ☐ CONSCIENT

- **EXAMEN ABDOMINALE :**

INSPECTION : ☐ BALLONNEMENT ☐ CICATRICE ☐ AUTRE :

PALPATION : ☐ SENSIBILITE ☐ DEFENSE ☐ CONTRACTURE

PERCUSSION : ☐ TYMPANISME ☐ MATITE

TOUCHE RECTAL : ☐ DOULEUR ☐ MELENA ☐ RECTORRAGIE

- **EXAMEN ORL:**

ORO PHARYNGE: ☐ OEDEME ☐ ULCERE ☐ NECROSE

- **ZONE LESEE :**

☐ ŒSOPHAGE SUPERIEUR ☐ ŒSOPHAGE MOYEN
☐ OESOPHAGE INFERIEUR ☐ FUNDUS
☐ ANTRE ☐ PYLORE ☐ DUODENUM

COMPLICATIONS/ DIAGNOSTIQUES DE GRAVITE:

☐ INGESTION SUPERIEURE A 150ML D'ACIDE OU BASE
☐ ETAT DE CHOC ☐ HEMATEMESES EMPHYSEME SOUS-CUTANE
☐ PERITONITE ☐ MEDIASTINITE ACIDOSE METABOLIQUE
☐ HYPOXIE ☐ TROUBLE PSYCHIQUES
☐ ADMISSION EN REANIMATION ☐ AUTRE :

PRISE EN CHARGE:

☐ LAPAROTOMIE ☐ THORACOTOMIE
☐ GASTRECTOMIE ☐ OESOPHAGECTOMIE ☐ OESOPHAGOPLASTIE
☐ DUODENO-PANCREATECTOMIE CEPHALIQUE ☐ SPLENECTOMIE
☐ JEJUNOSTOMIE ☐ OESPHAGOSTOMIE CERVICALE ☐ DILATATION
☐ MEDICALE ☐ PSYCHIATRIQUE ☐ ABSTENTION ET SURVEILLANCE

	Estimated quantity	Gastric	CT stage	CT stage esophagus	Endoscopic grade	Endoesophagien stade	stage	Endogastric	Reanimation	Jejunostomy	Surgical exploration		Stenosis	Death
N°1	41/H	A	Ac Fort	225	IIb	IIb	IIa	3a	2b	3a				
N°2	35/F	V	Ac Fort	150	IIa	I	IIa	3a	2a	3a				
N°3	23/F	V	Ac Fort	450	IIa	I	IIa	3a	3a	2a	oui	oui		oui	oui
N°4	71/F	V	Ac Fort	150	IIb	IIb	IIb	3a	2b	3a				oui	
N°5	23/H	V	Ac Fort	150	IIb	IIb	IIa	2b	2b	2b		oui		oui	
N°6	27/F	V	-	150	III	III	IIa	2b	2b	2a		oui		oui	
N°7	22/H	V	Ac Fort	150	IIb	IIa	IIb	3b	2a	3b		oui		oui	
N°8	40/H	A	Ac Fort	40	I	I	I	2b	2b	2a					
N°9	22/F	V	Base faible	150	I	I	I	2a	2a	1					
N°10	19/F	A	Oxydant	150	I	I	I	2a	2a	2a					
N°11	56/F	V	Ac Fort	150	IIa	I	IIa	2b	2a	2b					
N°12	18/H	-	Ac Fort	150	IIb	IIa	IIb	2b	2a	2b					
N°13	27/H	V	Ac Fort	450	III	III	III	3a	3a	3a	oui	oui			
N°14	41/H	V	Ac Fort	150	IIa	IIa	IIa	2b	2b	2a				oui	
N°15	26/H	V	Ac Fort	150	III	III	III	3b	3b	3b				oui	
N°16	39/F	V	Ac Fort	75	IIb	IIb	IIb	3b	2b	3b	oui				oui
N°17	42/H	V	Ac Fort	150	III	III	III	2b	2b	-				oui	
N°18	60/H	-	Ac Fort	300	III	IIb	III	3b	2b	3b				oui	oui
N°19	21/F	V	Ac Fort	300	IIa	IIa	IIa	3a	2b	3a				oui	
N°20	53/H	V	Ac Fort	300	IV	IV	IIb	-	-	-				-	oui
N°21	26/H	V	-	150	I	I	I	2b	2a	2b					
N°22	31/H	V	Ac Fort	300	I	I	I	3a	2b	3a				oui	
N°23	65/H	-	Ac Fort	300	-	-	-	3b	3b	3b	oui	oui	oui	oui	
N°24	44/H	V	Ac Fort	300	IIb	IIa	IIb	3b	2b	3b	oui		oui	oui	oui
N°25	53/H	V	Ac Fort	450	IV	IV	III	-	-	-	oui	oui	oui	-	oui
N°26	77/F	V	Ac Fort	450	IV	III	IV	-	-	-	oui		oui	-	oui
N°27	18/F	-	Ac Fort	-	IV	III	IV	-	-	-	oui		oui	-	oui
N°28	18/H	V	Ac Fort	150	IIb	IIb	IIa	3a	3a	3a				oui	

VI.CLASSIFICATIONS :

- Endoscopic classification :

the Endoscopic interpretation was based on the ZARGAR score [1] :

- **Grade 0:** Normal mucosa.
- **Grade 1:** Edema or erythema of the mucosa.
- **Grade 2 a:** hemorrhage, erosions, blisters, superficial ulcers
- **Grade 2b:** circumferential lesions
- **Grade 3a:** focal deep grey or brownish-black ulcers
- **Grade 4:** perforation
- Radiologic Classification:

Scan images were intepratated based in Bruzzi and Mircea Chirica classification [2] :

- - GRADE I: Normal

  - Regular parietal surface, without submucosal edema.
  - Homogeneous parietal enhancement.
  - Absence of mediastinal or peritoneal fat infiltration.
  - No signs of perforation or collection.

- GRADE II: submucosal parietal edema with parietal enhancement, may be associated with peri-digestive fat infiltration: divided into two subgroups:

- • IIa: Submucosal edema is limited by mucosal enhancement and of the outer layer “target aspect”. Without infiltration of peri-digestive fat.

- IIb: Submucosal edema with the enhancement of the external layer without enhancement of the mucosa due to necrosis + infiltration of peri-digestive fat.
- GRADE III: Transmural necrosis: Defined by the absence of enhancement of the wall with infiltration of peri-digestive fat
- GRADE IV: Presence of a complication:

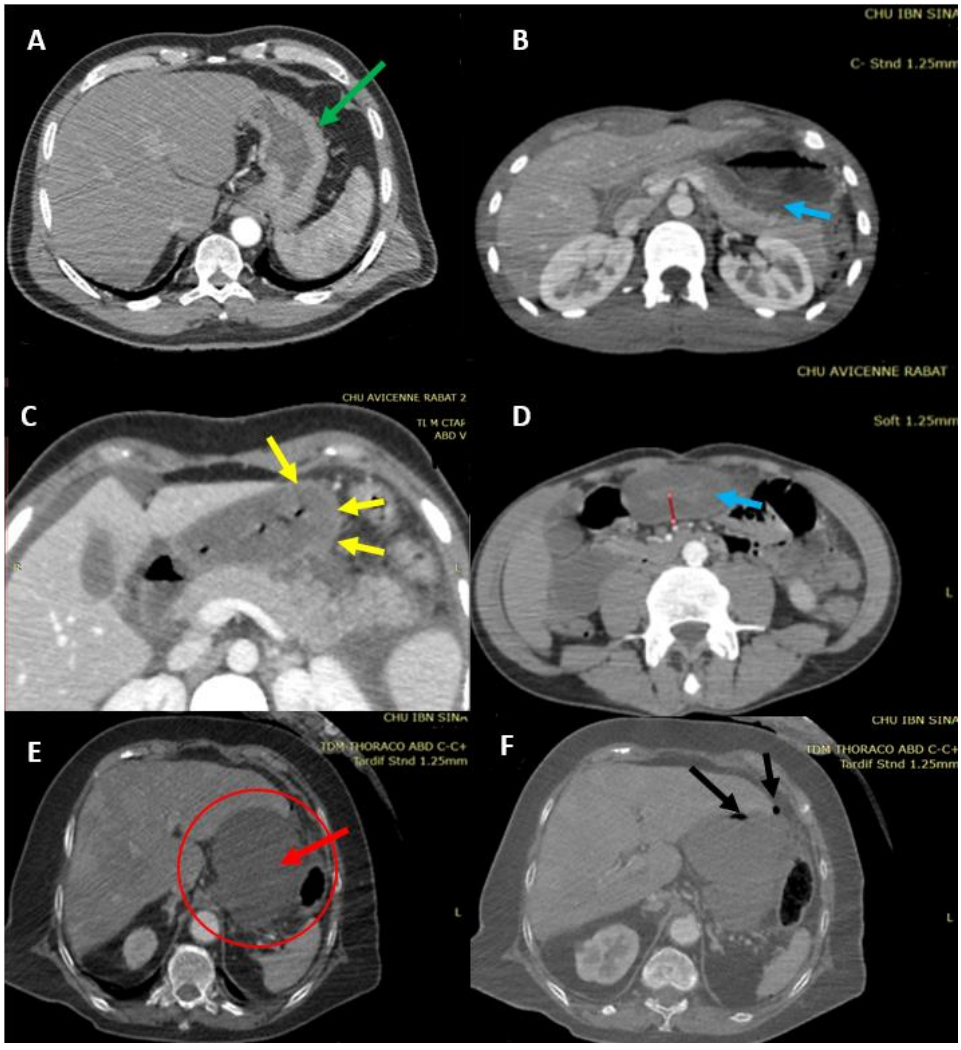


Figure 1: Axial scannographic images of patients having ingested caustic agents from our study, summarizing the radiological classification.

- Digestive perforation
  - Signs of peritonitis: pneumoperitoneum, peritoneal effusion, and Infiltration of mesenteric fat, solution of digestive continuity, peritoneal collection.
  - Signs of Mediastinitis: Pneumo-mediastinum, Mediastinal or pleuropericardial effusion, Infiltration of mediastinal fat, solution of continuity of the esophageal wall, mediastinal collection.
- Oeso-tracheal fistula.

Results

I. EPIDEMIOLOGIA :

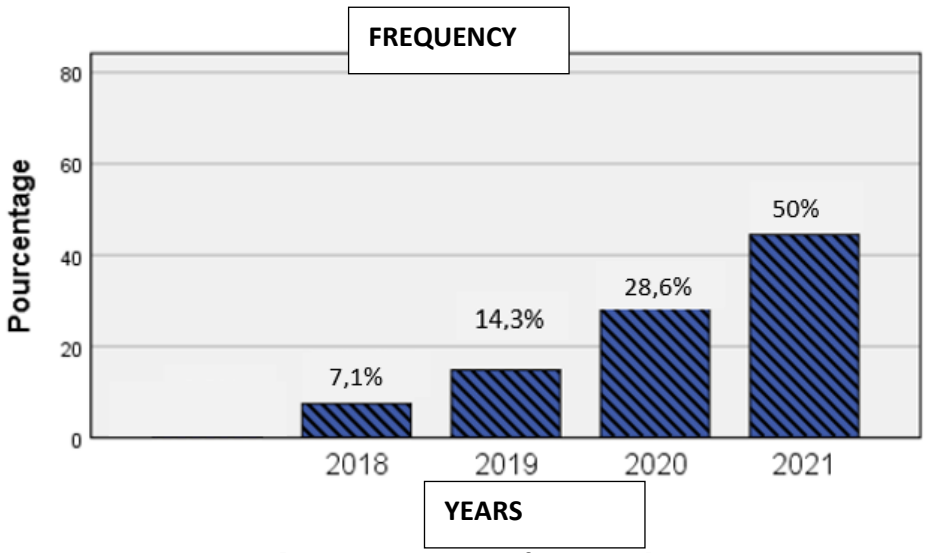
1. Frequency :

During the 3 years over which our study is spread, from September 6, 2018, to November 20, 2021, the annual average of caustic ingestions was 15 patients. About 65 patients were hospitalized in the emergency department of the CHU IBN SINA, but only 28 patients had inclusion criteria.

We note the increase in cases in 2021, concomitant with the period of SARS COV 2 « Covid19 » Virus pandemic and the period of lockdown.

FREQUENCY			
		Cases	Pourcentage in %
Year	2018	2	7,1
	2019	4	14,3
	2020	8	28,6
	2021	14	50
	Total	28	100,0

Table 1 : Frequency of patients included in our study by year.



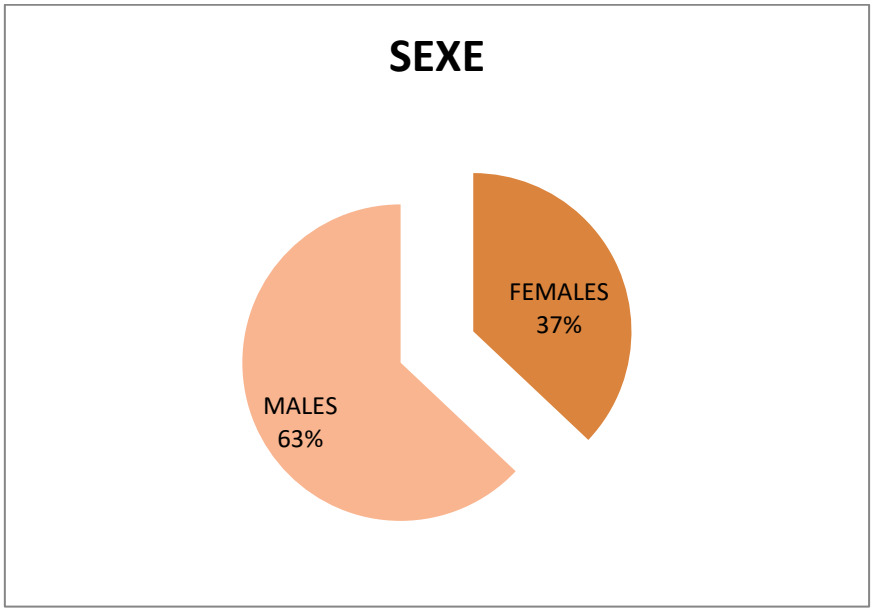
Graphic 1: Frequency of patients by year

2. Sexe :

A male predominance was noticed in our series of cases, with 17 men for 11 women, respectively 60.7% and 39.3% with a ratio of M/F = 1.5.

SEXE FREQUENCY			
		Cases	Percentage (%)
SEXE	Female	11	39,3
	Male	17	60,7
	Total	28	100,0

Table 2 : Fréquency of patients by sexe.



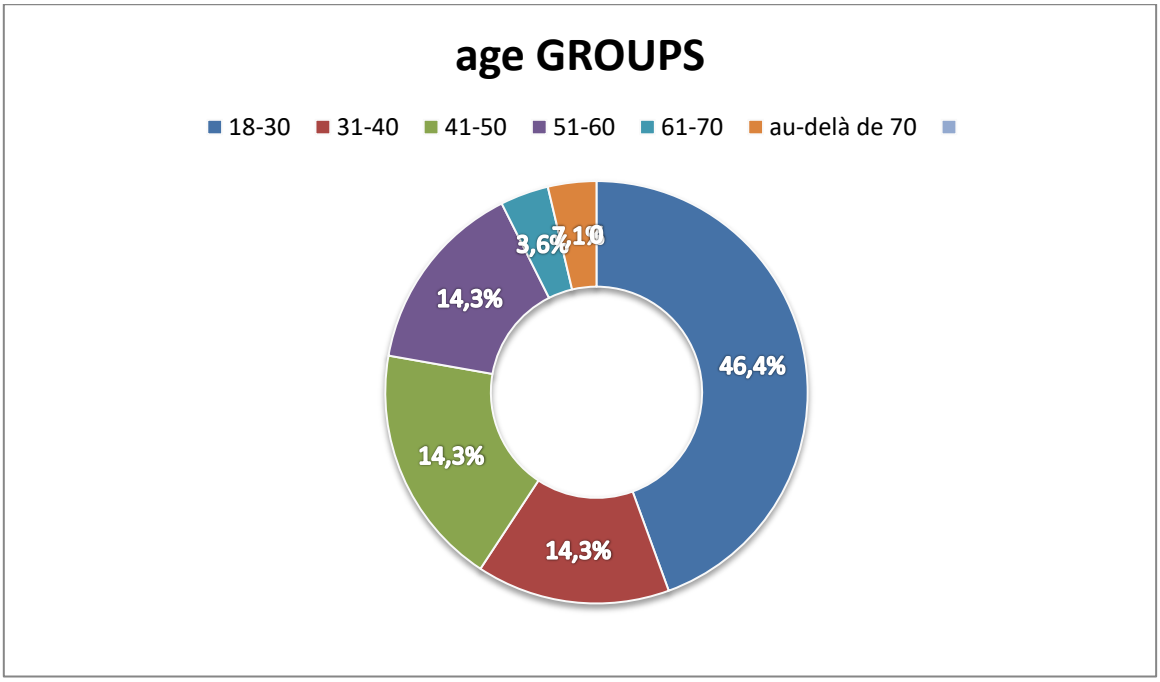
Graphic 2: Frequency of patients by their sexe.

3. Age :

The most concerned patients according to our study are the young age group between 18 and 30 years old. Indeed, this represents 46.4% of our studied population, with a medium age of 33 years and extreme ages of 18 and 77 years.

AGE GROUPS	Frequency	Pourcentage (%)
18-30	12	46,4
31-40	4	14,3
41-50	4	14,3
51-60	4	14,3
61-70	1	3,6
More than 70	1	7,1

Table 3: Frequency of patients by their age groups.



Graphic 3: Frequency by age groups

II. HISTORY:

1. . Background:

In our case series, the antecedents encountered are as follows:

a. Neuropsychiatric history:

- Out of 28 patients, 35.7% have a known **depressive syndrome** followed by a psychiatrist, including 1 patient with a history of sexual abuse with 2 suicide attempts before our episode.
- 10.7% of our patients are followed for documented **psychosis.**
- One patient in our series had **psychomotor retardation**.
- A **dementia syndrome** was found in one of our patients.

b. Toxic habits:

- Smoking is the most common toxic history in our population with 39.2% of patients.
- Addiction, including the use of drugs such as cannabis or cocaine, was reported in 17.8% of our patients.
- 5 out of 28 cases were alcohol consumers.

c. Cardiovascular risk factors:

- Diabetes was encountered in 4 of our patients, while hypertension in 5 patients.
- Heart disease is found in 2 out of 28 patients.

d. Surgical history:

- None of our patients had a history of surgery.

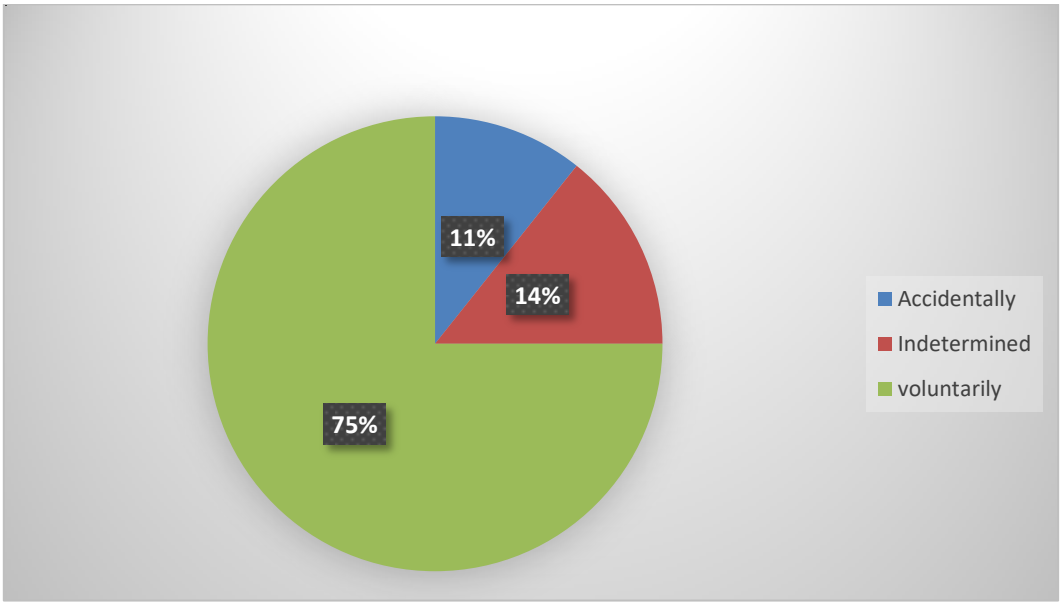
e. Others :

- Several other antecedents have been described, of varying severity and importance, such as a case of epileptogenic cavernoma and a case of systemic sarcoidosis.
- One of our patients had trisomy 21 with depressive syndrome.

2. Intentionality:

The psychiatric profile is a determining factor of the patient's prognosis and the seriousness of the situation, by understanding the circumstances of the ingestion.

In our study, 21 cases of attempted suicide were described, while 3 ingestions occurred accidentally.



Graphic 4: Frequency of the intentionality of caustic ingestion.

INTENTIONALITY		
	Frequency	Pourcentage (%)
Accidentelly	3	10,7
Indetermined	4	14,3
Voluntarilly	21	75

Table 4: Frequency by the intentionality of caustic ingestion.

3. Type of product:

The most incriminated product is hydrochloric acid in 85.7% of our cases, as a strong acid. Other products were found, such as sodium hydroxide or oxidants. In one patient, a combination of bleach “Sodium hypochlorite” as an oxidant and hydrochloric acid as a strong acid was mentioned.

In some cases, it is difficult to identify the ingested caustic due to the patient’s non-cooperation.

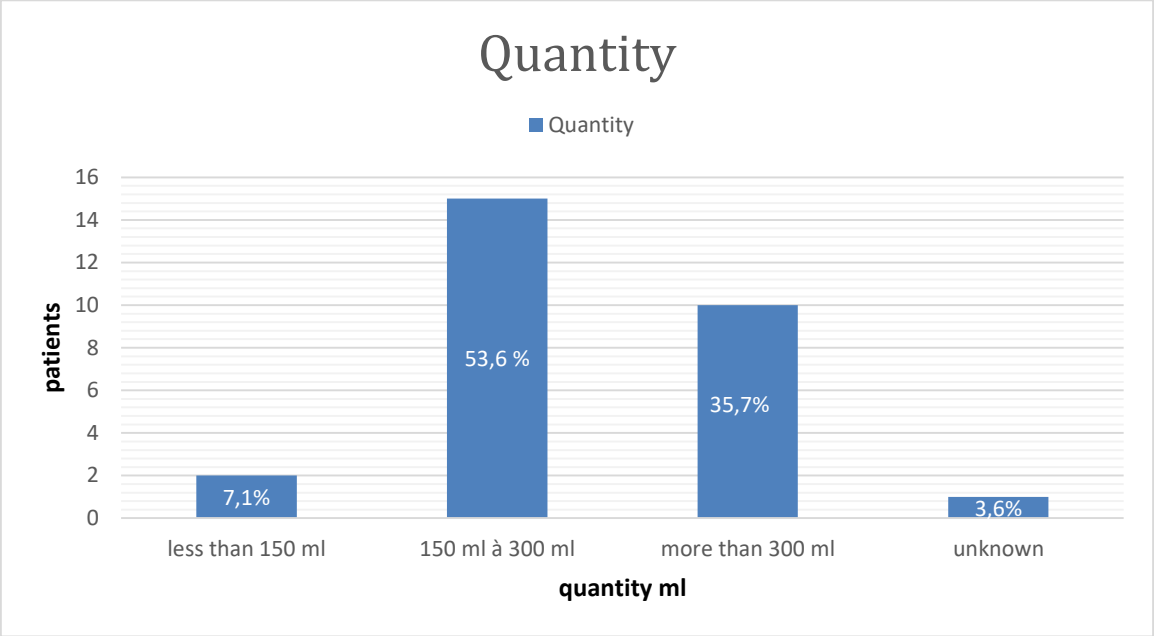
TYPE OF PRODUCT		
	cases	Percentage (%)
hydrochloric acid	24	85,7
sodium hydroxide	1	3,6
Indéterminé	2	7,1
Oxidants	1	3,6
Total	28	100,0

Table 5: Frequency of caustic products by the nature.

4. Quantity ingested:

In the majority of our patients who had recourse to the computed tomography, the quantity ingested was significant, greater than 150 ml, and 15 of them had ingested between 150 and 300ml, i.e. 53.6%, and 10 patients exceeded 300ml of product, i.e. 35 .7% of our population.

In 3.6% of cases, it was difficult to quantify the product ingested.



Graphic 5: Frequency of quantities ingested

Quantity ingested	Frequency	Percentage (%)
Less than 150 ml	2	7,1
150 à 300 ml	15	53,6
More than 300ml	10	35,7
Unknown	1	3,6
Total	28	100,0

Table 6: Frequency of quantities ingested

5. Consultation period:

The majority of our patients arrived in consultation within average times, between 3 to 6 hours of ingestion, representing 17.8% of our study population, and 10.7% arrived at the hospital before the first 3 hours of swallowing the product.

It happened to receive patients after a few days of ingestion with dysphagia, such as the case of 2 patients.

Consultation period	Frequency	Pourcentage %
Less than 3H	3	10,7
3 à 6H	5	17,8
6 à 12H	2	7,1
12à 24 H	1	3,6
24 à 48H	1	3,6
More than 48H	2	7,1
unknown	14	50
Total	27	100,0

Table 7 : Frequency of the consultation period.

To half of our patients, the consultation period was unknown.

III. CLINIC:

1. Stability:

In our case series, 21 patients had no clinical instability, i.e. 75%. 25% had hemodynamic, respiratory, or neurological instability on admission.

Indeed, 5 patients presented a state of shock, 1 of which had associated respiratory distress, against 1 case of neurological instability due to a refractory convulsive status.

A total of 2 patients presented with respiratory distress.

	Frequency	Pourcentage (%)
Hemodynamic Instability	4	14,2
Neurologic Instability	1	3,6
Respiratory Instability	1	3,6
Stable	21	75
Total	27	100,0

Table 8: Frequency of stability status.

2. Functional signs:

a. Vomiting:

Vomiting is the second most frequently encountered sign after epigastric pain, it was reported in 23 patients or 82.1% of our case series.

In 9 patients, vomiting was induced to evacuate the corrosive product, i.e. 36% of the population studied.

b. Hematemesis:

Hematemesis was seen in 6 of our patients, or 21.4% of our series.

c. Pain :

The most common type of pain is epigastric, found in 92.8% of cases, i.e. 26 out of 28 patients.

8 of our patients complained of chest pain, i.e. in 28.5% of cases.

d. Dysphagia:

Dysphagia was found in 9 patients, two of whom consulted the emergency room within a delayed period, 26 hours and 7 days after ingestion of caustic.

In the 6 other patients, who consulted within an average of 5 hours, dysphagia was described as difficulty swallowing, probably due to recent mucosal lesions and not necessarily to stenosis.

e. Hyper sialorrhea:

Hyper sialorrhea was reported in 9 patients, i.e. 32.1%.

3. Clinical examination:

a. Bucco-Pharyngeal Lesions:

In 20 patients or 71.4% of our cases, oropharyngeal lesions were described.

b. Abdominal defense:

Defense is a serious clinical sign, associated with contracture on palpation, raising the suspicion of perforation, such was the case for 5 patients, i.e. 17.8% of the series, 3 of whom presented with peritonitis.

4. Endoscopy :

a. Completion time:

Endoscopy was the first-line exploration, performed within 24 hours after patient admission to the emergency.

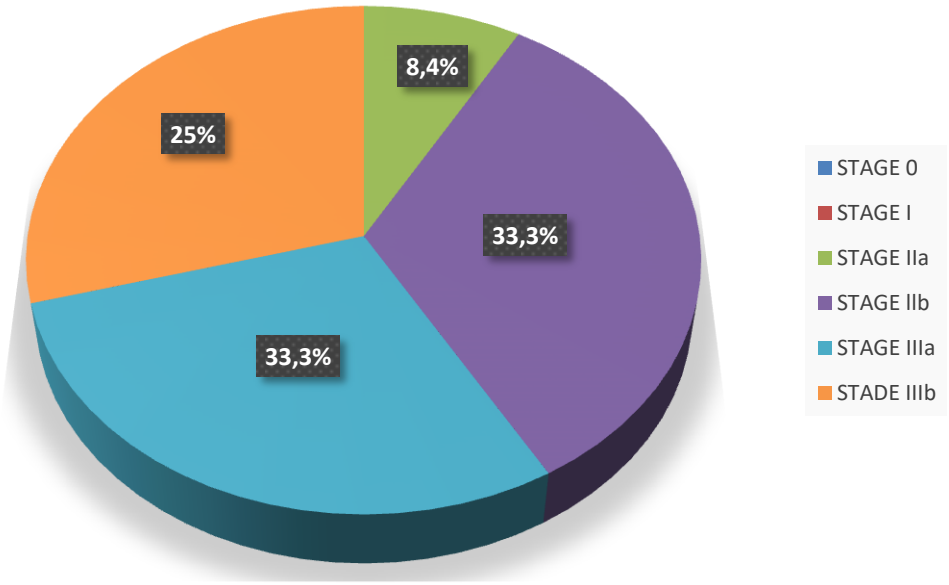
b. Frequency by stage:

Among the 24 patients who underwent endoscopy, 33.3% (N=8) had superficial ulcerations classified as Stage IIb according to ZARGAR, constituting the most frequent stage, followed by stages of necrosis IIIa and IIIb found in 29.2 % each, then stage IIa in 2 patients, i.e. 8.3%.

Stage 0 was not found in any of our patients.

Endoscopic stage of ZARGAR	cases	Frequency (%)
STAGE 0	0	-
STAGE I	0	-
STAGE Iia	2	8,4
STAGE IIb	8	33,3
STAGE IIIa	8	33,3
STAGE IIIb	6	25,0

Table 9: Frequency of endoscopic stages.



Graphic 6: Frequency of endoscopic stages.

5. Involvement according to the site:

In our study, the damage after caustic ingestion was most often extended along the upper digestive tract, involving both the esophagus and the stomach, this condition was found in 83.3% of our patients.

In the esophagus, the most common stage is stage IIb with deep ulcerations in 50% of cases (N=12), followed by stage IIa (N=7) in 29.2%. Esophageal necrosis stages IIIa and IIIb were encountered in respectively 12.5% (N=3) and 8.3% (N=2) of the 24 patients who underwent endoscopy.

While at the gastric level, stage IIIa was most frequently found, in 7 patients, or 29.1%, followed by stage IIIb with 6 patients, or 25%. Stages IIa and IIb were found in respectively 20.8% (N=5) and 16.6% (N=4) of endoscopic results.

The duodenum was usually without abnormalities (N=12) in 50% of cases. In 20.8% of patients (N=5) edema was found classified as Stage I. Stage IIa was encountered in 3 patients and stage IIIa in 2, i.e. 12.5% and 8.3% respectively.

AFFECTED ORGAN	cases	Percentage (%)
Esophagus	1	4,2
esophagus and stomach	20	83,3
esophagus and stomach and duodenum	3	12,5
stomach	0	0
Total	24	100,0

Table 10: Frequency of topography of lesion.

6. Limits:

In 4 cases, endoscopy could not be performed because of digestive perforation.

The exploration was not complete in two patients, due to impassable narrowing of the lumen. In one, only the esophagus was explored, and in the other, the pylorus and duodenum were unexplored.

IV. SCAN :

1. Directions:

During the years covered by our study, only 28 patients benefited from a CT scan after their admission with different indications:

- If there is a contraindication to endoscopy: suspicion of perforation. In 4 patients, i.e. in 14.2% of our patients.
- In case of incomplete endoscopic examination, due to impassable stenosis. In 3 patients, i.e. 10.7%.
- Suspicion of trans-parietal necrosis. That was in all of our patients.
- Postoperative control CT scan or after clinical worsening. Achieved in 3 of our patients, i.e. 10.7%.

2. Completion time:

Two types of scenographic examinations are performed in our study according to the completion time:

- Admission scanner: In our study, the initial scanner was performed on 27 patients, i.e. 96.4% of our patients after the endoscopic examination, at variable intervals. In the majority of cases, the delay in the ingestion of the caustic agent and the performance of fibroscopy was not specified.
- The control or evolution CT scan: was performed in 3 patients, i.e. 10.7% of our cases, after clinical worsening postoperatively or during monitoring.

NB: *We note that only one of these 3 patients underwent a postoperative CT scan after esophageal stripping, which did not allow the radiological signs of caustic damage to be described, thus the interest of its inclusion in our study, was to highlight the interest of CT in post-operative monitoring.*

3. Protocol:

The CT protocol in our study was not standardized given the retrospective nature of our study, and therefore depends on the specific indications of each patient:

- Type of examination:
  - Cervico-Thoraco-Abdominal CT: In 25 patients, i.e. 89.2% of our cases.
  - Abdomino-pelvic CT: In 2 patients, one of which was for post-operative control of jejunostomy.
  - Cervicothoracic CT: in a single patient.
- Injection of contrast product:

The injection of contrast product was systematic in our study, except in one patient because of impaired renal function.

Performed using an automatic injector or manually, if not available, by injecting 2cc/kg of Ultravist 300 iodinated contrast product at a rate of 3cc/second.

The injection times differed from one examination to another, with acquisition at 25 or 45 seconds for the arterial time and 75 seconds for the portal time:

- Arterial and portal time: performed in 5 patients, i.e. 17.8%.
- Portal time: in 22 patients, i.e. 78.5%.
- Without injection only: in one patient, i.e. 3.6%.

- Oral digestive opacification with water-soluble contrast products is a means of exploring the complications caused by the ingestion of caustic: in our study, no patient benefited from this technique.

4. Radiological signs:

• Digestive wall:

In this paragraph, we have studied the scans of the lesions carried out on admission of the patients, i.e. 27 examinations, of which 5 patients had a scan without anomalies (18.5%). In the other patients, several signs and radiological changes were observed:

- Submucosal edema:

Constitutes the sign most frequently found in our series, in all patients with a pathological scanner, i.e. 22 patients.

This sign appears in the form of non-enhanced parietal hypodensity, which may be associated with contrast enhancement of the mucosa and the external part of the wall.

Submucosal edema	Cases	Pourcentage (%)
Absent	5	18,5
Present	22	81,5
Total	27	100,0

Table 11 : Frequency of submucosal edema.

- Increase in parietal enhancement:

This sign was associated with submucosal edema in all our cases, involving the mucosa and the outer layer of the digestive wall.

Concerning the enhancement of the mucosa, its increase was found in 9 examinations out of 26, i.e. 34.6% of the cases, associated with an enhancement of the external part of the wall, achieving a target appearance.

Increase in parietal enhancement	Frequency	Pourcentage (%)
Absent	14	53,8%
Présent	12	46,2%
Total	26	100,0

Table 12 : Frequency by an increase in parietal enhancement.

- Parietal enhancement defects:

The absence of parietal enhancement is the major sign of necrosis. It can be localized to the mucosa (superficial mucosal necrosis) with the persistence of peripheral contrast enhancement (the external part of the wall) or extended to the entire wall (transmural necrosis).

The absence of mucosal enhancement with peripheral contrast enhancement was encountered in 10 out of 26 patients, i.e. 38.4% of lesion CT scans were injected.

The absence of parietal enhancement indicating transmural digestive necrosis was present in 8 patients, i.e. 30.8% of lesion CT scans were injected.

Trans parietal necrosis	Frequency	Pourcentage (%)
Present	18	69,2
Absent	8	30,8
Total	26	100,0

Table 13: Frequency of Parietal enhancement defect (Trans-parietal necrosis)

- Solution of parietal continuity:

Represents the direct sign of digestive perforation. It was found in 2 out of 27 patients, i.e. 7.4% of cases, including 1 case of esophageal perforation and 1 case of gastric perforation. In 2 other patients with digestive perforation, the solution point was not visualized.

✓ Infiltration of peri-digestive fat:

Sign present in 12 out of 28 patients, i.e. in 42.8% of cases.

✓ Digestive dilation:

Digestive dilation was found in 9 patients out of 28 patients, ie a percentage of 32.1%.

✓ Peritoneal effusion:

Peritoneal effusion is found in 5 cases, i.e. 18.5%, associated with peritonitis or pancreatitis in 40% and 20% respectively.

✓ Pneumoperitoneum:

This sign was encountered in 3 patients in our study, following a digestive perforation with peritonitis in 2 patients, and of postoperative origin in 1 patient.

✓ Pneumomediastinum:

Found in 3 patients in our series (10.7%), with mediastinitis on esophageal perforation in 2 patients, and of postoperative origin in another, appearing in the form of retro-esophageal air bubbles with individualization of the solution of continuity in one case.

✓ Pleural effusion:

It is present in 3 patients, or 10.7%, in the context of pneumopathy, peritonitis, or mediastinitis.

✓ Pericardial effusion:

Pericardial effusion was not found in any of our patients.

✓ Subcutaneous emphysema:

Subcutaneous emphysema occurred in one patient, associated with severe pneumomediastinum and pneumothorax, in the context of mediastinitis by esophageal perforation.

5. Lesional stage :

the interpretation of the scans performed in our study was standardized, based on the classification of Bruzzi and Mircea Chirica [2] :

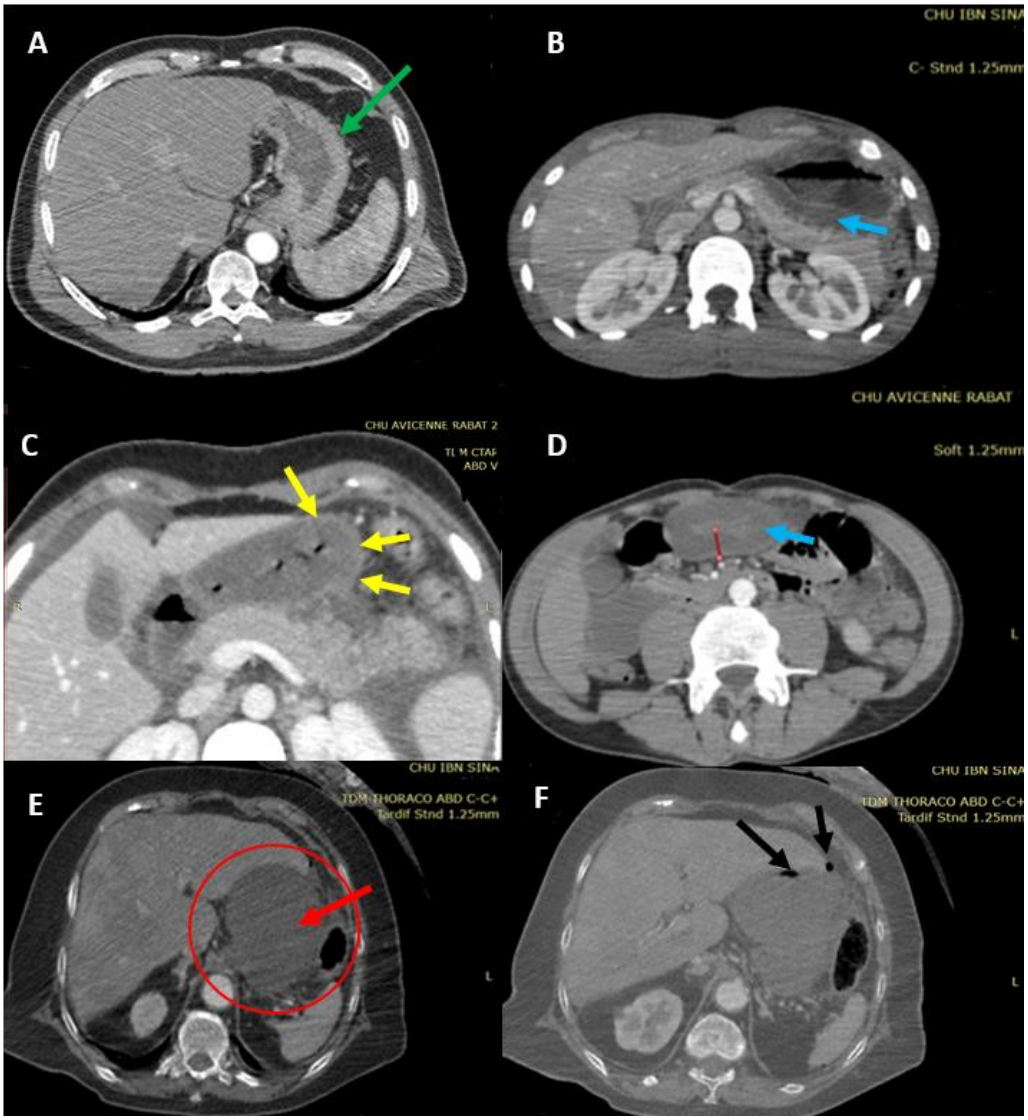


Figure 2: Axial scannographic images of patients having ingested caustic agents from our study, summarizing the radiological classification.

A. Stage I: Normal parietal enhancement (green arrow).

B. And D.: Stage IIa Submucosal edema (blue arrows).

C. Stage IIb: ring-shaped external parietal enhancement (yellow arrows).

E. Stage III: Trans-parietal necrosis with no enhancement (red arrow).

F. Stage IV: Gastric perforation with parietal necrosis.

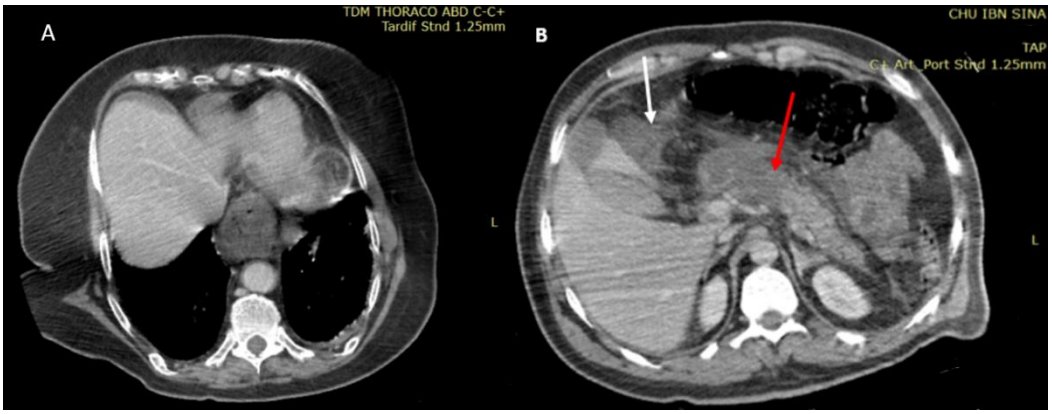


Figure 3: Axial scan sections of patients after ingestion of the caustic product.

A. A. Stage III: Transparietal esophageal necrosis.
B. B. Stage IV: Peritonitis with caustic necrotic pancreatitis of the head and pancreatic isthmus (red arrow) with peritoneal effusion (white arrow)

- Frequency of radiological stages:

	patients	Pourcentage (%)
Stage I	5	18,5
Stage Iia	5	18,5
Stage IIb	8	29,7
Stage III	5	18,5
Stage IV	4	14,8
Total	27	100

Table 14: Frequency table of pejorative CT stage of patients

In our series, stage IIb with submucosal edema was the most frequently encountered in 25.9% of cases (N=7).

Trans-parietal necrosis, classified as Stage III, represents 29.7% of our series, i.e. in 8 patients.

Stage IV perforation was encountered in 4 patients, two of whom presented with esophageal perforation and two others with gastric perforation.

The scannographic examinations without abnormalities, classified Stage I

represent 18.5% (N=5).

V. OTHER EXAMS:

1. Standard chest X-ray:

The standard X-ray was performed systematically on all our patients after their admission. And in patients admitted to intensive care or for control of pneumonia.

2. Abdomen without preparation:

It was performed systematically on all patients.

3. Barium contrast :

Barium contrast was requested in only one of our patients, as part of the control of staged esophageal strictures.

4. Abdominal ultrasound:

Abdominal ultrasound was performed on a single patient in intensive care, admitted for caustic peritonitis on organ perforation, for control of the effusion.

5. Bronchoscopy:

None of our patients benefited from a bronchoscopy.

VI. BIOLOGY:

Normal biology was found in 10 patients or 35.7% of our series. The most common anomaly was an inflammatory syndrome with hyperleukocytosis and an increase in CRP in 57% of cases.

Anemia was found in 5 patients, i.e. in 17.8% of cases.

Metabolic or mixed acidosis is encountered in 4 patients, all of whom stayed in intensive care in a state of shock.

Hydro-electrolyte disorders were noted in 8 patients, some of whom stayed in intensive care and others in surgery with esophageal stenosis.

Two patients presented with pancreatitis with elevated lipase.

VII. DEADLINES AND EVOLUTION:

1. Duration of hospitalization:

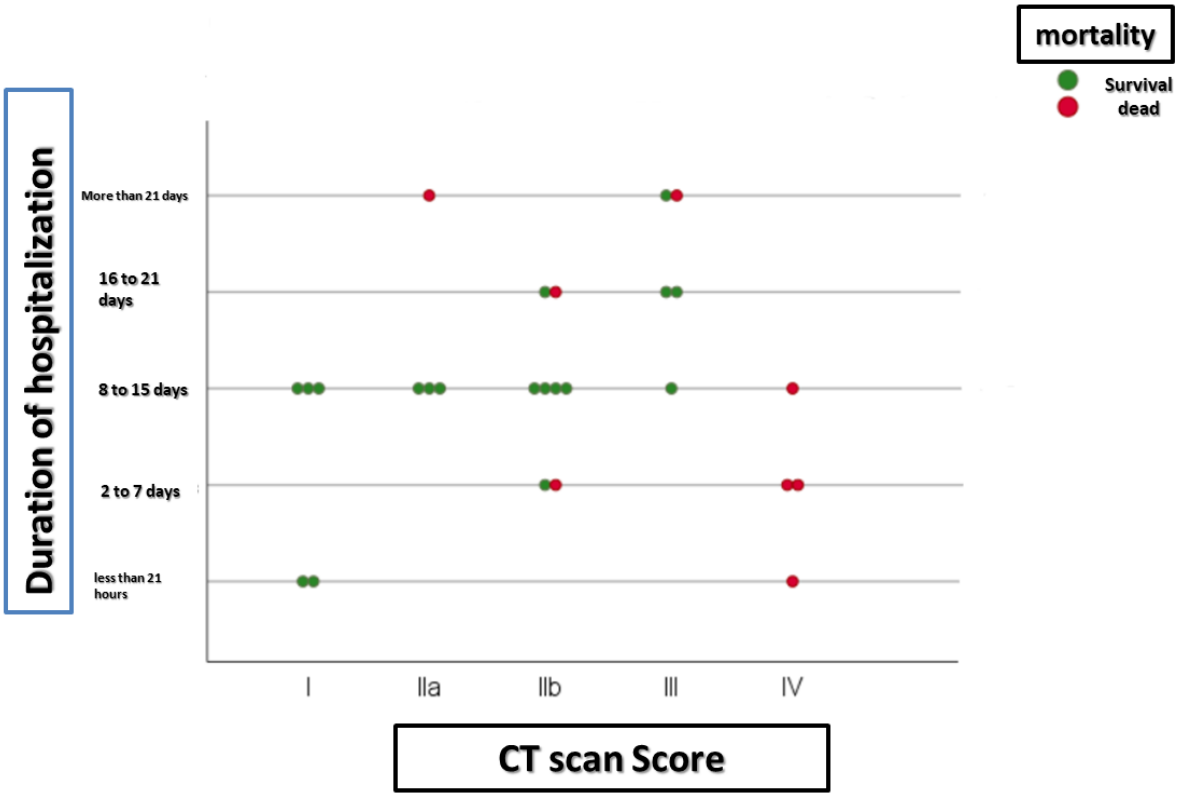
	patients	Pourcentage
Less than 24 H	3	10,7%
2 to 7 days	4	14,3%
8 to 15 days	12	42,9%
16 to 21 days	4	14,3%
More than 21 days	3	10,7%
Unknwon	2	7,1%

Table 15: Frequency of duration of hospitalization

The median duration of hospitalization for our patients was 14 days, with 42.9% having stayed between 8 and 15 days, knowing that in 2 patients the duration could not be determined.

In cases with CT stage III, the median hospital stay was 21 days compared to 15 days for stage IIa, and 13 days for stage IIb.

While patients with stage I (normal scanner), were hospitalized on average for 8 days of monitoring and medical treatment.



Graphic 7: Scatter diagram representing the respective duration of hospitalization at the scannographic stages found, correlated with mortality.

NB: 2 patients were hospitalized for an indefinite period.

Concerning patients with CT stage IV, the duration of hospitalization was shorter with a median of 7 days, given the fatal evolution towards rapid death explained by the complications.

In the other stages, mortality was linked to a long hospital stay.

2. Stay in intensive care:

In our series of cases, we had a total of 8 patients who required hospitalization in intensive care, i.e. 28.5% of cases, including 4 with radiological stage IV. One patient with stage IIa was admitted to intensive care mainly on neurological criteria after the onset of convulsive status, and two with stage IIb for pancreatitis.

Two cases with stage III on CT were also hospitalized in intensive care for hemodynamic and respiratory instability of septic origin.

3. Complications:

✓ Peritonitis:

Found in 3 patients in our series, i.e. 10.7%. This was a consequence of caustic perforation of the stomach in 2 cases and peritonitis on necrotic pancreatitis in another.

✓ Mediastinitis:

As a consequence of an oesophageal perforation, it is encountered in two patients classified as IV on the radiological score, and in whom endoscopy was not performed.

✓ Pneumonia:

Pneumopathy occurred in 9 patients out of 28, i.e. 32.1% after ingestion of caustic by inhalation, or during hospitalization, especially in intensive care in the context of VAP (Ventilator-acquired pneumonia).

In one of these patients, a Covid19 viral pneumonitis was found, initially suspected on the CT scan and confirmed by PCR.

- Necrotic pancreatitis of caustic origin:

Pancreatitis was encountered in our series in 2 patients (7.1%). The diagnosis was made in the face of hypodensity and lack of enhancement of the pancreatic head in one and the pancreatic tail in the other with infiltration of the peri-digestive fat and associated with increased lipasemia, the caustic origin is retained in front of the absence of obstacle with the association of these radiological signs and the clinical-biological context.

In both patients, a radiological stage IIb was found.

- Active bleeding:

Bleeding is not often visualized on the CT scan, however, clots formed sloping or active hemorrhage may be encountered in spontaneous contrast images, such as in the case of 2 patients in our series, all of whom presented digestive hemorrhage of the type hematemesis, with hemodynamic instability in one of them.

- Obstruction:

Postoperative complications occurred in one patient in our series after stripping of the esophagus and jejunostomy. The scanner then made it possible to make the diagnosis of occlusion due to intussusception.

- Stenosis:

Stenosis occurred in 14 patients, i.e. half of the 50% cases, including 3 who died.

- Iatrogenic perforation:

Oesophageal perforation following endoscopic dilation occurred in one patient in our study.

4. Mortality:

The death occurred in 7 patients, or 25% of our series. Including one death occurring one year after the ingestion episode in a patient complicated with stenosis, 4 deaths occurred after digestive perforation, including two esophageal with mediastinitis and two gastric with peritonitis. 1 patient died of peritonitis due to necrotic pancreatitis of caustic origin and 1 patient died of shock due to nosocomial pneumopathy acquired after intubation on neurological criteria.

VIII. MANAGEMENT :

1. Medical:

All our patients have benefited from medical care, which essentially includes:

- Discontinuation of food associated with parenteral nutrition with OLICLINOMEL N4 with a variable delay not specified in our research.
- Gastric protection by double-dose proton pump inhibitor during hospitalization and after discharge.
- Unsystematic antibiotic therapy. Administered to patients with an infectious syndrome, 9 cases in number (Pneumopathy / Mediastinitis / Peritonitis).
- Systematic preventive anticoagulation in patients who have been hospitalized.
- Vasoactive drugs in patients with hemodynamic instability, ie in 5 patients (17.8%).
- Oxygen therapy in patients with different indications:
  - Inhalation of nosocomial pneumonia (N=7).
  - Mechanical ventilation decision for the state of shock (N=4) or status epilepticus (N=1) patient.
  - Viral pneumonia due to covid19 (N=1).

2. Dilatation :

In our series, balloon dilation was performed in 5 patients with caustic stenosis, i.e. 35.7% of patients complicated by stenosis, 4 of whom improved after dilation, i.e. 80% success.

In two patients, dilation failed, one of whom was complicated by esophageal perforation after dilation and the other died after one year of ingestion.

3. Surgical:

✓ Exploratory laparotomy:

Performed in 4 cases admitted in a clinical picture of peritonitis (N=3) and Mediastinitis (N=1) who could only perform a CT scan classified IV.

✓ Total esogastrectomy:

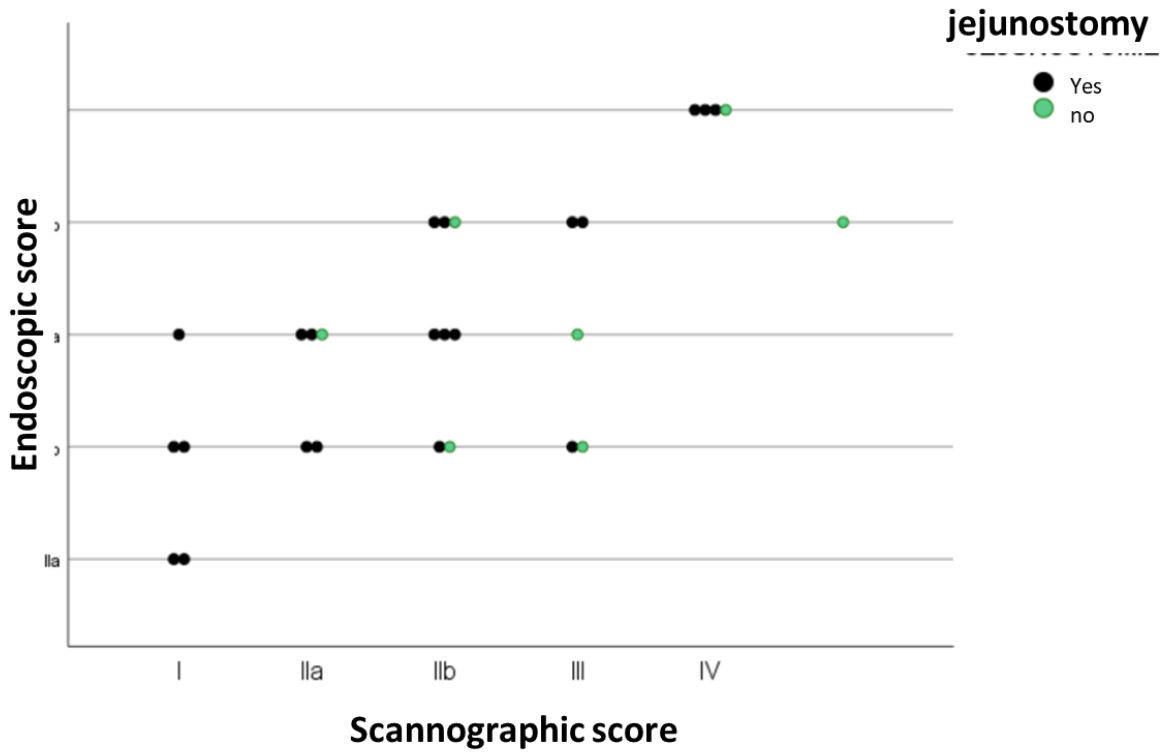
Carried out in 2 patients, one of whom had undergone esophagectomy by stripping, with an endoscopic score IIIb having only benefited from a postoperative CT scan, and the other had stage III CT scan and IIb endoscopic, operated after perforation on esophageal dilation.

The indication was made in a patient, admitted to intensive care with convulsive status and septic shock, who could not benefit from this surgery given her instability and fatal evolution towards death.

✓ Feeding jejunostomy:

A jejunostomy was performed in 7 patients, i.e. 25% of patients.

The CT scan was used to check the location of the jejunostomy in 2 patients, one of whom only performed a post-oesogastrectomy CT scan.



Graphic 8: Scatter plot of indications for jejunostomy according to established endoscopic and radiological scores.

IX. ENDOSCOPY-CT CORRELATION:

1. Prediction of transmural necrosis:

Considering transmural necrosis from radiological stage III and endoscopic stage 3b, we have established a descriptive table calculating the number of concordant and discordant results between the two examinations, recalling that trans-parietal necrosis could not be confirmed, given the lack of histological evidence and the small number of patients who underwent surgery.

a. In the stomach:

In 16 patients the endoscopic and radiological data were concordant on the absence of trans-parietal necrosis and in two patients on its presence.

A discrepancy in the results was encountered in only 4 cases, 3 of which required hospitalization in intensive care and jejunostomy, with an endoscopic Zargar score predicting trans-parietal necrosis (3b) and a CT score IIb, suggestive of necrosis limited to the mucous membrane only, associated with significant infiltration of the peri-digestive fat and necrotic pancreatitis in two of them who died.

In one patient, the CT scan was suggestive of transparietal necrosis with an endoscopic score of 3a for localized mucosal necrosis, requiring intensive care with intubation and jejunostomy.

Stomach		Necrosis CT		TOTAL
		yes	No	
Endoscopic Necrosis	Yes	2	3	5
	No	1	16	17
Total		3	19	22

Table 16 : Correlation of necrosis of endoscopic and radiological scores in the stomach.

NB: 6 patients with missing data excluded.

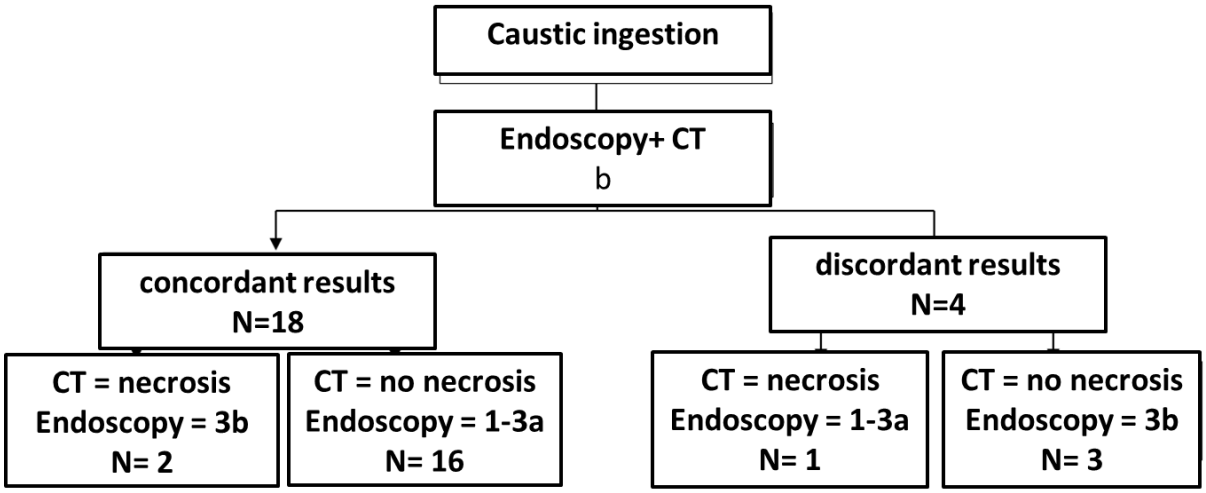


Table 17: Concordance diagram CT-Endoscopy at the level of the stomach.

b. In the esophagus:

The results agreed on the absence of trans-parietal necrosis in 19 cases, and its presence in a single patient.

In only 3 cases, the results of the two examinations were discordant at the level of the esophagus, in which the scanner was in favor of trans-parietal necrosis while the endoscopy was in favor of focal mucosal necrosis in the patient. one of them, having required a jejunostomy with hospitalization in intensive care, and deep confluent ulcers in the 2 others.

ESOPHAGUS		CT Necrosis		TOTAL
		yes	No	
Necrosis Endoscopy	Yes	1	0	1
	No	3	19	22
TOTAL		4	19	23

Table 18: Correlation of necrosis of endoscopic and radiological scores in the esophagus.

NB: 3 patients with missing data excluded.

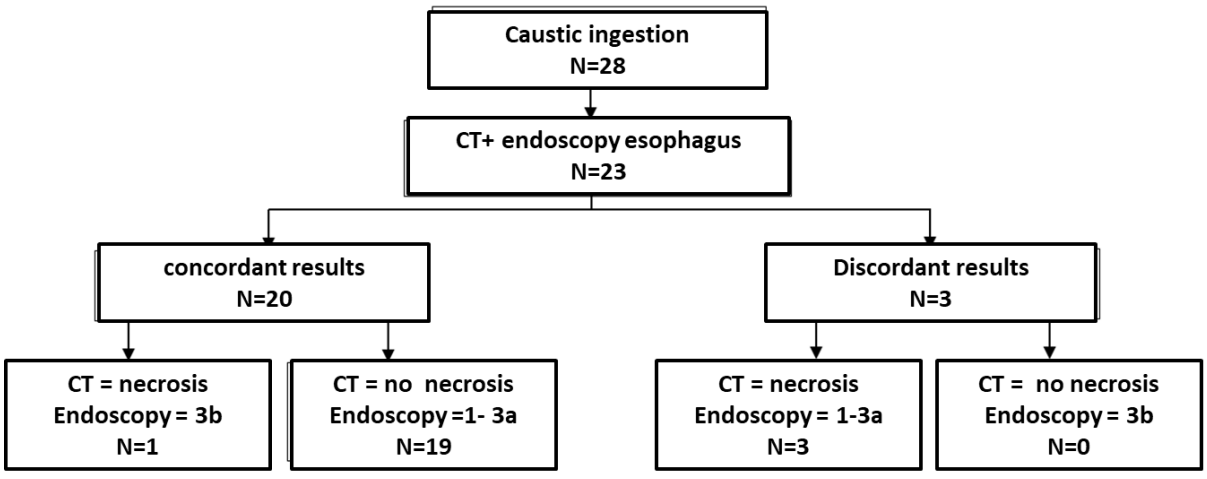


Table 19: Diagram of CT-Endoscopy concordance at the level of the esophagus.

2. Surgery:

We established a table of correlations between the radiological and endoscopic stages of necrosis with the indication for exploratory surgery and digestive reconstruction, excluding the placement of jejunostomy.

4 patients with a radiological stage greater than or equal to III underwent surgery, and 17 patients with a score lower than III underwent conservative management. The sensitivity and specificity of CT stages III and IV concerning surgical necessity were 80% and 77.3%, respectively, with a negative predictive value of 94.4% versus a poor positive predictive value of 44.4%. Similar values for endoscopy were found.

CT		Digestive surgery	
		Yes	No
Scannographic Score ≥ III	Yes	4	5
	No	1	17

Sensitivity	Specificity	PPV	NPV
80 %	77,3 %	44,4 %	94,4 %

Table 20: Correlation table of surgical necessity and trans-parietal necrosis on CT scan.

**Pearson Chi-squared test: With p value = 0.014 significant.*

ENDOSCOPY		Digestive suegery	
		Yes	No
Endoscopic score 3b	Yes	2	4
	No	1	17

Sensitivity	Specificity	PPV	NPV
81 %	66,7%	33,3 %	94,4 %

Table 21: Correlation table of surgical necessity and endoscopic stage of necrosis 3b.

**Pearson Chi-squared test: With p value = 0.075 non significant.*

3. Morbidity:

Considering that an abnormal CT scan predicts short- or long-term complications, we established a cross-tabulation to calculate the sensitivity, specificity, PPV, and NPV of the CT scan about complications.

Thus, the presence of CT lesion has a positive predictive value of 81.8% and a negative predictive value of 80% of the occurrence of a complication in the short or long term, with a sensitivity of 94.7% and specificity of 50%. And a score ≥ IIb is an 88.2% predictor of complications with less sensitivity.

CT*		Morbidity	
		Yes	No
Radiological effect Stade ≥ II a	Yes	18	4
	No	1	4
Sensitivity	Specificity	PPV	NPV
94,7 %	50 %	81,8 %	80 %
TDM**		Morbidity	
		Yes	No
Stade ≥ IIb	Yes	15	2
	No	4	6
Sensitivity	Specificity	PPV	NPV
78,9 %	75 %	88,2 %	60 %

Table 22

**Pearson Chi-squared test: With p-value = 0.006 highly significant*

***Pearson Chi-squared test: With p-value = 0.008 highly significant*

Regarding endoscopy, a Zargar stage of 2b or higher has a positive predictive value of 88.2% and VPN of 85.7% with a sensitivity of 93.8% and specificity of 75% compared with the occurrence of complications.

Endoscopy		Morbidity	
		yes	No
Endoscopic Stage ≥ 2b	Yes	15	2
	No	1	6

Sensitivity	Specificity	PPV	NPV
93,8 %	75 %	88,2 %	85,7 %

Table 23 : Prediction of morbidity by endoscopy.

**Pearson Chi-squared test: With p-value = 0.008 highly significant*

4. . Stenosis:

In 24 patients, whose evolution is known, we considered that the risk of stenosis is from endoscopic stage 2b, and compared the risk in patients with radiological stages [IIa to III] and [IIb to III] at the level of the esophagus. We established cross-tabulations between the endoscopic and computed tomography scores predicting stenosis, and its occurrence in the corresponding patients, by calculating the specificity, sensitivity, and positive and negative predictive value.

The results are represented by the following tables:

• At the level of endoscopy:

ENDOSCOPY		Stenosis		
		yes	No	
Esophageal stage Endoscopy ≥ 2b	Yes	13	4	
	No	1	6	
Sensitivity		Specificity	PPV	NPV
92,9 %		60 %	76,5 %	85,7 %

Table 24: Risk of stenosis correlated with endoscopy.

*Khi-2 : p value = 0,005 significant.
4 patients with missing data were excluded.

- CT scan :

CT*		Stenosis	
		Yes	No
Esophageal stage IIa-III	Yes	11	4
	No	2	6

Sensitivity	Specificity	PPV	NPV
84,6	60%	73,3%	75%

CT**		Sténose	
		Yes	No
Stade œsophagien IIb-III	Yes	7	3
	No	6	7

Sensitivity	Specificity	PPV	NPV
53,8%	70%	70%	53,8%

Table 25 B: **Risk of stenosis correlated with radiological stages. 5 patients with missing data were excluded.**

*Pearson chi-square: With p-value = 0.026 significant.

**Pearson chi-square: With p-value = 0.253 not significant.

5. Mortality:

Considering that the risk of death is from stage III trans-parietal necrosis on CT and stage 3b necrosis on endoscopy.

The establishment of cross tables allowed us to calculate the sensitivity, specificity, the positive and negative predictive values for each of the scannographic and endoscopic scores.

The results are represented by the following tables:

• Prediction of mortality / Endoscopic stage:

ENDOSCOPY		Mortality	
		Yes	No
Endoscopic score 3b	Yes	3	3
	No	5	17
Sensitivity	Specificity	PPV	NPV
37,5 %	85 %	50 %	77,3 %

Table 26 : **Prediction of mortality by endoscopy.**

*Pearson chi-square: With a non-significant value of p=0.111.

•• Prediction of mortality / CT score:

CT		Mortality	
		Yes	No
Scannographic score ≥ III	Yes	5	4
	No	3	15
Sensitivity	Specificity	PPV	NPV
62,5 %	78,9 %	55,6 %	83,3 %

Table 27 : **Prediction of mortality by CT score.**

*Pearson chi-square: With p-value=0.037 significant

CT		Mortality	
		Yes	No
Score Scannographique IV	Yes	4	0
	No	4	19
Sensitivity	Specificity	PPV	NPV
50 %	100 %	100 %	82,6 %

Table 28 : **Prediction of mortality by CT score.**

*Pearson chi-square: With p-value=0.001 highly significant.

Discussion

I. GENERAL:

The ingestion of caustic agents is a frequent emergency in Morocco, requiring multidisciplinary management, between emergency physicians, visceral surgeons, gastrologists, and radiologists.

Most often benign with good evolution, this emergency can jeopardize the prognosis as well as vital by its redoubtful complications and functional, the damage which it can cause, and its heavy management. This depends essentially on the nature and quantity of the caustic agent ingested, but also on the initial management and the right therapeutic decision.

The role of the CT scan, still little indicated today, is becoming more and more obvious by its non-invasive nature: in particular, in the evaluation of the extent of the lesion, the establishment of surgical indications, and the search for complications. In other situations, imaging can replace endoscopy in the event of a contraindication or difficulty of realization.

The main objective of our study is then to establish the interest of the CT in the management of patients who have been victims of caustic ingestion, to clarify its indications, and standardize a practical course of action, the least invasive. possible while keeping appropriate care for each patient to avoid unnecessary surgeries.

II. RADIOLOGICAL AND ENDOSCOPIC ANATOMY

1. Esophagus:

a. external configuration :

- The esophagus is a musculo-membranous duct, when empty its thickness measure 3 mm, with a normally flattened lumen.it constitutes the first segment of the digestive tract, starting from the hypopharynx and ending with its junction at the stomach.

- It has 3 portions: [Figure 3]

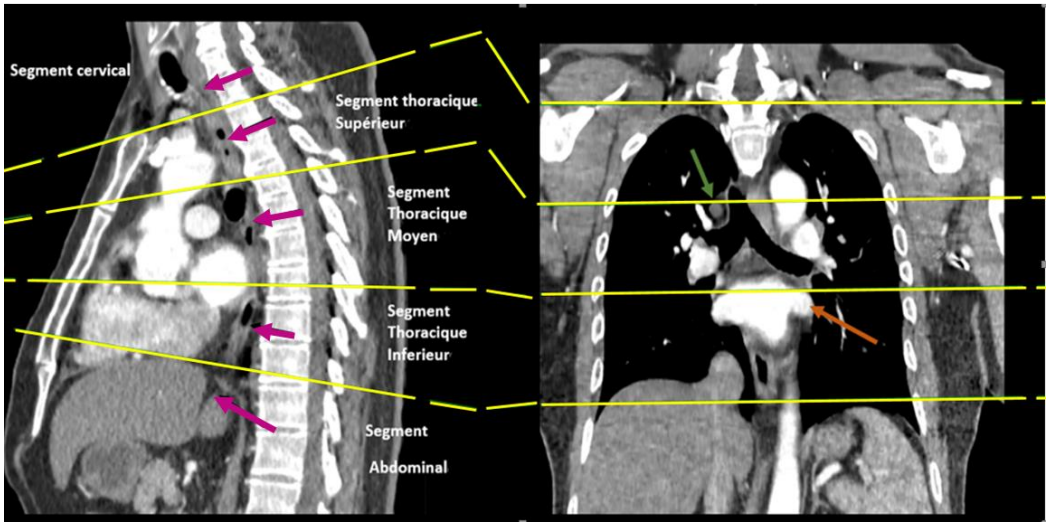
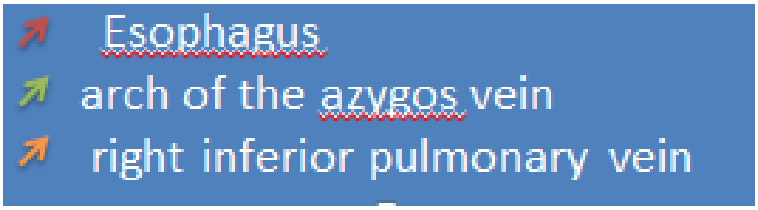


Figure 4: axial and coronal CT scan images with mediastinal window after contrast medium injection demonstrating different segments of the esophagus



- **Cervical portion**: from the inferior border of the cricoid cartilage to the sternal manubrium at the level of 6 th vertebrae.

- **Thoracic region**: where the esophagus is divided into three levels: upper, middle, and lower: [Figure 3]
- Upper 1/3: from the manubrium to the arch of the Azygos vein.
- Medium 1/3: from the arch of the Azygos vein to the left lower pulmonary vein.
- lower 1/3: from the left lower pulmonary vein to the diaphragmatic hiatus.
- **Abdominal region:** after passing through the diaphragm till the gastroesophageal junction where it continues with the stomach through the cardia.

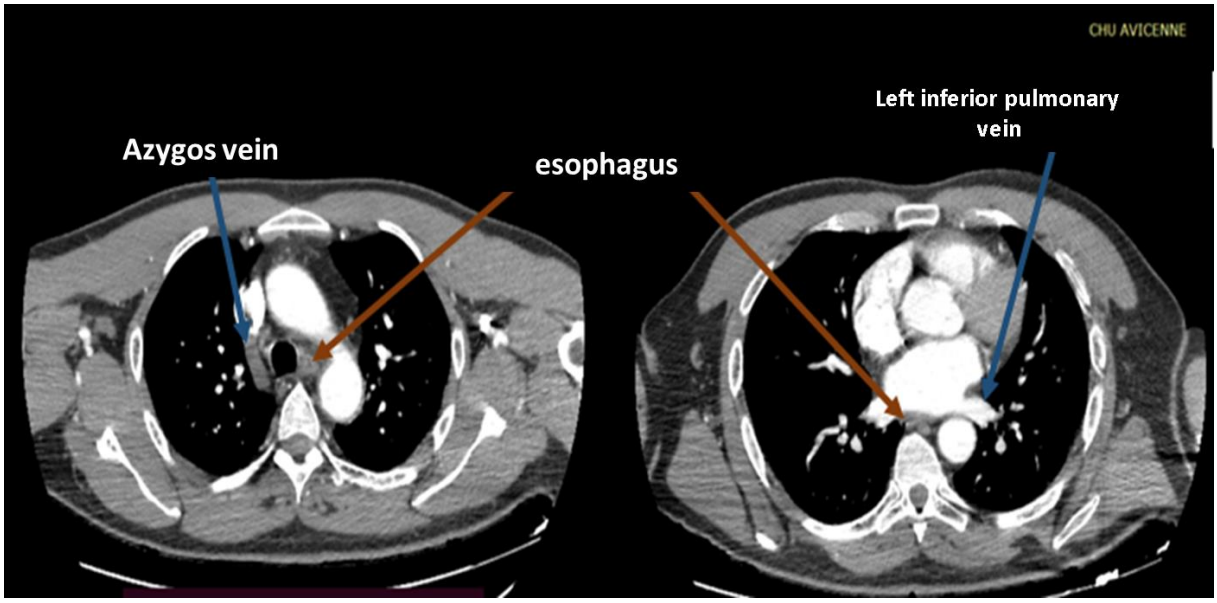


Figure 5: axial CT scan images of the thorax after medium contrast injection showing anatomical landmarks in the division of the thoracic esophagus

b. internal configuration

The esophagus has 4 layers: [Figure 5]

• Adventitia or esophageal fascia: Loose connective tissue in continuity with the phrenic-esophageal fascia.

• Muscular: Made of an outer layer of longitudinal bundles and an inner layer formed by circular bundles. Striped type in its upper part and smooth in its lower third, with a mixture of fibers in its middle third.

• Submucosa: Loose connective tissue, enclosing the esophageal vessels and glands in the thoracic part of the esophagus.

•Mucosa: Stratified squamous epithelium. [3]

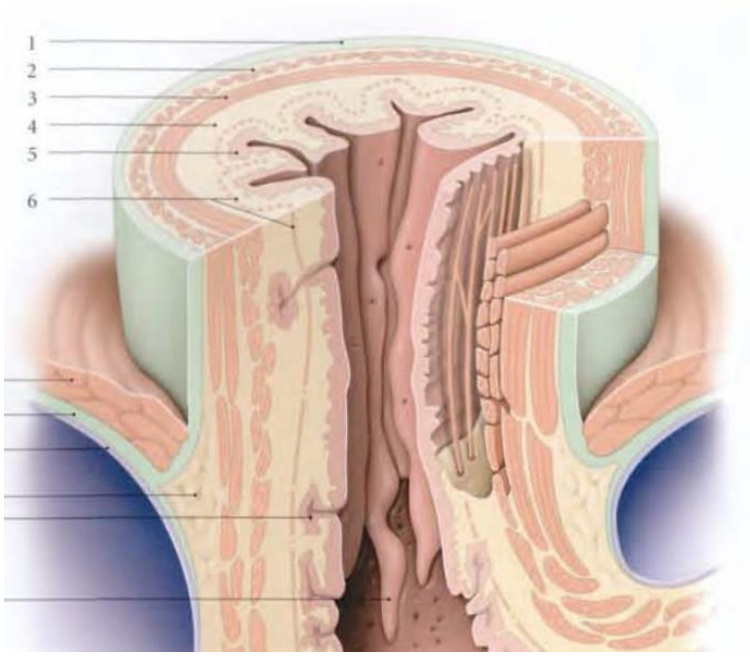


Figure 6: Representative diagram of the layers of the esophagus.
1. Adventitia, 2. Longitudinal muscle layer. 3. Circular muscle layer, 4. Submucosa, 5. Esophageal gland, 6. Muscularis mucosae.

c. endoscopy :

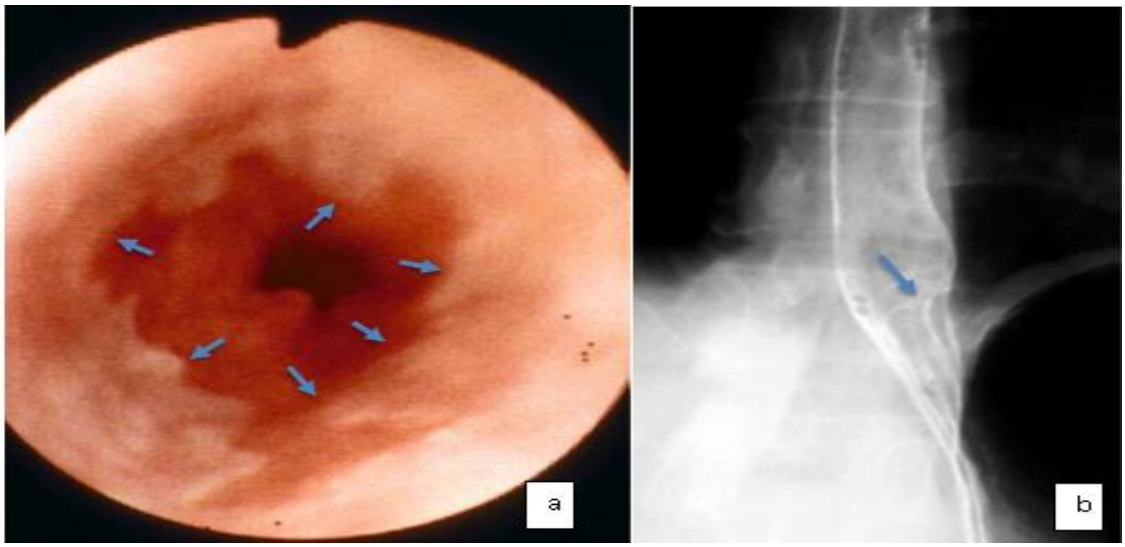
Contrary to radiology the piriform sinuses are not possible to inspect in detail due to the physiologic reason that the hypo-pharynx closes with the passage of the instrument, however, the Opening of pharyngeal diverticula is overlooked when small, as are shallow membranes at the oesophageal inlet.

The aortic arch creates a shallow indentation in the upper third of the esophagus in the elderly. Sometimes a slight indentation in the mid-esophagus is also encountered, caused by the adjacent left main bronchus. At its distal end, the esophagus tapers slightly to the left just before it enters the stomach.

Another identifiable landmark is the diaphragmatic inlet, which varies in width with respiration.

When distended with air the distal esophagus demonstrates the margin between the pale squamous epithelium of the esophagus and the reddish cylindrical epithelium of the cardia [Figure 6a]. This margin or transition zone is referred to as the “Z-line” because of the interdigitations of the mucosa. In healthy individuals, the “Z-line” is normally found close to the diaphragmatic inlet, usually at 40 cm from the incisor teeth. The Z-line is difficult to depict at the radiological examination; even on high-quality double-contrast oesophagograms [Figure 6 b], because of the subtle change of mucosal surface structure between the squamous and columnar epithelial lining.

The normal surface of the squamous epithelium is even, though slightly elevated islands of glycogen deposits may be seen [Figure 6 c], we can find also submucosal vessels that are more prominent in the elderly with mucosal atrophy.



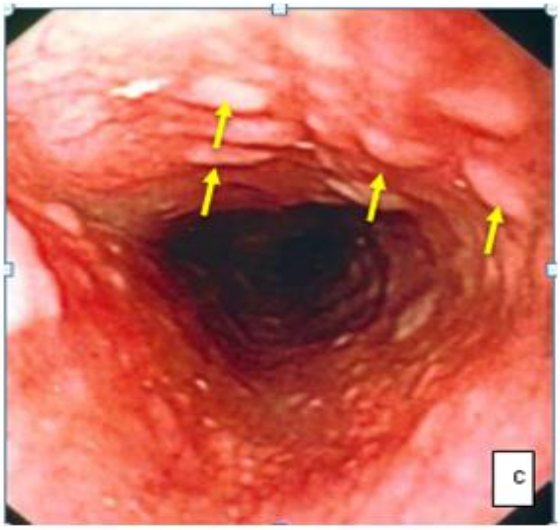


Figure 6: The normal oesophagogastric junction with a well-demarcated Z-line ( ) as seen in an endoscopy (**a**) and the equivalent radiographic view (**b**), we can also see mucosal deposits of glycogen ( ) that is a normal finding (**c**)

Figure 7

2. stomach:

a. External configuration :

located between the esophagus and the duodenum, a muscular reservoir endowed with an abundantly secreting mucous membrane, and consisting of two faces, anterior and posterior and two edges, the major and the lesser curvature as well as two orifices the cardia and the pylorus, it is thus made up of four parts [Figure 7]:

- the cardia: Junction with the esophagus.
- fundus: Upper pole corresponding to the gastric air pocket.
- BODY: Vertical middle part
- PYLORUS: Made up of the pyloric antrum which is the sloping point of the stomach and the pyloric canal which connects it to the duodenum.

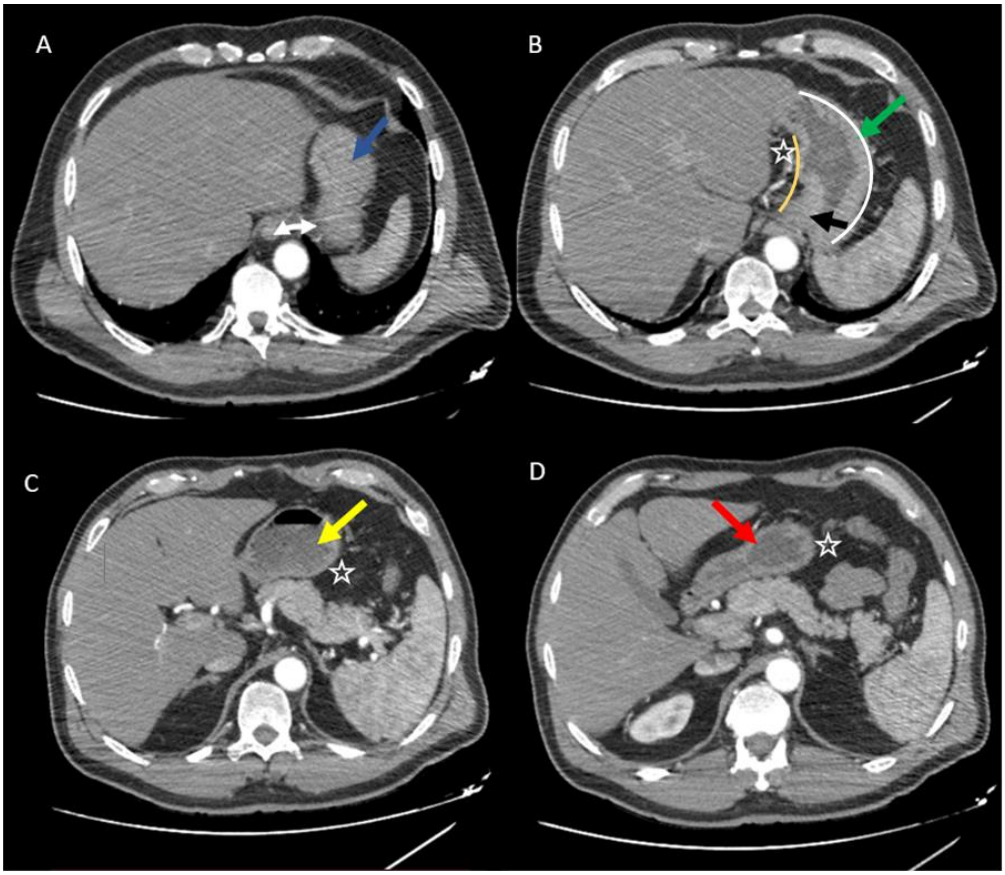


Figure 8: Axial CT scan images of the abdomen after injection in the arterial phase :

- A : fundus
- esogastric junction
- gastric body
- Cardia
- Lesser curvature
- Greater curvature
- Antri
- Pylori
- Perigastric fat normally hypodense

b. Internal configuration

The stomach wall is made from the outside to the inside of [Figure 8]

- **Serous**: gastric visceral peritoneum.
- **Subserosa**: Loose connective tissue.
- Muscular: Made of three layers of smooth muscle fibers, longitudinal, circular, and oblique.
- **Submucosa**: Loose areolar tissue.
- **Mucosa:** Simple, thick, and resistant prismatic epithelium, made up of folds presenting small depressions where the glands open:
  - ✓ CARDIA: Cardial glands: mucus.
  - ✓ FUNDUS and BODY: Proper gastric glands: Hydrochloric acid.
  - ✓ PYLORI: pyloric glands: mucus. [3]

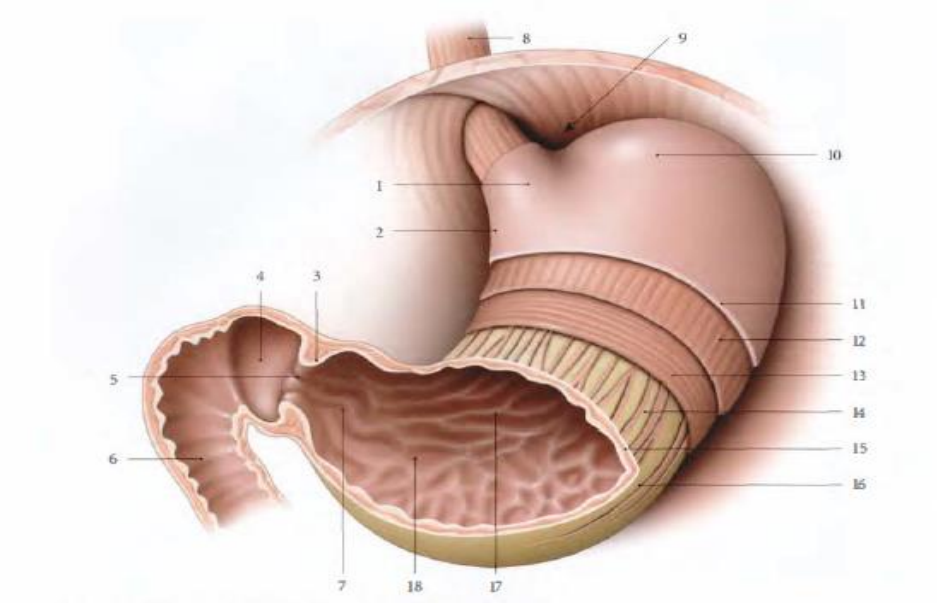


Figure 9: Representative diagram of the internal and external configuration of the stomach:

1. Cardia, 2. Lesser curvature, 3. The pyloric sphincter, 4. Duodenal bulb, 5. Pyloric orifice, 6. Duodenum, 7. Pyloric canal, 8. Esophagus, 9. Cardial fissure, 10. Fundus of the stomach, 11. Serosa, 12. Longitudinal layer, 13 Circular layer, 14. Oblique fibers, 15. Mucosa, 16. Greater curvature, 17. Longitudinal folds, 18. Pyloric antrum.

c. endoscopic :

Easily recognized endoscopic landmarks in the stomach are the cardia, the greater curvature marked by its tortuous longitudinal folds [Figure 9a], the incisura angularis, and the pylorus.

Less obvious landmarks are the even surfaces of the lesser curvature and gastric antrum. All these landmarks may all change in disease.

The structure of the gastric mucosa shows a faint, regular mosaic pattern, which is enlarged and prominent in conditions of gastropathy and correlates to the area gastric as well known to radiologists [Figure 9 b]. This may be particularly marked in the condition of portal gastropathy, where we may find tortuous fundic veins in more severe cases.

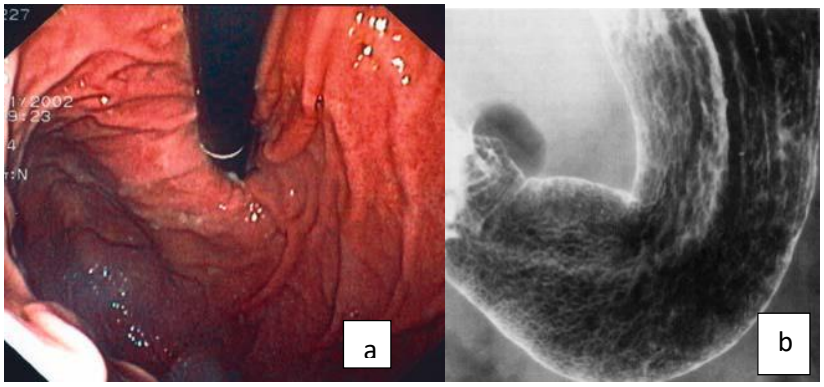


Figure 10: Normal gastric fundus and body. The gastroscope is retroverted, demonstrating the instrument emerging through the gastric cardia (a) which is known in double contrast image as polygonally shaped radiolucent tufts of mucosa outlined by barium in grooves (b)

3. Duodenum:

a. External configuration

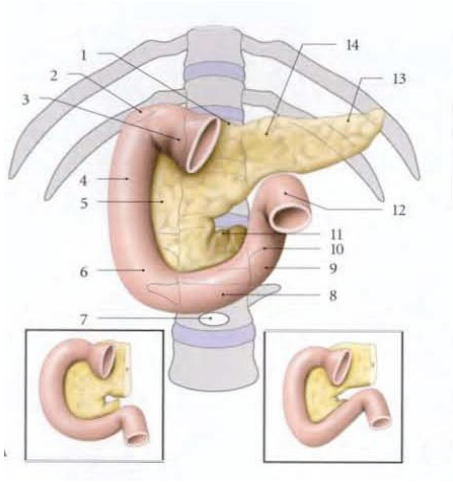
Represents the initial part of the small intestine, localized between the pylorus and the jejunum at the level of the duodenojejunal angle. It consists of four parts: [Figure 8]

- D1: Has a mobile initial part and a fixed one.
  - ✓ Mobile part: Responds to the pancreas and represents the most frequent seat of duodenal ulcers.
  - ✓ Fixed part: Responds to the square lobe and the gallbladder: explaining the possibility of cholecysto-duodenal fistulas.

- D2: is just to the right of the midline, and contains the major duodenal papilla, the common entrance for the bile and pancreatic ducts, and the minor duodenal papilla, which is the entrance for the accessory pancreatic duct. [Figure 10]

• Horizontal D3. is the longest section, crossing the inferior vena cava, the aorta, and the vertebral column. It is crossed anteriorly by the superior mesenteric artery and vein.

•Ascending D4: terminates at the duodenojejunal flexure.



2. superior Angle of Duodenum.
3. superior partion of Duodenum
4. ascending partion of Duodenum
5. head ofpancreas.
6. inferior Angle of Duodenum
8. descending partion of Duodenum
9. left inferior Angle

Figure 11: Representative diagram of the external configuration of the duodenum.

b. Internal configuration

The duodenal wall is made up from the outside to the inside of [Figure 10]:

- The serous tunic: formed by the visceral peritoneum.
- The subserous layer: a thin layer of loose connective tissue.
- The muscular tunic: with a circular deep layer and a longitudinal superficial one, between which sits the myenteric plexus.
- The tunica submucosa: loose connective tissue.

- The tunic mucosa: formed of surface epithelium with a lamina propria and muscular mucosa. Lining its inner surface is characterized by numerous villi and microvilli forming circular folds. [3]

Figure 12: axial CT scan of the abdomen at the level of the D2 (↗

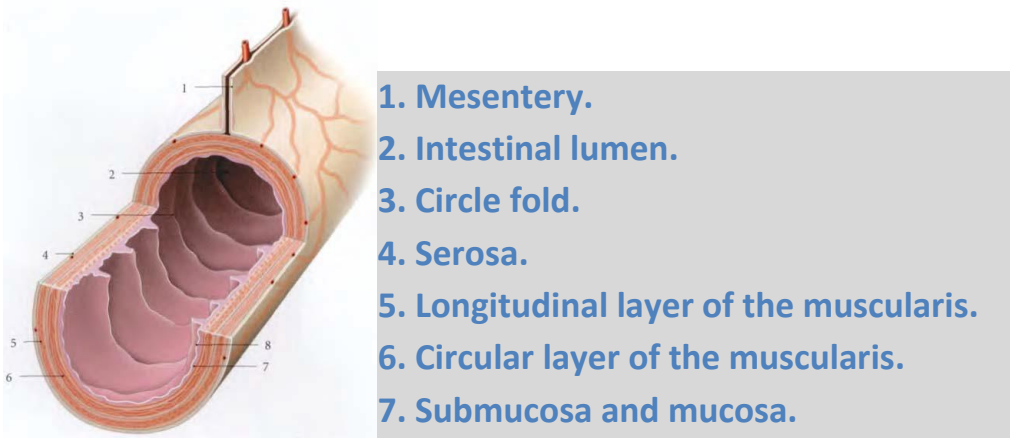


Figure 13: Representative diagram of the histological layers of the duodenal wall.

c. Endoscopy

The duodenal bulb or first part of the duodenum is wide, and its mucosa yellowish and even. The superior duodenal flexure demarcates the commencement of the second part of the duodenum, and this is normally well recognized. From this point onwards the mucosal pattern is characterized by folds of Kerckring [Figure 13a]. Most of the folds measure around 1 mm in height and run in a semicircular way with small longitudinal folds in between.

A distended gallbladder may cause an indentation of the proximal duodenum. In the mid-point of the second part of the duodenum, a normal-sized papilla of Vater may be identified

with a forward-viewing gastroscope [Figure 13b]. but is better appreciated by the use of a side-viewing instrument. Transition to the third or horizontal

Figure 13 a: Normal duodenum mucosa demonstrating the folds of Kerckring

Figure 13b: Descending duodenum with the minor papilla

Figure 14

4. Pancreas

The pancreas lies mostly posterior to the stomach [Figure 14]. It extends across the posterior abdominal wall from the duodenum, on the right, to the spleen, on the left.

The pancreas is (secondarily) retroperitoneal except for a small part of its tail and consists of a head, uncinate process, neck, body, and tail.

- ✓ The **head of the pancreas** lies within the C-shaped concavity of the duodenum.
- ✓ Projecting from the lower part of the head is the **uncinate process**, which passes posterior to the superior mesenteric vessels.
- ✓ The **neck of the pancreas** is anterior to the superior mesenteric vessels. Posterior to the neck of the pancreas, the superior mesenteric and splenic veins join to form the portal vein.
- ✓ The **body of the pancreas** is elongated and extends from the neck to the tail of the pancreas.
- ✓ The **tail of the pancreas** passes between layers of the splenorenal ligament.

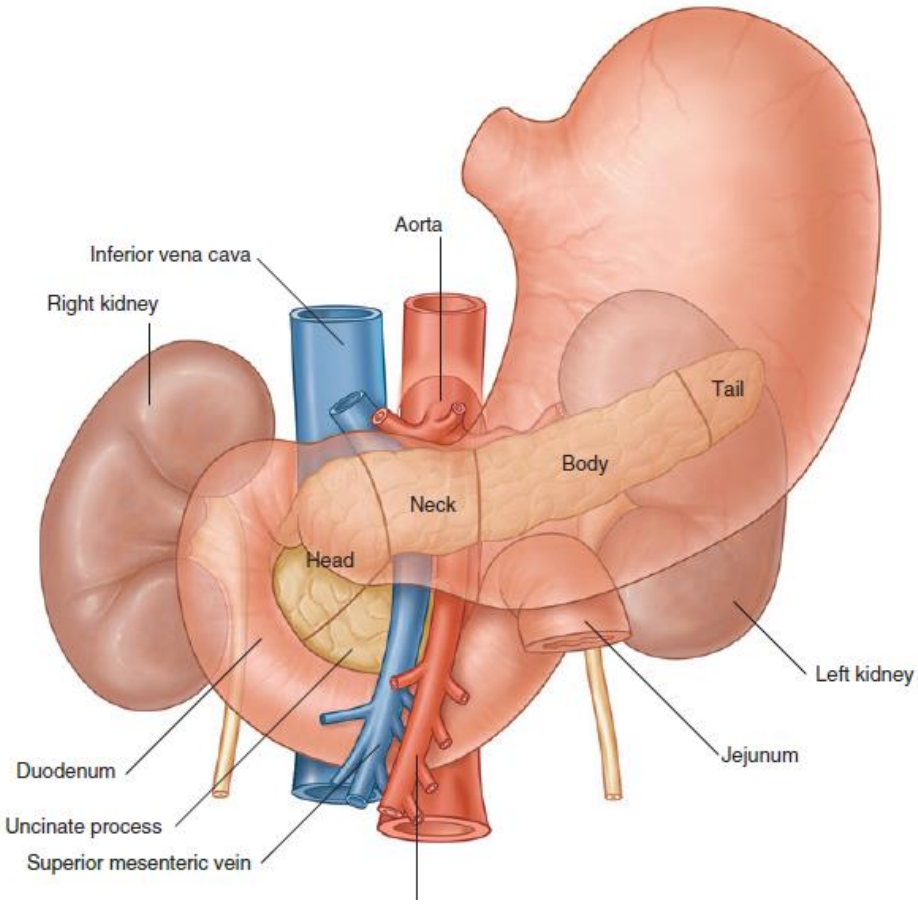


Figure 15: the external configuration of the pancreas.

III. CAUSTICS:

The major caustic products are mainly classified into 3 groups, strong acids, strong bases, and oxidants.

The identification of the type of product is essential to assess the prognosis of the extension of the lesions and to prevent and monitor possible systemic consequences.

1. Pathogenesis of caustic products:

•The degree and extent of the lesions depend on their nature, their form, and the quantity ingested. Combinations of products can worsen the prognosis.

a. Nature :

• **Strong bases** cause proximal and deep lesions: induced by **liquefaction** necrosis (protein hydrolysis) and **saponification** of lipids. With secondary inflammatory phenomena and hypertrophic tissue reconstruction.

• **Strong acids** cause more distal and superficial damage (apart from massive ingestions) but the detersion is slow. Induced by surface necrosis due to dehydration and **cellular coagulation**.

•**Oxidants** induce chemical burns with the condition of contact with the mucous membrane for a sufficient duration and at a sufficient concentration, resulting in the **denaturalization of proteins** [4]

• Products with a systemic effect that may worsen the clinical condition: include boric acid, Paraquat, and other substances with a calcium-chelating effect. [5]

Corrosive Product	Classe	Utilization	toxicity
Chloridric acid	strong acid	descaling	Pulmonary edema
Fluohydric acid	strong acid	Rustproof	Hypocalcemic
Nitric acid	strong acid	metal stripper	Pulmonary edema methemoglobinemia
Sulfiric acid	strong acid	battery fluid	Pulmonary edema
Oxalic acid	weak acid	Rustproof	Hypocalcemic
Acetic acid	weak acid	Descaling	
Bleach	Oxydant	Disinfectant	Pulmonary edema If mixed with acid
oxygenated water	Oxydant	Disinfectant	
Potassium permanganate	Oxydant	Disinfectant	
sodium salt	Weak base	laundry detergent	
Potasse	strong base	laundry detergent	
caustic soda	strong base	drain unblocker	
Ammoniac	strong base	household detergent	Pulmonary edema
Paraquat	Various	Herbicide	Pulmonary fibrosis
phenol	Various	Disinfectant	cardiogenic collapse

Figure 16: List of main corrosive products**.**

b. Forms :

•**Products in powder and crystals**, which are difficult to swallow, essentially cause oropharyngeal and proximal oesophageal lesions.

•**Gels** by their consistency, lead to a prolonged contact time with the digestive tract aggravating the lesions and are consequently responsible for burns in casts of the oropharynx and the esophagus.

•**Foams** are mainly ingested in small quantities responsible for oral-pharyngeal and laryngeal lesions, or even inhalation lesions if the quantity is large.

• **Liquid caustics** progress rapidly through the digestive tract and cause more distal lesions reaching the stomach and even the duodenum.

• The combination of a **surfactant** increases the contact time and aggravates these lesions [4].

c. Topography of lesions:

- **and Abdomen:**
  - Their involvement is often due to delayed management.
  - Massive ingested quantity and/or strong causticity.
  - In certain cases, burn and necrosis lesions can occur by contiguity with the lung, the heart, and the surrounding abdominal organs, namely the tail of the pancreas, the left lobe of the liver, the spleen, and the colon.
- **small intestine and duodenum involvement :**
  - Its impairment is due to a reflex gap in the pylorus caused by burning, or during the ingestion of large quantities of liquid products.
- **Tracheo-bronchial involvement:**

The consequence of vomiting, diffusion, or inhalation of foamy products, which can be the cause of:

- Tracheal or bronchial perforation.
- Tracheo- or broncho-oesophageal fistulas.
- stenosis.
- Bronchomalacia.

2. The natural evolution of lesions:

The ingestion of caustic is responsible for macroscopic and microscopic lesions evolving in four successive phases described in the literature: [6] [7]

a. Initial phase: D1

Characterized by a series of histological lesions:

- Mucosal and submucosal edema and hyperaemia.
- More or less deep ulcerations and necroses.
- Thrombosis of the mucous and serous veins.
- Lymphatic stasis.

Serious complications can appear early:

Local:

- **Perforation:** peritonitis or corrosive mediastinitis.
- **Hemorrhages** of medium abundance.
- **Edema** of the aero-digestive tract: dysphagia and dysphonia.

Locoregional:

- Laryngeal edema.
- Lesional lung edema.
- Intraperitoneal organ burns [liver, spleen, duodenum, pancreas, colon].
- More rarely, Thrombosis of the supra-hepatic veins.

General:

- Hemolysis, metabolic acidosis, state of shock, disseminated intravascular coagulation.

b. Cleansing phase: D2-D15

Major vascular and blood manifestations:

- congestion.
- Hemorrhage.
- Edema affects all layers of the digestive wall.
- Venous thrombosis and arteriolar sectors result in ischemia and aggravation of necrosis.
- Significant perilesional infiltration (PNN diapedesis).
- Foci of necrosis are gradually eliminated, and ulcerations are more or less deeply into the digestive wall.

The sepsis of the digestive tract promotes the development of bacterial colonies with an increase in the inflammatory reaction, thus aggravating the lesions.

- This evolution leads to multiple complications:

  - Local: Digestive perforation due to significant parietal fragility.
  - Locoregional:
    - Lesional pulmonary edema.
    - Pleuropulmonary superinfections.
  - General:
    - Hemolysis, metabolic acidosis, sepsis, and septic shock.
    - Acute tubulointerstitial nephritis.
    - Acute necrotizing pancreatitis.

c. Repair phase: D15-D30

- Phase characterized by the formation of a **fleshy inflammatory** bud due to fibroblastic proliferation.
- Complications are:
- Local:
    - Hemorrhage due to rupture of the neo-capillaries of the inflammatory bud. Perforation remains possible [until the 14th day].
    - Fistula from the esophagus to the tracheobronchial tree or more rarely to the aorta.
    - Early stenosis from the eighth day.
- General: septic complications: Systemic, pulmonary, deep collection…

d. Sequelae phase: after 30 days

Phase characterized by the formation of scar fibrosis as a result of re-epithelialization of connective tissue with collagen forming retractile sclerosis of the wall of the digestive tract and sometimes adhesions to adjacent organs.

Lesions can progress to:

- Healing without sequelae.
- The eso-bronchial fistula, oeso-aortic fistula, secondary hemorrhage, obstructed perforation. Onset around 20 days.
- Tracheal stenosis and bronchomalacia, are rare but dreadful. Around 3 months at the esophageal level and 6 months at the Pharynx level.

IV. EPIDEMIOLOGY :

1. Frequency:

The frequency of caustic ingestions varies between studies. In France, this is around 15,000 and 20,000 cases per year, which represents 1-2% of overall poisonings with approximately 10% mortality [5], While in the United States it is estimated in studies that the number of cases varies between 5000 and 15000 cases per year [8], [9].

In Morocco, work by the anti-poison center in Rabat concluded that a frequency varying between 35 and 50 cases per year [10], a study carried out at the CHU of IBN-SINA in Rabat shows an average of 11 patients per year [11], which converges with the results of our study of approximately 15 patients per year.

2. Age:

A predominance in children has been noted in multiple studies, in adult subjects a high incidence in people aged between 18 and 65 years has been reported, with an average of 35-40 years in a study in Iran [12], compared to Morocco, the average age was 17 ± 16 years in patients ranging from 1 day of life to 96 years according to studies by the poison control center in Rabat. [13]

The average age in a study carried out in the UCV department at the University Hospital of Rabat was 35 years, with extremes of 15 to 70 years [11], which is close to our results where the most frequent age interval was 18 to 30 years old with a median age of 33 years.

3. Sex:

A discrepancy noted between studies from different countries shows a ratio of one man for two women in France [14], as in Morocco according to a study carried out at the poison control center in Rabat which concludes with a sex ratio (M/F) of 0 .89 where the female sex represents 52.8% [13].

The series studied at UCV, on the other hand, shows a male predominance with 63.79% in men, which is consistent with a study carried out in Iran finding 55% of men compared to women [11] and with ours which shows a predominance 60.7% male.

4. Background:

In the context of caustic ingestion, the history constituting a poor prognostic factor is essentially the psychiatric history associated with toxic and alcoholic habits. Indeed, these are a predictor of the intentionality of ingestion. A general history, in particular cardiovascular, immunosuppression, or a history of digestive surgery, can worsen the patient's condition by promoting complications or by their decompensation.

In our study, the predominant antecedents were essentially psychiatric and toxic.

5. Circumstances:

Two contexts should be distinguished: accidental ingestion of caustic or following an attempt at autolysis.

- **Attempts at autolysis:** represents the most frequent context in approximately 75% of cases, with a psychiatric ground of depression or psychosis. This is the situation with the most serious complications and sequelae, given the massive quantities as well as the drug-toxic associations.
- **Accidental ingestions:** less frequent, encountered in children or adults with a particular terrain, especially in people with psychomotor retardation, dementia, or alcoholics. [14]

Similar results obtained in Morocco, conclude to 79% of cases of voluntary intoxication with the aim of autolysis [11], which agrees with our study which finds 75% of cases of attempted suicide.

6. Product:

At the head of the products involved in caustic intoxications, is hydrochloric acid, which appears in multiple studies, including that carried out by ELHAMMOUMI at the University Hospital of Rabat, with 63% of cases intoxicated by this product, leaving second place to oxidants, then to the other products. [11]

In our study, hydrochloric acid was found in 85.7% of cases, as a strong acid in liquid form.

V. CLINICAL DIAGNOSIS:

The ingestion of corrosive products is a circumstantial emergency, the positive diagnosis of which is essentially anamnestic and clinical, hence the importance of a well-organized approach with a complete and oriented history and clinical examination, within a framework of trust with the patient, which is often difficult to obtain at the beginning.

1. Interrogation:

- Approach the patient calmly and reassure him.
- **Specify the age, history, and added comorbidities** that may decompensate and thus worsen the patient's prognosis.
- **Establish intentionality**: in a context of attempted autolysis, isolation, rupture, bad sentimental experience or behavioral disorders and psychoses can frequently be noted, which can make treatment more delicate, in this case, the damage is more often serious or fatal compared to the accident context.
- **Specify the ingestion schedule and the consultation period.**
- **Identify the Nature, Form, and Quantity of the product ingested:** this information makes it possible to establish a prognosis and guide treatment. "Quantity superior than 150ml = gravity +++"
- **Specify drug/fluid combinations**: some of which complicate the condition through their various systemic effects.
- **notion of induced vomiting**: During provoked vomiting, the contact time with the digestive wall is lengthened by a double path, which constitutes an additional factor of serious digestive lesions.

2. Clinical feature: [4]

Apart from hemodynamic and respiratory instabilities or signs of perforations such as peritonitis and subcutaneous emphysema requiring immediate surgical management and resuscitation, other signs include retro-sternal pain, and epigastric pain or buccopharyngeal lesions, which reflect neither the extent nor the severity of the lesions [15].

The clinical presentation then includes digestive and extra-digestive signs:

2.1 Digestive Signs:

- **Abdominal pain**: diffuse or epigastric.
- **Vomiting**: alimentary or more often hemorrhagic.
- **Oral and pharyngeal pain**.
- **Dysphagia.**
- **Hyper-Sialorrhea**.

2.2. Extra-digestive clinical signs:

- **skin burns.**
- **Chest pain.**
- **Cervical or thoracic subcutaneous emphysema**: signs of oesophageal perforation.
- **Hypothermia or hyperthermia.**
- **Respiratory distress :**
  - Of ENT or epiglottic origin.

- Pulmonary damage by inhalation.
- Lesion of the tracheo-bronchial tree.
- Metabolic origin by compensation of acidosis.

- **Hemodynamic distress:** Initial state of shock:
  - Hypovolemic: hematemesis of great abundance.
  - Septic: most often on pulmonary infection.
  - Cardiogenic or Vasoplegic of toxic or medicinal origin, if the notion of toxic-drug association is present.
- **Neurological Distress:**
  - Disturbance of consciousness of septic or toxic origin.
  - Convulsions (metabolic disorders or toxic).
- **Psychic disorders**: Behavioral disorder, anxiety, agitation, prostration…

3. Clinical examination:

- **Lesions of the ORL sphere:** Mouth ulceration or necrosis.
- **Pulmonary auscultation:** swelling or crackles.
- **Defense and abdominal contracture.**
- **Abdominal dullness:** a peri-hepatic sign of gastric perforation.
- **Abdominal distension:** occlusion of functional origin (reflex ileus).

VI. ENDOSCOPIC DIAGNOSIS:

Digestive endoscopic exploration is so far the Gold-standard for diagnosis and management of caustic ingestion cases for several reasons, widely used in all studies, performed in 94.8% of patients according to ELHAMMOUMI in his work carried out at the UCV department of the CHU IBN-SINA [11].

This examination allows the exploration of the upper digestive tract, by the direct visualization of the caustic lesions, and thus, establishing exact cartography in the cases of digestive attack, helps to avoid the patient and the hospital hospitalizations and unnecessary treatments. in the absence of lesion. It also plays an important role in medico-surgical management, which is essentially based on fibroscopic grading. [16]

However, the digestive fibro-endoscopy has certain limits, therefore requiring the realization of the examination by the hands of an experienced operator, as well as a good preparation of the patient, to avoid under or overestimating the lesions, and the complications it can cause due to its invasive nature.

In our study, endoscopy had a major place, carried out systematically in almost all of our patients (85.7%) apart from those who presented with digestive perforation (N=4).

1. Principle and technique:

• TECHNICAL: [17], [18]

Digestive endoscopic exploration is an invasive procedure that must be performed by experienced hands in favorable conditions with a team made up of a nurse anesthetist and an assistant, preferably with a resuscitator and visceral surgeon for patients who are unstable or at major risk of complications.

The digestive endoscopy technical platform must ensure optimal conditions for the work of the medical and paramedical team, as well as comfort and safety for patients, by meeting well-defined standards, in particular the dimensions of the room "of at least 25m2 on the ground", cleanliness by establishing a cleaning circuit "Sorting DASRI and DAOM waste" and compliance with disinfection procedures for the room and equipment.

Instrumentation equipment must be adapted to diagnostic and therapeutic practices, with good maintenance and storage, ensuring a sufficient quantity of consumables. This equipment is essentially made of a flexible endoscope, equipped with a lighting device and a visualization system, with the possibility of suction and air insufflation, which can be associated with endoscopic hemostasis tools such as electrocoagulation forceps or clips if needed.

Regarding fibroscopy carried out late revealing the presence of caustic stenosis, mechanical dilation would be indicated and will require candles with appropriate size and caliber guides

The nasogastric sounding can facilitate the gesture by washing or aspiration, but its indication is done cautiously and the risk of perforation must always be put forward.

The endoscopic gesture is generally done under sedation, for a double comfort of the patient and that of the operator, ideally in the presence of a resuscitator.

• Precautions: [16]

- Slow and careful introduction through the crico-pharynx under visual control.
- Avoid blowing in air as much as possible.
- Avoid retroversions and retroflections of the probe.
- Use extra caution through high severity areas.
- Do not force the passage in the event of high-grade necrosis or impassable areas.

2. Indication:

In the majority of the studies carried out, such as that of ZARGAR et al. [16] this was a Gold-standard with a broad indication for all patients. However, some studies have limited the indication of this examination which has proven to be useless, expensive and consuming several resources in 23% according to the study by CABRAL et al. [19]

In another study by NUNEZ et al. The male sex, the circumstance of autolysis, and the presence of clinical signs as well as the nature of the product ingested were predictive factors of high-grade lesions, since patients with less than two of these factors had a 91% probability of having a normal endoscopic examination or in favor of low-grade lesions requiring only conservative management [20], which has prompted some authors to suggest limiting the indications, indeed, CABRAL et al. Spared endoscopy for some asymptomatic patients who had ingested less than 125ml of the mildly corrosive agent in an accidental context.

Matthieu Bruzzi and Mircea Chirica excluded endoscopy from the initial management of caustic ingestions in 2015, it only had its place in patients who complained of swallowing disorder after the acute phase of the episode and in the treatment of strictures.

In our study, all our patients benefited from esophageal fibro-endoscopy, except for some who presented with digestive perforation (N=4). The latter is a formal contraindication to this examination.

3. Delay: [15] [16]

In the majority of studies, digestive fibroscopy was performed between the first 6 and 96 hours after ingestion, with the possibility of repeating remote controls.

It was suggested in a study conducted by E. SARFATI, to redo an endoscopic examination after 48 hours in the event of an incomplete, unsatisfactory, or inconclusive initial assessment. While advising against endoscopic exploration between the 5th and 15th day following ingestion according to the studies of A. ZARGAR and E. SARFATI, this is to avoid perforation due to the passage of the probe through a still friable wall and brittle.

A fibroscopy control was strongly suggested between the 3rd and 4th week, then repeated monthly until confirmation of healing of mucosal lesions or in a therapeutic setting for dilation of digestive stenoses.

4. Result: [15], [16]

The endoscopic evaluation is organized in the form of scores, the most used of which are made by **ZARGAR** and **DI COSTANZO**, which are closely similar:

• **ZARGAR CLASSIFICATION**: [17]

- **Grade 0:** normal.
- **Grade 1:** Erythema, edema.
- **Grade 2**: Mucosal ulceration.

a. Superficial ulcerations, false membranes, mucous hemorrhages.

b. Burrowing and confluent ulcerations.

- **Grade 3:** Mucosal necrosis.

has. Focal (non-circumferential) necrosis

b. Diffuse (circumferential) necrosis

• **DI COSTANZO CLASSIFICATION: [22]**

- **Stage 0:** Normal mucosa.

- **Stage I:** Erythema, edema, and mucosal congestion.

- **Stage II**: Mucosal ulceration.

a. Superficial, longitudinal.

b. Circumferential or two ulcers on the same floor "Kissing ulcers", weak bleeding.

- **Stage III:** Deep ulcerations, profuse bleeding, area of necrosis

a. Localized necrosis.

b. Extensive necrosis.

- **Stage IV:** Total necrosis, pan-parietal, significant hemorrhage, perforation.

NB: In our study, the Zargar classification was used by endoscopists.

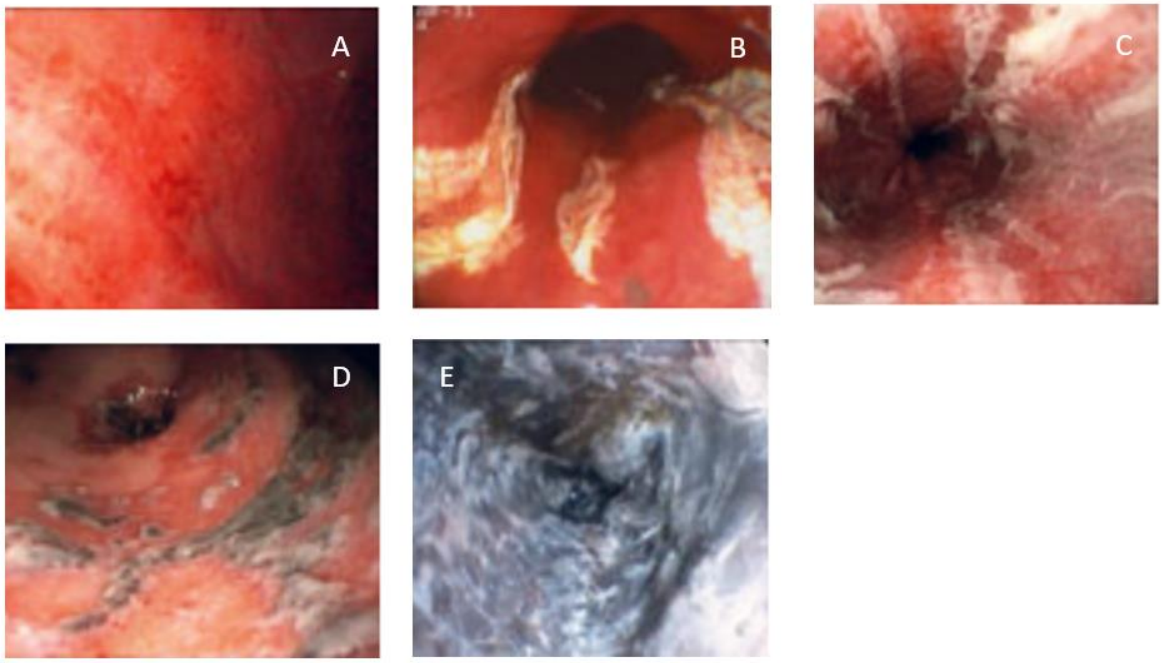


Figure 17: Endoscopic images showing the different stages of ZARGAR:

A. Stage 1: congestive edema.

B. Stage 2a: Superficial ulcer and false membranes.

C. Stage 2b: Confluent ulcers.

D. Stage 3a: Localized necrosis.

E. Stage 3b: Confluent necrosis.

• FREQUENCY OF LESION STAGES:

According to the study conducted by ZARGAR, grade 2a lesions are the most frequent lesions, followed by more severe grade 3 lesions that cause complications:[16].

- Grade 0 (8,6%).
- Grade 1 (12,4%).
- Grade 2 (40,7%) with a predominance of the first degree “2a”.

- Grade 3 (38,3%).

The same results are found in various studies, in particular in the one carried out at UCV by EL. HAMMOUMI with 72% of lesions classified 2 and 51% 3 according to the DI COSTANZO classification[11].

In our study, stage 2b was the most frequently found, in 33.3% of patients who underwent endoscopy, followed by stages 3a and 3b.

• Site of the attack: [11], [16], [21]

In the vast majority of studies, both the esophagus and the stomach were affected, 81.5% in the ZARGAR study versus 100% in the EL HAMMOUMI study.

Isolated topography damage is more frequent in the stomach than in the esophagus, with a percentage of 58% of isolated gastric damage according to EL HAMMOUMI. According to DI COSTANZO, gastric involvement was revealed by endoscopy in 70 out of 76 patients, including 40 cases with associated esophageal involvement and 30 isolated gastric involvement.

The esophageal portion most responsible for lesions is the lower esophagus, while at the level of the stomach it is the antrum, This can be explained by a gravitational reason, in fact, in a standing or sitting position, it is the lower sloping parts of these two organs and essentially the stomach which collects the most ingested erosive product.

Duodenal involvement is rare, this is explained by a reflex pylorospasm which limits the extension of the corrosive product downstream. [22]

All the studies confirm the lack of correlation between the severity of oropharyngeal lesions and the presence of upper digestive tract lesions.

These results converge with those of our study the associated attack on the esophagus and the stomach was the most encountered in 83.3%. On the other hand, no isolated attack of the stomach was found, against a single esophageal attack alone, i.e. 4.2%. We had 3 duodenal lesions all associated with esophagogastric lesions.

5. Interest: [16], [22]

The digestive fibroscopy has a double interest in the ingestions of caustics, diagnostic and in certain cases, therapeutic.

The main advantage it offers is the direct visualization of caustic lesions, allowing firstly to make the mapped diagnosis of the lesions, and secondly to assess their severity by classifying the lesions according to the ZARGAR or DI scores. COSTANZO described above. Therefore, fibroscopy also plays the role of guiding management and clarifying the prognosis, it also makes it possible to predict complications and evolution according to grade. The prospective ZARGAR study assigned early complications (GI bleeding, perforation, esophageal-tracheal fistula) to Zargar stage 3, and stricture formation to grade 2b or higher.

Endoscopy also helps in follow-up by the possibility of controls, until confirmation of complete healing.

In case of esophageal stenosis, dilation is necessary. Digestive fibroscopy then offers the choice between mechanical dilation using guides and candles, and pneumatic dilation.

• Interpretation limit:

Some limitations call endoscopy into question, mainly concerning the interpretation of the lesions, indeed, some authors describe the blackish necrotic lesions as being a deposit of hematin at the level of the mucous surface formed by the acid after bleeding, and not necessarily reflecting a deep parietal attack. [16]

The false membranes are an element making the parietal exploration vicious, these cover the external surface making it difficult to assess the depth of the attack. For this, E.SARFATI suggests the presence of a surgeon during the endoscopic examination to support a good interpretation [15], as long as ZARGAR offers control endoscopic examinations within 48 hours.

In some cases, endoscopy leads to an overestimation of the lesions, thus leading to the performance of heavy surgical procedures, which may prove to be useless or only aggravate the patient.

Significant edema, especially at the level of the upper or lower esophageal sphincter, can be seen in certain situations, where the passage of the endoscopy probe becomes difficult or even dangerous, which implies the absence of an evaluation of the lesions upstream of the edema. This situation was encountered in our study in 2 patients.

Endoscopy thus has certain limitations in the interpretation of mucosal necrosis as well as in the trans-parietal exploration of the digestive tract and the assessment of the state of neighboring organs.

VII. SCAN DIAGNOSTIC: [23]–[27]

Until today, the interest in the scanner as an initial assessment of the ingestion of caustics remains uncertain. CT is mainly performed postoperatively or after the patient has worsened. This is considered an inadequate examination for the evaluation of upper digestive tract lesions after ingestion of caustics by several authors, practitioners, and radiologists [16].

Hence the purpose of our study is to define the place of the scanner. Some authors have opened the question for discussion, by comparing CT and endoscopic results with postoperative

1. Technical:

In the sum of the various studies carried out, there are some differences in reported techniques, mainly concerning the acquisition region, Y. LURIE and V. GAULT in their studies proceeded by thoracoabdominal CT without and with an injection of contrast product iodized, while M. CHIRICA was modestly wider, including the cervical region in his field, this is explained by the prospective nature of his study, allowing him to control certain parameters.

In our retrospective study, the majority of CT scans were performed as lesional assessment in the first hours of ingestion, including the cervical and Thoraco-Abdominal regions (89%), with and without injection of contrast product, however, some patients only benefited from a Cervico-Thoracic scanner (3.5%) where the bulb, duodenum, and pancreas were not visible or Abdominal (7.1%) performed for post control -operative where the thoracic and cervical esophagus are not assessable. In only one patient the injection was not performed because of severe renal insufficiency.

In the prospective studies of MATTHIEU BRUZZI and MIRCEA, the scanning technique was well developed:

- Scanner: “Philips Medical System Brilliance 40 CT” 40-barette multi-detector.
- Timing: initial, after the endoscopic examination.
- Cervico-Thoraco-Abdominal CT scan, without and with an injection of iodinated contrast product.
- Injection: IOMERON 350 2ml/Kg or Xenetix 350 at a speed of 2-3 ml/s
- Acquisition: arterial: 18-25 seconds, portal 90 seconds, with a delay of 3-12 hours from ingestion of the corrosive.
- Thin cuts 2mm /1mm soft reconstruction.
- Endoscopy was performed first in patients with renal insufficiency, with intravenous hydration if a CT scan was required unless the clinical-biological or endoscopic data immediately indicated urgent surgery.
- The reading is done by the radiologist available when the examination is carried out.
- The radiological analysis was based on the search for transmural necrosis defined by the presence of 2 or 3 of the following criteria:
    - Infiltration of peri-digestive fat after injection of contrast medium.
    - Infiltration of the digestive wall after injection of contrast medium.
    - Absence of enhancement of the esophageal wall.

Oral digestive opacification with contrast product is a complementary means allowing the detection of perforation, so-tracheal fistula, or more rarely oesopleural as well as to look for stenosis or as part of a postoperative assessment. Its indications are limited to the suspicion of perforation or post-operative complication and should be avoided in other patients because its administration may aggravate necrotic lesions or be responsible for inhalation in patients at risk. [42]

2. Radiological semiology:

After the ingestion of caustics, several radiological signs have been described, poorly known, and interpreted until now, some studies have tried to group them and set exact definitions, based on the experience of radiologists specializing in gastrointestinal diseases. intestinal.

We cite in our work two major studies on the ingestion of caustic on which we based ourselves, that of M. CHIRICA and Y. LURIE, the classification of CHIRICA seeming more detailed, taking into account the enhancement after injection of the product of contrast seemed to us to be more precise in the analysis of CT images.

Several signs described in the literature have also been reported in our patients:

a. Parietal thickening:

The evaluation of the gastric wall in normal times requires preparation before and during the examination, with a markup with water to obtain sufficient gastric distension, making it possible to evaluate the parietal thickness.

A study conducted by PJ. PICKCHARDT concludes with an average value of the thickness of the gastric antrum of 5mm, on the condition of complete distension, with variations along with the other parts of the stomach. In distality, a physiological thickening was noticed which can reach 12mm. [28]

Thus, given the difficulty of carrying out this preparation, gastric thickening remains a subjective sign, which is essentially taken into consideration if the values exceed 12mm of maximum thickness or are associated with submucosal edema.

About the esophagus, the CT evaluation of the parietal thickness has been studied by a few authors, in a South Korean study conducted by Ryu et al., An esophageal wall strictly greater than 3mm is considered thickened in its classification. Reining et al. Have demonstrated in another study that a thickness exceeding 3mm is linked to a tumoral or inflammatory pathology. [29]

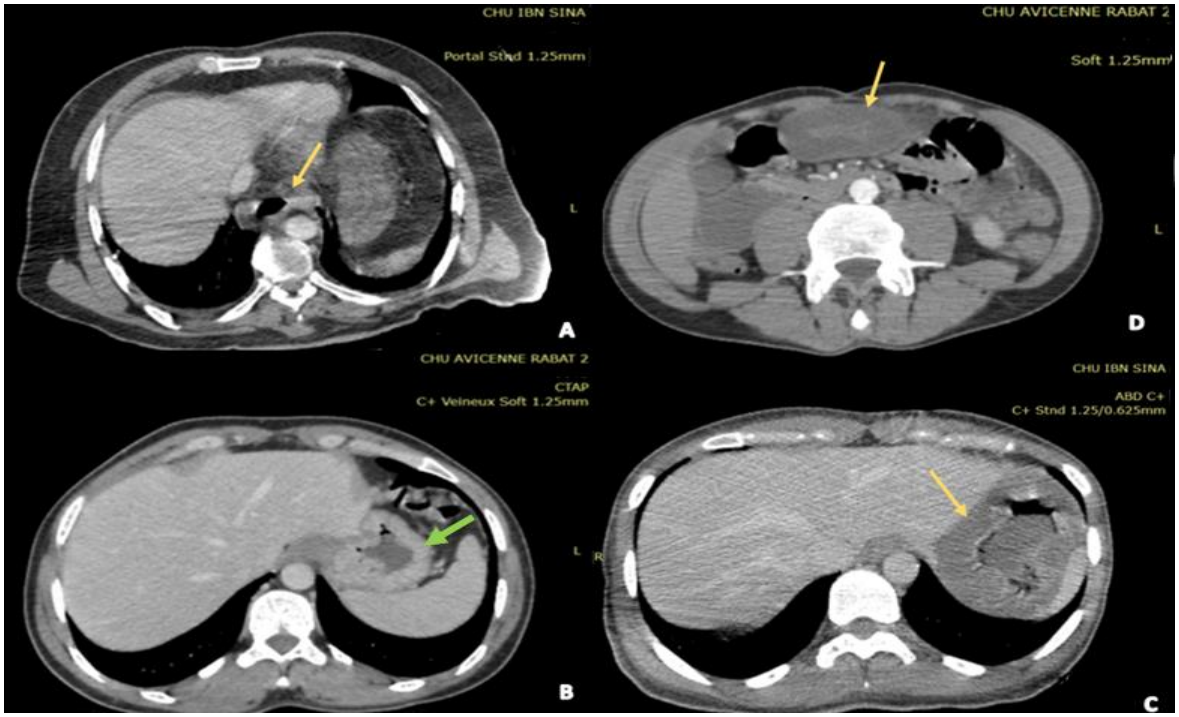


Figure 18: Axial TAP CT scan showing digestive wall thickening:

A. Section through thickening of the lower esophagus (yellow arrow)
B. Gastric wall of normal thickness and enhancement (green arrow)
C. Section through gastric thickening with enhancement defect (yellow arrow)
D. Section showing pyloric thickening with submucosal edema and target enhancement (yellow arrow)

b. Submucosal parietal edema:

Submucosal edema appears on imaging as intra-parietal hypodensity responsible for parietal thickening, not enhancing after injection of contrast product. It can be associated with an external enhancement of the digestive wall and the mucous membrane, testifying to a parietal inflammatory reaction associated with hyperemia. As it can testify to parietal necrosis at its advanced stage where no parietal enhancement will be noted.

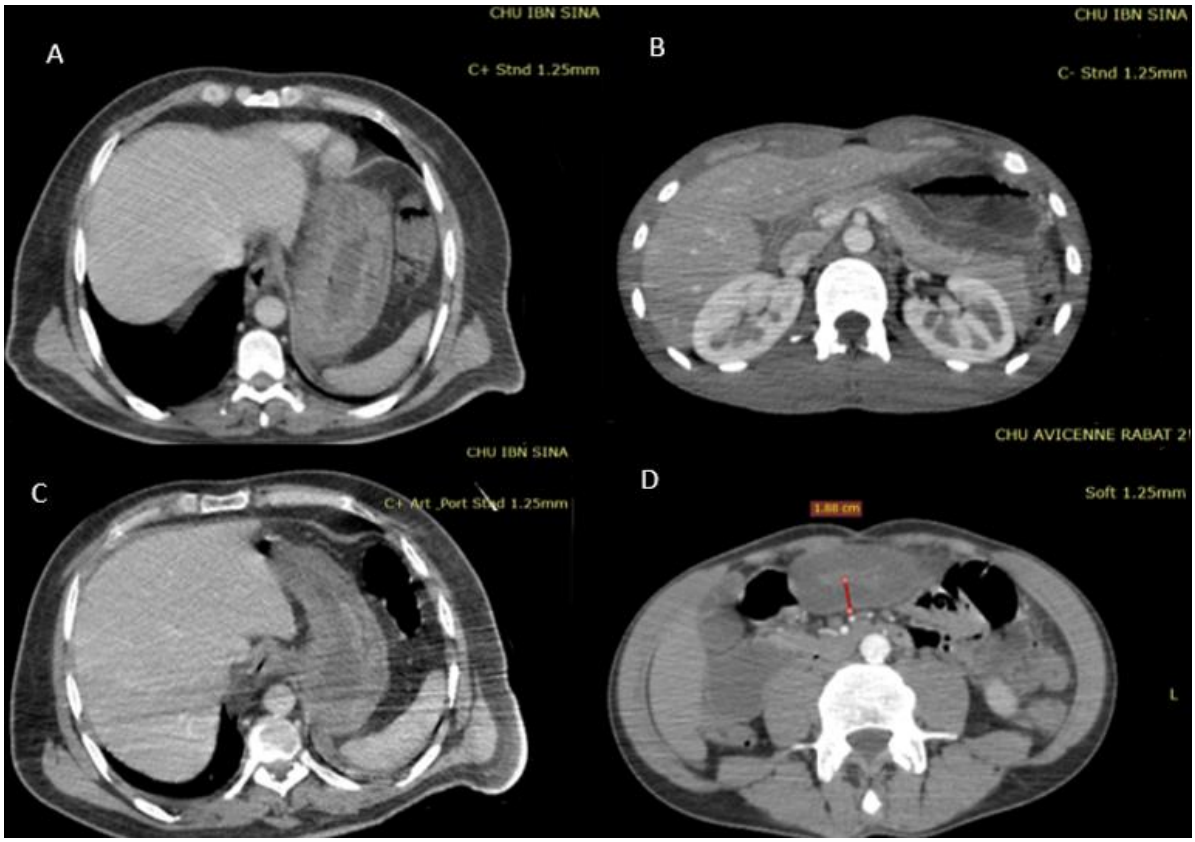


Figure 19: CT scan axial sections showing submucosal edema:

A + B. Sections passing through a gastric submucosal edema with enhancement of the mucosa and the external wall.

C. Parietal edema with no gastric wall enhancement.

D. Section through pyloric thickening with submucosal edema and target enhancement.

c. Parietal enhancement:

A normally vascularized digestive wall is enhanced in the portal phase, after injection of iodinated contrast product.

This element corresponds to a major sign in the evaluation of the digestive wall after ingestion of corrosive products, making it possible to detect trans-parietal necrosis. In ascending order of severity, there are 3 aspects of enhancement:

• NORMAL PARIETAL ENHANCEMENT:

After injection of the iodinated contrast product, the digestive wall is normally enhanced homogeneously in the portal phase.

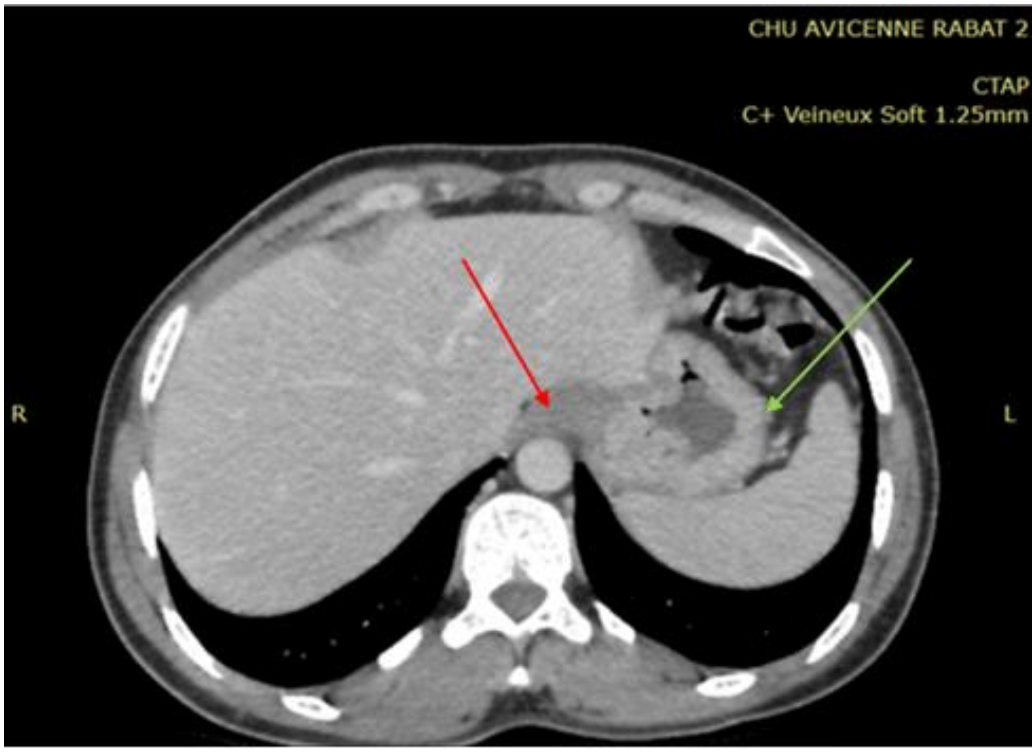


Figure 20: Abdominal axial slice in portal phase, showing normal gastric parietal enhancement (green arrow) and absence of enhancement of the lower esophagus and the esophagogastric junction (red arrow)

• UPGRADE OF "TARGET SIGN" PARIETAL RAISING:

Corresponding to a parietal reaction due to a caustic attack. The enhanced mucosa appears hyperdense, associated with an external parietal enhancement which seems to testify to inflammatory hyperemia without trans parietal necrosis.

NB: It should be noted that the mucosa may appear spontaneously hyperdense about active bleeding, which may be mistaken for enhancement, hence the interest of the examination without C- injection.

Figure 21: Axial section of a C+ CT scan passing through the pylorus: Target sign (green arrow)

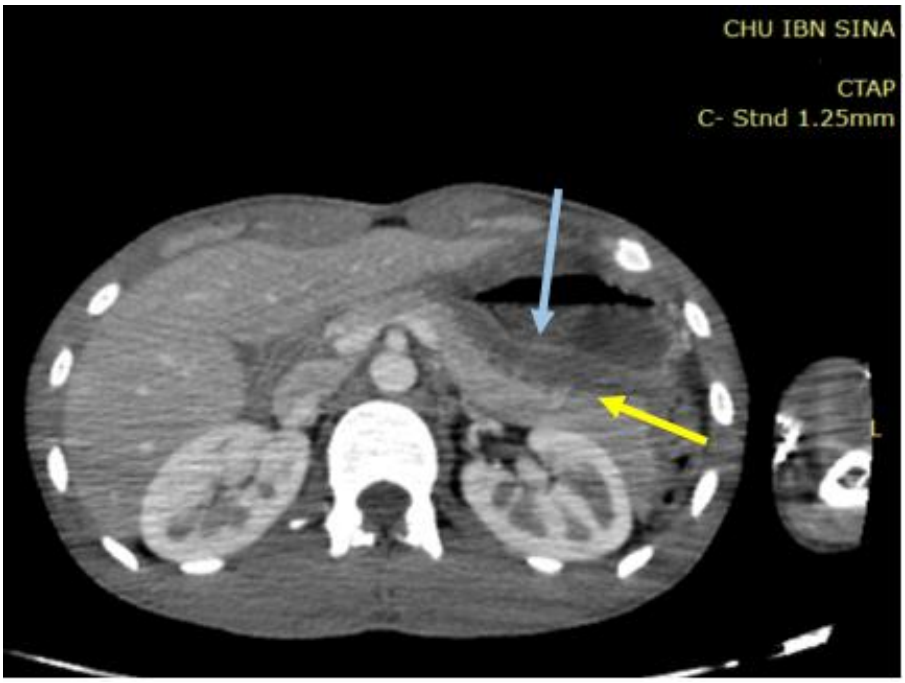


Figure 22: Axial section of a C+ CT scan, passing through the abdomen, showing mucosal enhancement (blue arrow) with external parietal hyperemia (yellow arrow)

.

• ABSENCE OF PARIETAL ENHANCEMENT:

The absence of parietal enhancement is the most suggestive sign of necrosis. Objectified after injection of iodinated contrast product in the portal phase and often associated with infiltration of adjacent fat or involvement of neighboring organs.

The absence of mucosal enhancement is justified by its necrosis, this sign may be associated with peripheral enhancement of the digestive wall signifying superficial necrotic involvement of the mucosa only, as it may be generalized to the entire digestive wall in connection with transmural necrosis

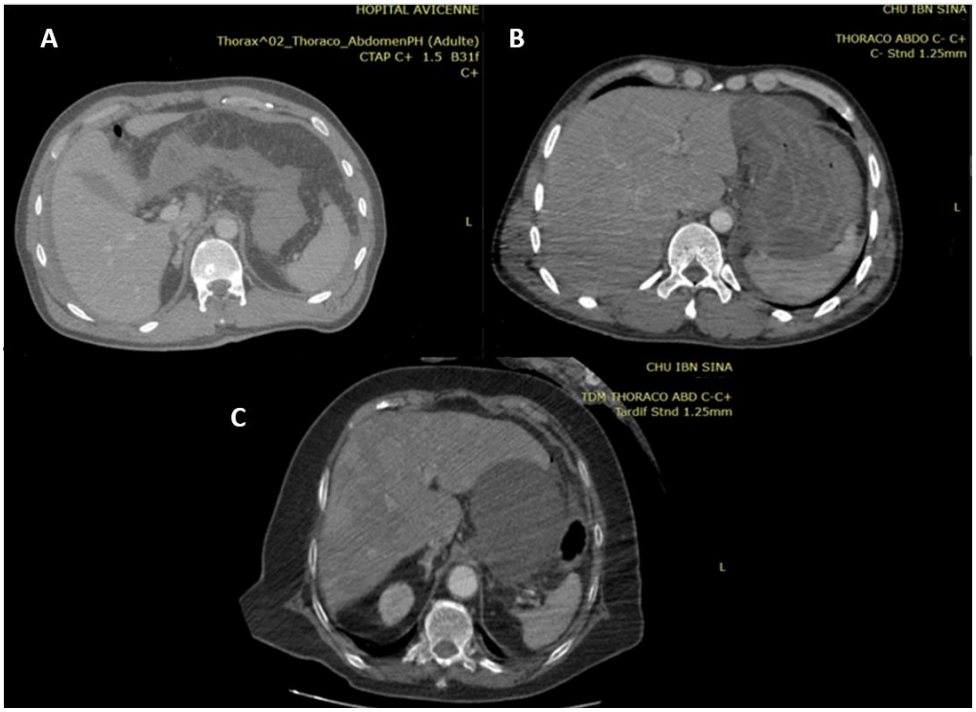


Figure 23: Axial sections of injected abdominal CT of different patients showing transmural necrosis:

A. Absence of parietal enhancement with retraction and deformation of the gastric contours.

B. Absence of gastric enhancement, associated with a hyperdense aspect of the mucosa related to hemorrhagic streaks.

C. Absence of enhancement of the fundal wall, related to transmural necrosis.

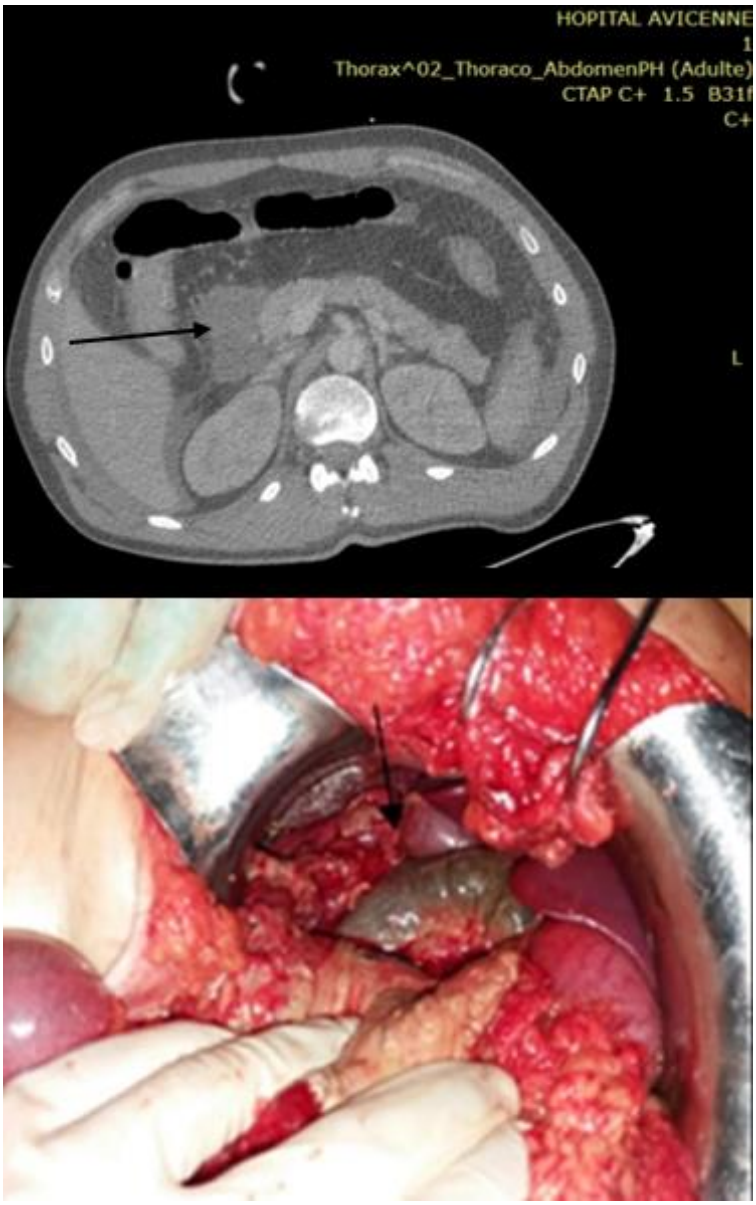


Figure 24: Images demonstrating transmural duodenal necrosis in a patient who ingested 3 glasses of spirits of salt (black arrows).

A. Axial section of an abdominal CT showing the absence of duodenal enhancement related to duodenal necrosis.
B. Intraoperative image of abdominal exploration highlighting duodenal necrosis.

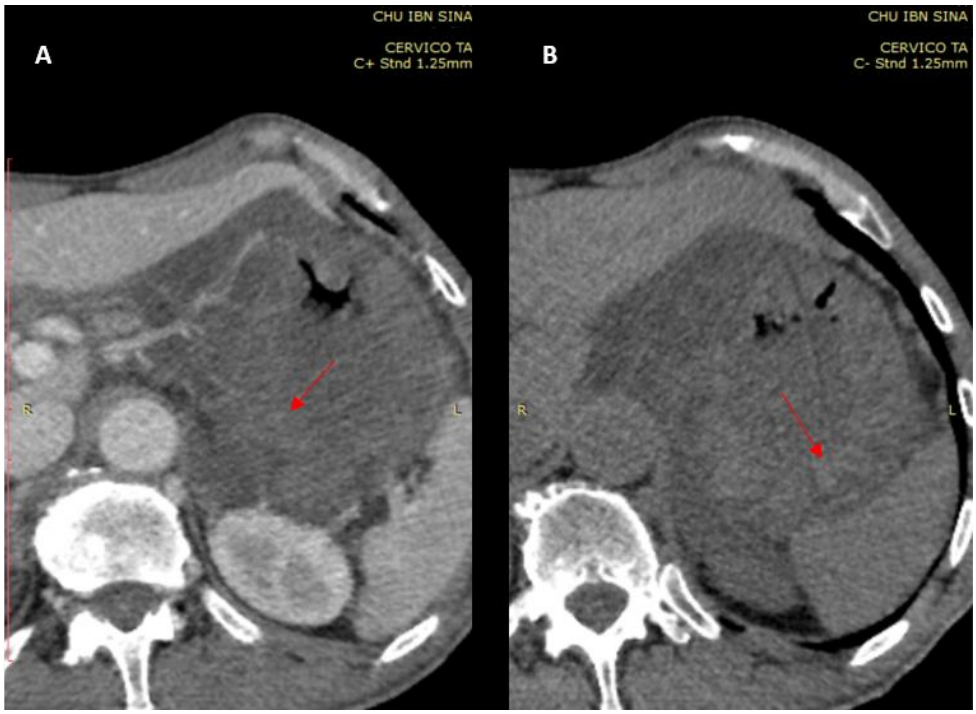


Figure 25: Axial sections of the same patient, presenting trans-parietal gastric necrosis of caustic origin. Objectifying a spontaneously hyperdense appearance of the non-enhanced mucosa (red arrow) in connection with a necrotic and hemorrhagic mucosa.

A. Axial slice after injection of in portal phase.
B. Axial slice in spontaneous contrast.

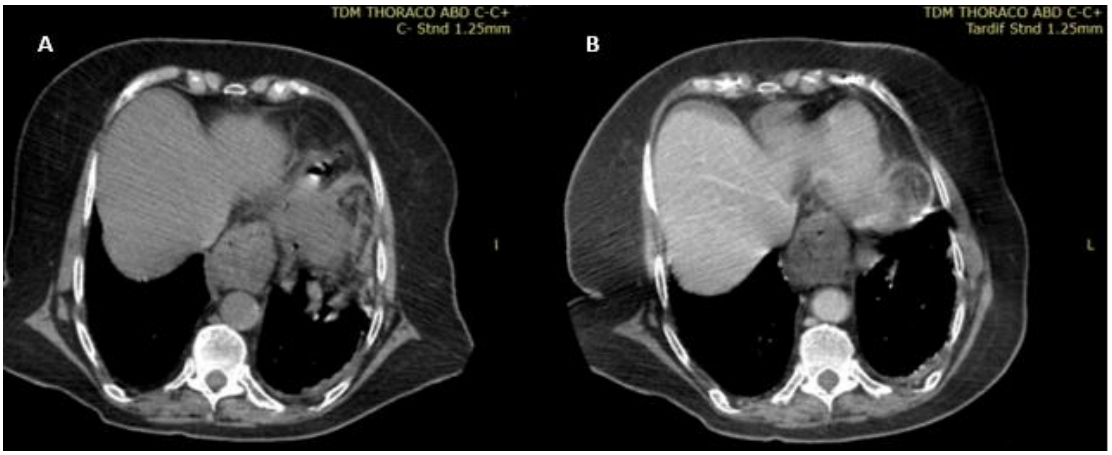


Figure 26: Axial sections demonstrating transparietal esophageal necrosis with dilation of the esophagus. A. Without injection, B. After injection in the portal phase

d. Infiltration of peri-digestive fat:

Is characterized by a dense aspect of the fat which is normally hypodense, and is seen at the peri-esophageal level at the mediastinal level, and the level of the peritoneal fat at the abdominal level. Hypervascularization may be associated.

May be associated with parietal edema, trans-parietal necrosis, pancreatitis, or peritonitis.

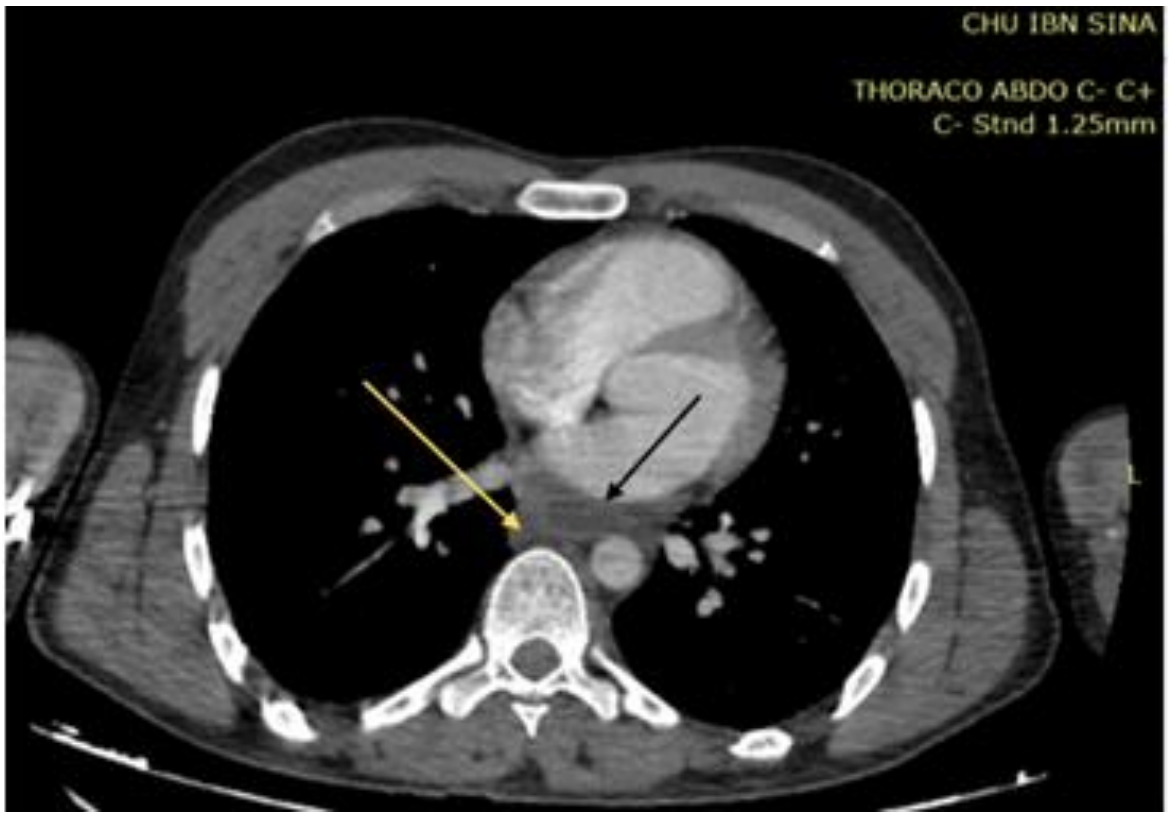


Figure 27: Axial section of a C+ CT, in portal phase, showing infiltration of the mediastinal fat (yellow arrow), with esophageal dilation (black arrow) after ingestion of the caustic product.

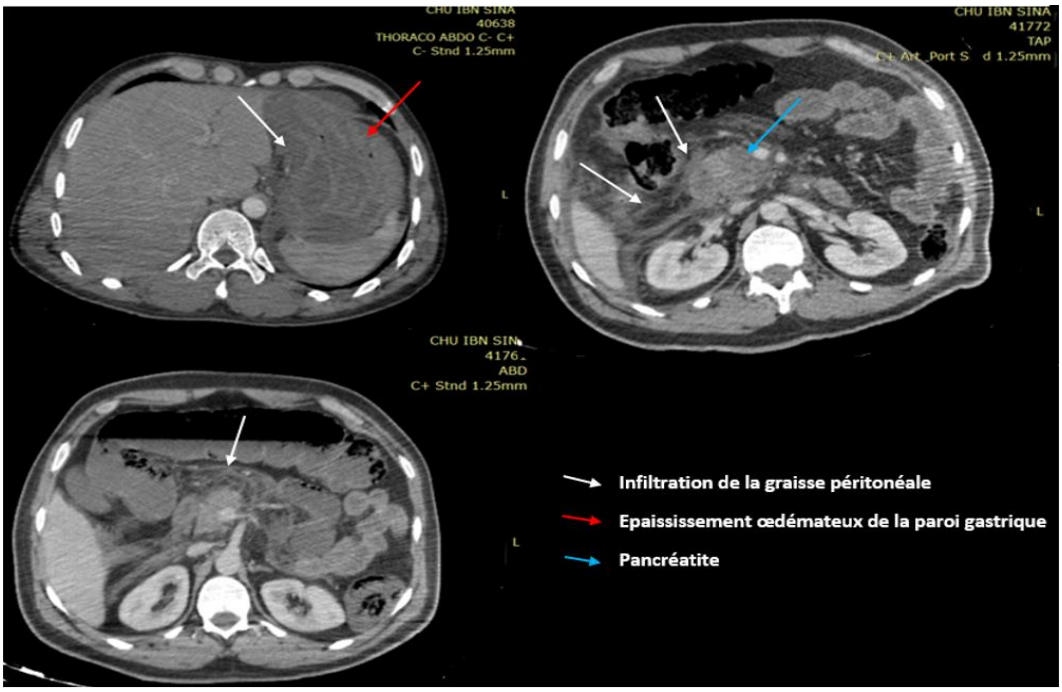


Figure 28: Captioned axial sections of a CT scan after injection of contrast medium of different patients, showing infiltration of peritoneal fat after ingestion of a caustic agent.

e. Digestive dilation:

Dilatation is a frequently found sign. It is the consequence of caustic stenosis, LES, or pyloric spasm associated with stasis.

A. Dilatation of the lower esophagus (white arrow) with stasis on lower esophageal stenosis and trans-parietal necrosis.
B. Dilatation of the middle esophagus (white arrow) with air-fluid level.
C. Dilatation of the lower esophagus with parietal thickening.

Figure 29: Axial sections of Thoracic CT of different patients, showing esophageal dilation

It is important to look for stenosis if it is visible and to evaluate the lung parenchyma because the risk of complications by inhalation is high in the event of esophageal stasis which can worsen the vital prognosis.

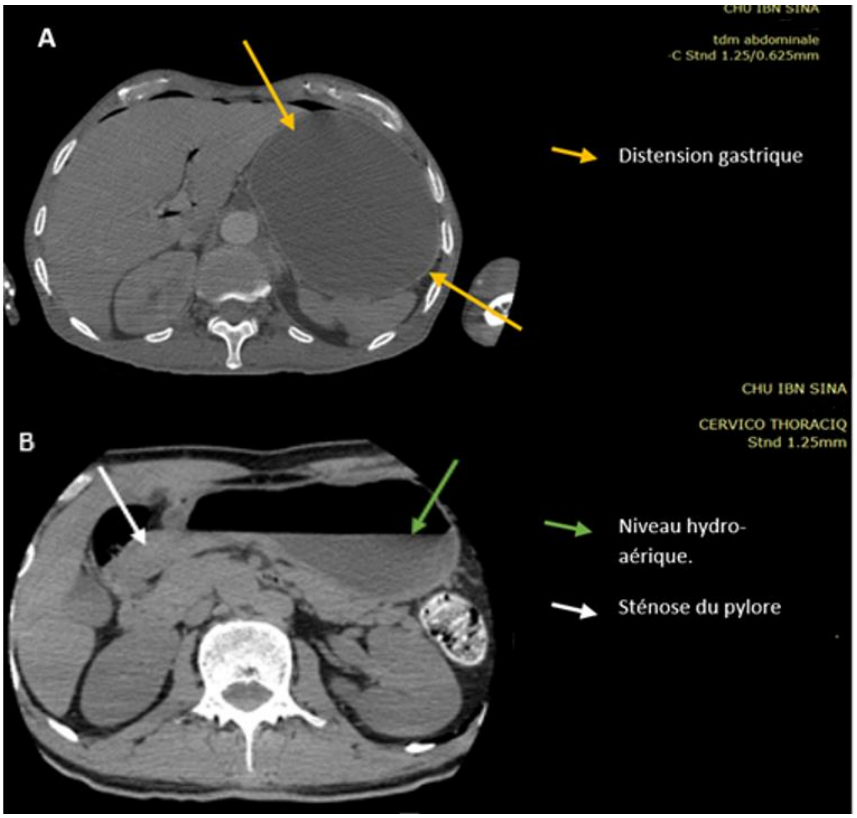


Figure 30: Axial sections of a CT in spontaneous contrast, objectifying a stomach of stasis on a caustic spasm of the pylorus.

f. Parietal emphysema:

Parietal emphysema is a pejorative sign, testifying to parietal suffering at a pre-rupture stage or associated with parietal perforation.

Other associated signs are described, as the direct or indirect complications caused by the ingestion of caustics:

g. Perforation:

Hollow organ perforation is essentially manifested by the presence of Pneumomediastinum by esophageal perforation or pneumoperitoneum by perforation of the stomach or duodenum.

Direct sign: the issue of air bubbles from the lumen to the peritoneum or the mediastinum through a solution of parietal continuity.

Indirect signs: Peritoneal/pleuro-pericardial and mediastinal effusion, infiltration of mediastinal or peritoneal peri-digestive fat, Pneumoperitoneum, and pneumomediastinum, collections.

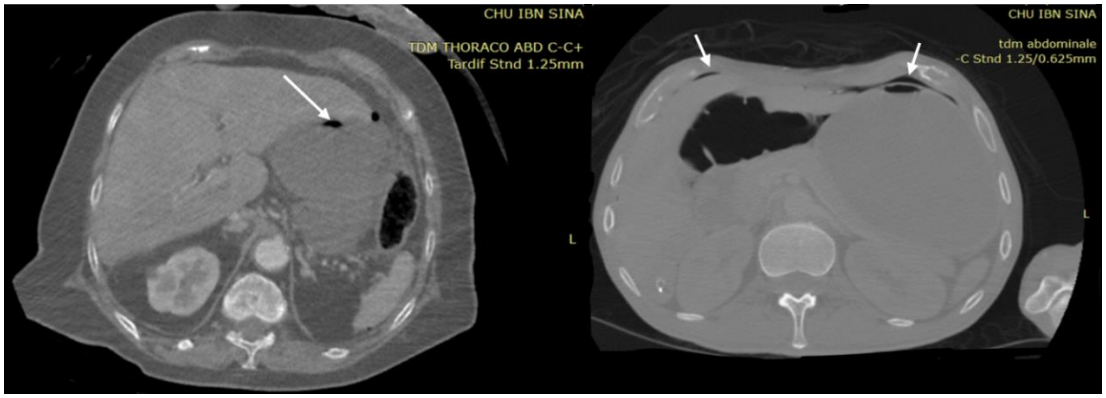


Figure 31: Axial CT scan showing gastric perforation.

A. Gastric perforation with bubbles of pneumoperitoneum (white arrow) and gastric necrosis.

B. Bone window: Pneumoperitoneum on digestive perforation in bone windowing (white arrows)

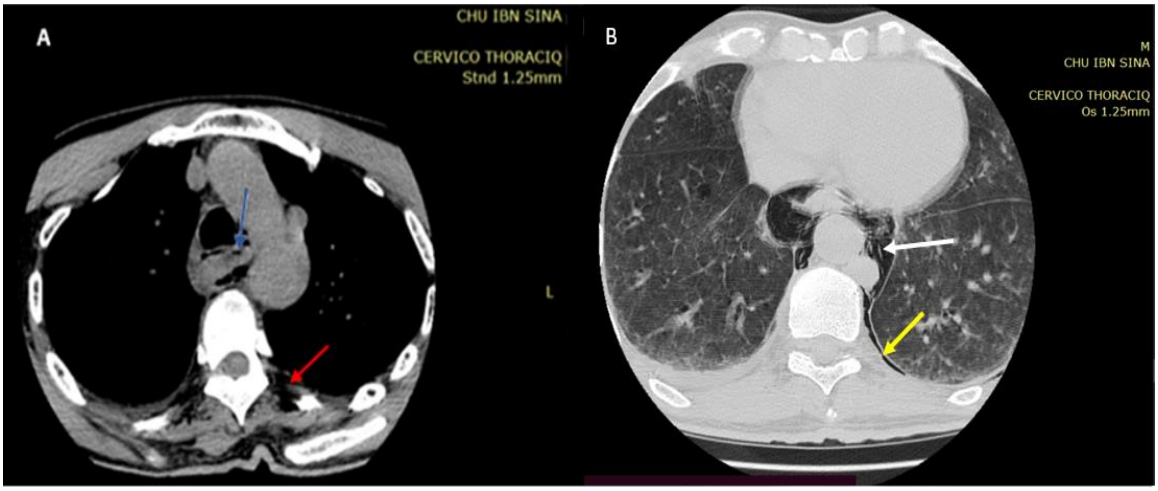


Figure 32: Axial sections of a CT scan at the thoracic level showing caustic perforation of the esophagus with parietal pneumatosis (blue arrow) 'A'; and pneumo-mediastinum (white

arrow), extended to the pleura ‘pneumothorax’ (yellow arrow) and the posterior chest wall ‘parietal emphysema’ (red arrow) ‘B’.

h. Pancreatitis:

We have described in our series 2 cases of lithiasis pancreatitis secondary to the ingestion of a caustic agent. Only a few rare cases have been described in the literature [30]. In a study carried out in the pediatric surgery department, 17 cases of acute pancreatitis after ingestion of caustics were reported over 10 years, this was explained by a mechanism of oxidative stress, which is summarized in the production of free radicals of oxygen and lipids by peroxidation, acting as a mediator of remote inflammation, following thermal or chemical trauma, this can affect the pancreas as well as other organs such as the kidneys or the lung [31]

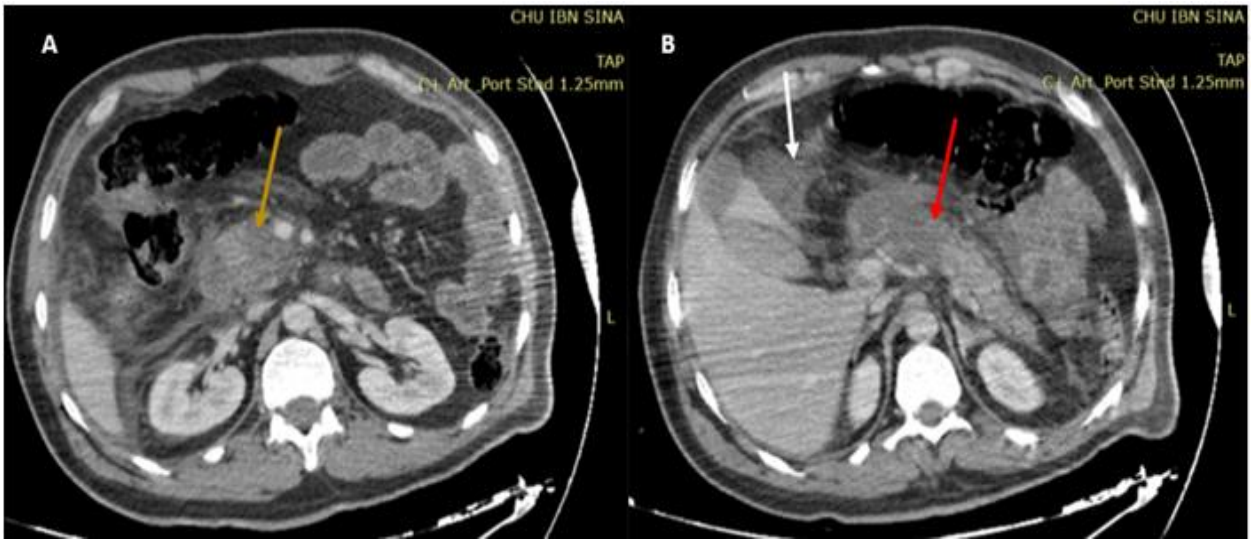


Figure 33: Axial sections of a C+ CT showing caustic necrotic pancreatitis.

A. Necrosis of the pancreatic head: absence of cranial enhancement and peripancreatic infiltration (yellow arrow) associated with peritoneal effusion.
B. Necrosis of the isthmus and pancreatic body (red arrow) with peritoneal infiltration (white arrow).

i. Mediastinitis:

Mediastinitis is a serious complication of caustic ingestion, due to perforation of the esophagus after caustic ingestion.

The role of the scanner is to look for stigmata of esophageal perforation if the clinic does not allow a precise diagnosis to be made.

This is mainly manifested by the issue of air bubbles towards the fatty, mediastinal, pleural, and pericardial spaces, associated with their infiltration or effusion. Collections may appear.

In our series, two cases of mediastinitis were described.

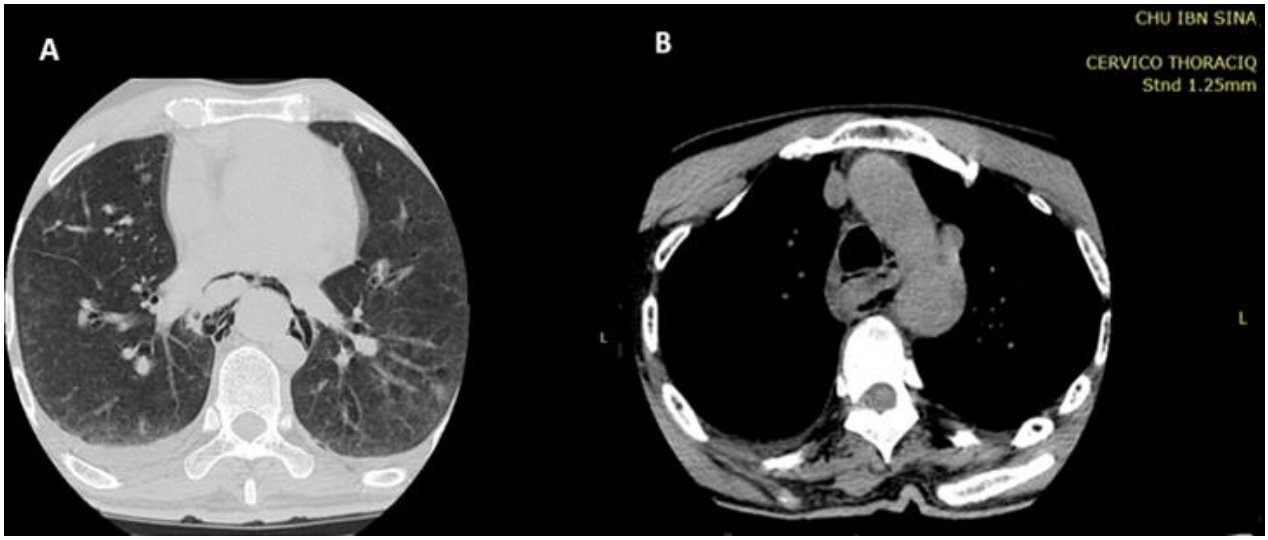


Figure 34: CT axial sections passing through the thorax, showing post-caustic mediastinitis. A: pulmonary window and B: mediastinal window, with air bubbles and mediastinal fat infiltration associated with bilateral pleurisy.

j. Peritonitis:

Peritonitis is a serious complication, often due to organ perforation with an obvious clinical presentation requiring surgical exploration, as it may be secondary to caustic necrotizing pancreatitis. The scanner is performed in case of diagnostic doubt to identify the cause and locate a possible perforation or deep collection.

Figure 35: Axial sections of an abdominal CT scan of a patient presenting with peritonitis on necrotic pancreatitis of caustic origin.

k. Active bleeding:

Bleeding is the most common complication of ingestion of caustic, this can be seen on CT in the event of active bleeding, by the spontaneously hyperdense and heterogeneous appearance of the gastric or esophageal contents, in the form of clots.

NB: the ingested caustic product may appear on the scanner in the form of a spontaneously hyperdense sloping liquid image, the frank density as well as the sloping character forming a liquid-liquid level makes it possible to differentiate it from active or coagulated bleeding, which tends to have a heterogeneous oval shape, irregular contours as well as a slight enhancement after injection of the PDC testifying to the activity of the bleeding.

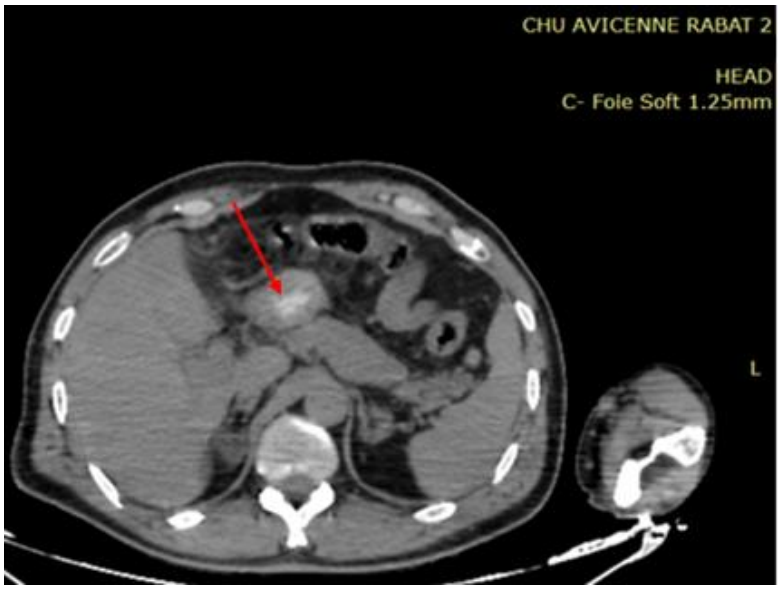


Figure 36: Axial section of abdominal C-CT showing a spontaneously hyperdense appearance of the pyloric lumen related to the ingested corrosive product that could be mistaken for active bleeding. (Red Arrow).

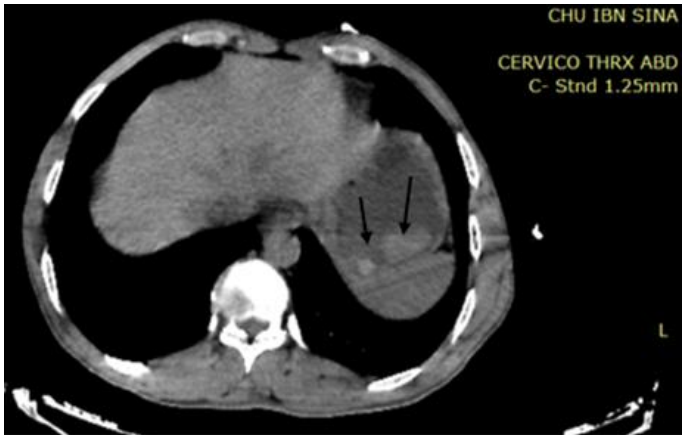
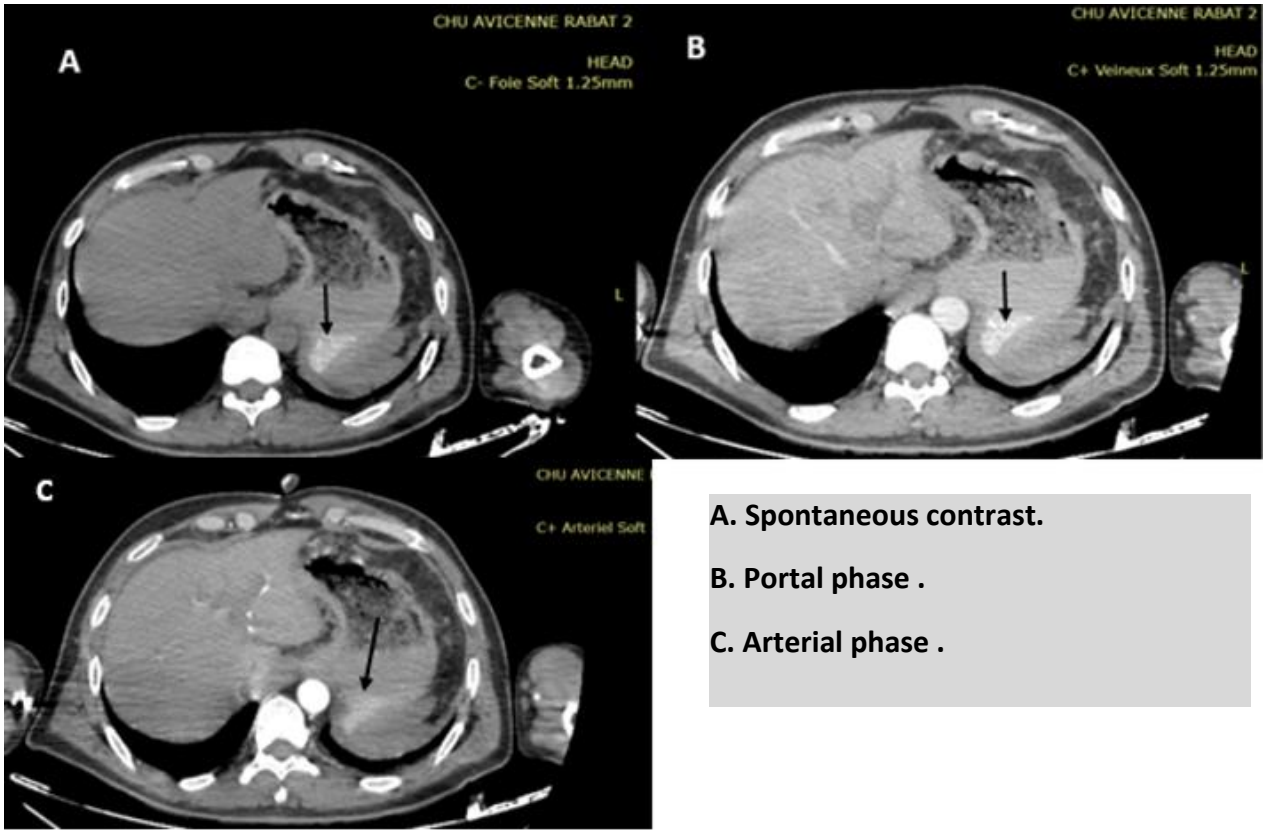


Figure 37: Coupe axiale d'une TDM abdominale passant par le corps gastrique, mettant en évidence des caillots de sang en rapport avec une hémorragie d'origine caustique.



A. Spontaneous contrast.

B. Portal phase .

C. Arterial phase .

Figure 38: CT axial sections before 'A' and after injection of iodinated contrast medium 'B', in a patient victim of caustic ingestion. Objectifying a spontaneously hyperdense aspect of the gastric contents without enhancement kinetics about the ingested product (black arrows).

l. Pneumonia:

It is a frequent complication, a consequence of inhalation or acquired on mechanical ventilation, and can appear from the admission or the days which follow.

The major signs of pneumopathy are tree-shaped micronodules and condensations, which may be associated with pleurisy.

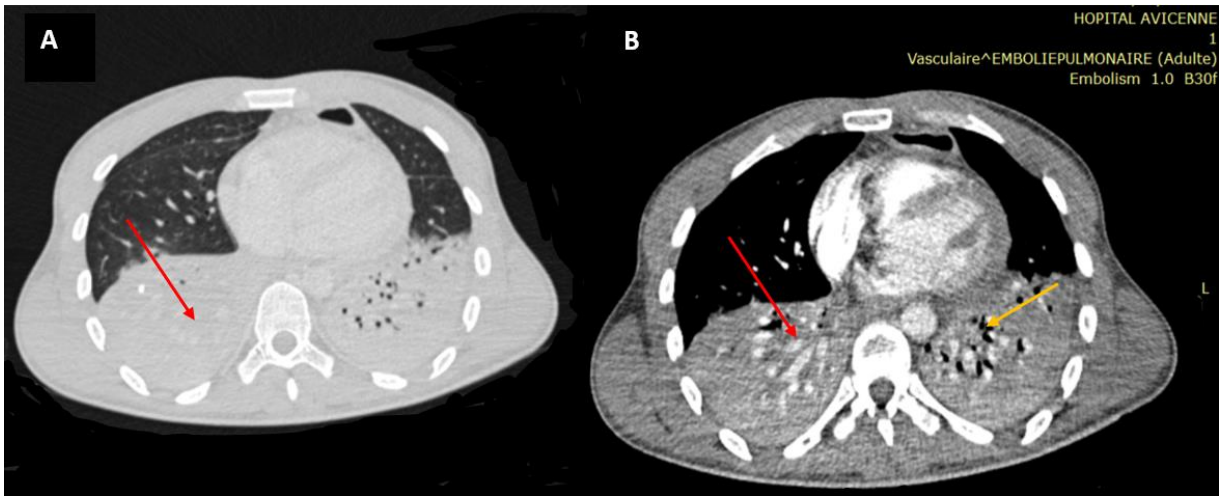


Figure 39 Axial sections of thoracic CT showing pneumopathy acquired on mechanical ventilation, in a patient operated by jejunostomy after ingestion of caustic.

A: pulmonary window showing a bilateral focus of condensation (red arrow)
B: mediastinal window showing bilateral foci of condensation (red arrow) with air bronchogram (yellow arrow).

3. Interpretation:

The interpretation of the images was initially carried out by the on-call radiologist present during the examination in all the studies.

A reinterpretation by radiologists specializing in gastrointestinal diseases was then imposed in the study by Bruzzi et al. Whereas in the study by V. GAULT about 14 cases, the scanners were interpreted by 3 independent radiologists.

In our study, the CT scans were first interpreted by the radiologist present during the examination, with subsequent re-reading to determine the severity score of the examination.

Finally, we retain three scores established in the literature:

Y. LURIE's Score: [25]

- **GRADE 0:** Normal appearance of the organs.

- **GRADE 1**: Parietal thickening due to submucosal edema.

- **GRADE 2:** Grade 1 with infiltration of peri-digestive fat.

- **GRADE 3:** Grade 2 is associated with one of the following signs:

• Parietal emphysema or air bubbles in the mediastinum or peritoneum.

• Mediastinal effusion or collection.

• Peritoneal effusion or collection.

The Score of BRUZZI and MIRCEA CHIRICA: [22]

- **GRADE I**: Normal

• Regular parietal contours, without submucosal edema.

• Contrast uptake after injection is moderate and above all homogeneous.

• Absence of mediastinal or peritoneal fat infiltration.

- **GRADE II:** submucosal edema, increased parietal contrast enhancement, infiltration of peri-digestive fat, divided into two subgroups:

•Submucosal edema is limited by mucosal enhancement and of the outer layer "target aspect".

• Enhancement of the outer layer without enhancement of the mucosa with submucosal edema 'Necrosis of the mucosa'.

- **GRADE III:** Transmural necrosis: Absence of parietal enhancement which may be associated with:

• Infiltration of the peri-digestive fat after injection of contrast medium.

• Infiltration of the digestive wall after injection of contrast medium.

The Ruy et al. : [33]

- **Grade I**: CT scan without anomalies.
- **Grade II:** Esophageal parietal edema with thickness >3mm without infiltration of the peri-oesophageal fat.
- **Grade III**: Esophageal parietal edema with thickness >3mm with infiltration of the peri-oesophageal fat.
- **Grade IV:** Esophageal parietal edema with thickness >3mm with infiltration of the peri-oesophageal fat associated with:

• Erasure of adjacent tissues.

• Perioesophageal collection.

In light of these scores and the radiological signs found in our patients, our interpretation was based on that of the Bruzzi et al. With some changes:

- **GRADE I:** Normal

• Regular parietal contours, without submucosal edema.

• Homogeneous parietal enhancement after injection of PDC.

• Absence of mediastinal or peritoneal fat infiltration.

• No signs of perforation or collection.

- **GRADE II:** submucosal parietal edema with increased parietal contrast enhancement that may be associated with peri-digestive fat infiltration: divided into two subgroups:

• IIa: Submucosal edema limited by mucosal enhancement and of the outer layer “target aspect”. Without infiltration of peri-digestive fat.

• IIb: Submucosal edema with the enhancement of the external layer without enhancement of the mucous membrane 'Mucous necrosis' + infiltration of peri-digestive fat.

- **GRADE III:** Transmural necrosis: Defined by the absence of enhancement of the wall with infiltration of peri-digestive fat after injection of PDC.
- **GRADE IV:** Presence of a complication:

• **Digestive perforation:**

- **Signs of peritonitis**: Pneumoperitoneum, peritoneal effusion, and Infiltration of mesenteric fat, solution of digestive continuity, Peritoneal collection.
- **Signs of Mediastinitis:** Pneumo-mediastinum, Mediastinal or pleuropericardial effusion, Infiltration of mediastinal fat, solution of continuity of the esophageal wall, mediastinal collection.

• **Oeso-tracheal fistula.**

VIII. EVOLUTION :

The evolution of patients essentially depends on the endoscopic lesion stage. In patients with stage 0 or 1, the evolution is favorable with a possible discharge without specific treatment, after 24 or 48 hours of monitoring and restriction of oral food. Stage 2a and some patients with stage 2b generally have a favorable outcome after medical treatment, leading to lesion regression and discharge after an average of 7 days [16].

Beyond stage 2b, several complications can occur, immediate or long-term, in particular stenosis, which is the most frequent complication described in the majority of studies, pneumopathy during hospitalization, or even death from metabolic disorders. , hemorrhage or perforation. [16].

1. Complications:

Complications were noted in 66.6% of patients in our series, compared to 24% in the study by Bruzzi and Chirica as well as other studies with a similar percentage.

Several factors affect the prognosis of patients: Age, pre-morbidity, intentionality, and the nature as well as the quantity ingested.

The most frequent short-term complications are in order of frequency:

- Pneumopathy: found in the majority of patients in the literature, by inhalation and extension of the lesions towards the tracheobronchial tree or by nosocomial pneumopathy [2], encountered in our study in 32.1% of cases, 88% of whom had used resuscitation with oxygen therapy by non-invasive means or mechanical ventilation.

- Gastrointestinal bleeding: Described in 6 patients in our series with hematemesis.
- Perforation: occurred in our series in 14.8% (n=4) of cases, including two gastric with peritonitis, and two esophageal with mediastinitis.
- Pancreatitis: We described in our series 2 cases of caustic pancreatitis among the early complications.
- The oeso-tracheal fistula: rare. Not found in our study.

In the long term, digestive stenosis is the most frequent complication, predominant at the esophageal or antropyloric level, encountered in 37% in the study by Bruzzi and Chirica against 50% (n=14) in our study.

Cancerization secondary to caustic ingestion is described in the literature as a rare complication of late-onset with an average delay of 40 years. This explains the need for long-term follow-up, especially in patients with necrosis, recurrent dilated stenosis, or who have benefited from a surgical replacement of the burnt area[34]

2. Mortality: [11], [34]

Mortality in our study was 25% with 85.7% of these patients having a radiological stage greater than or equal to III with trans-parietal necrosis, mainly linked to direct complications of caustic ingestion.

In the other studies, mortality is mainly linked to post-operative complications, at 13.3% in a study carried out at UCV by RKIZAT in 2013 and at 5.3% in the EL HAMMOUMI study.

We explain the high mortality rate in our study by performing the CT scan as an inclusion criterion, which was performed in patients who were unstable or most often presented with complications.

IX. CARE MANAGEMENT :

1. Conditioning and medical treatment: [4]

Medical treatment is implemented systematically in all patients, constituting an essential phase of care after proper conditioning.

Certain actions are prohibited:

- **No induced vomiting or gastric lavage precipitating** perforation or aggravation of the lesions, and favoring inhalation pneumopathies.
- **No nasogastric tube**: this increases the risk of perforation, especially in the presence of necrotic lesions with a fragile digestive wall.
- **No gastric dressing or neutralizer**, which is useless for management and only hinders the assessment of lesions on endoscopic examination.
- **Avoid laying central or peripheral venous lines** at the cervical jugular or supraclavicular level, this region constitutes an emergency surgical approach that should be preserved.
- **No EGD** if the clinic suggests a digestive perforation.

Thus, in the face of caustic ingestion, the patient must first be put in condition:

- **Seated or semi-seated position,** favoring the single passage of the ingested product and avoiding inhalation.
- **Venous route of good caliber** preferably at the level of the distal superficial venous network of the upper limbs.

- **Oxygen therapy** with a discussion of mechanical ventilation in the event of severe respiratory distress, impaired consciousness, or state of shock.
- **Washing** contaminated skin surfaces with water and cleaning the oral cavity.

Initial treatment should be implemented without delay, consisting above all of the correction of a state of shock and the management of pain, while avoiding sedatives or morphine without clear indication, which may hide surgical pain or increase the risk of inhalation in the initial phase.

The questioning must be complete and directed, to determine the nature, the concentration, and the volume of the product as well as the hour and the delay with the circumstances of the ingestion, while specifying possible associations "Alcohol, drug, drugs…", this will allow you to think about the toxicities to look for and to direct a specific treatment.

Antibiotic therapy was installed upon admission in some authors and is administered only after the appearance of signs of infection in others. In EL Hammoumi's study at the UCV, it was given in 12.5% of cases, after clinical or biological suspicion of infection [11].

Corticosteroid therapy is reserved for cases of laryngeal dyspnea or refractory septic shock, it is useless in the acute phase [35], and can be reserved in low doses associated with endoscopic dilatations, to improve the evolution of stenoses according to some studies [36].

Systematic use of corticosteroid therapy or antibiotic therapy is not indicated and can be dangerous. [21]

Parenteral nutrition after stopping feeding: allows the protection of burns by avoiding trauma and superinfections due to oral feeding, or the installation of an esophagogastric tube, in fact, this theoretically promotes the formation of long-term strictures by exacerbation of fibroblast reaction due to inflammation resulting from repetitive strain injuries. Parenteral nutrition also reduces the risk of inhalation resulting from aspiration. The exact caloric intake is difficult to determine, however, an optimal dose of 40-50Kcal/Kg/Day or more would allow healing and avoidance of hydro-electrolyte and infectious complications and would prevent the risk of perforation. [21]

Digestive rest is indicated in all endoscopic stages, with different delays depending on the authors, for a duration of 24 hours on average in patients with stage I, around 8 days in patients with Grade IIa, and up to 21 days. for patients with a grade greater than IIb.

2. Surgical management:

The clinical examination is a sufficient element to indicate an emergency surgery in case of ingestion of caustics, after detection of signs of perforation, namely abdominal contracture. Despite surgery and improved techniques, the prognosis in these cases remains grim and fatal.

Emergency surgery within 12 hours generally consists of primary exploration that can lead to trans-Hiatal esophageal resection without thoracotomy or gastrectomy in patients who have ingested large quantities of caustics, supplemented by a feeding jejunostomy, to limit the extension of the necrosis towards the mediastinum and neighboring organs, with remote revision for corrective surgery. However, trans-parietal necrosis with extended intra-abdominal can lead to therapeutic abstention or only the placement of a feeding

jejunostomy, considering that postoperative complications in these patients most often lead to death.

NB: Some authors insist on resection of the affected digestive region, as long as others recommend a more conservative approach consisting of monitoring to avoid post-operative risks, linked to heavy and dangerous surgery on a digestive tract. brittle. [15]

At this phase, in the event of extensive endoscopic Stage IIIb necrosis, two attitudes are adopted according to the authors, the first is a wait-and-see conservative approach, consisting of the preservation of the digestive tract with monitoring, while the second is that of early excision.

a. Directions:

➢ **Emergency surgery:**

- Severe clinical signs: massive ingestion of more than 150ml or hemodynamic, respiratory, or neurological instability linked to the caustic ingested:
  - Abundant bleeding.
  - Signs of peritonitis: abdominal pain and defense with "wooden tummy" contracture.
  - Signs of Mediastinitis.
- Biological disorders: Massive acidosis or coagulation disorders with "DIC" fibrinolysis, acute renal failure, or hyperleukocytosis greater than 20,000/mm3 [35]

- Radiology: Signs of digestive perforation.
- Restorative surgery: during the healing phase, after resting the digestive tract and parenteral nutrition, avoids the postoperative complications encountered during hasty surgeries. In some studies, the time to surgery represents the time required for the healing of the burns, situated at a distance of 2 months for isolated gastric damage or 3 months if it is associated with oesophageal burns. [15]

b. Techniques and means:

Mucosal necrosis often justifies complete or partial resection of the esophagus or stomach, to prevent perforations on 3rd-degree lesions, hemorrhages, and progression to strictures in the presence of 2nd or 2nd-degree burns. 3rd degree endoscopic.

- Means of excision:

- Closed chest esophagectomy: by stripping the esophagus, approaching it through a midline and cervical abdominal incision. It is a fast, simple, and safe intervention.
- Total esogastrectomy.
- Extended resection to neighboring organs: in the event of lesions extending to the digestive tract or neighboring organs (cephalic duodenal-pancreatectomy, splenectomy, cholecystectomy).

- **Feeding jejunostomy or gastrostomy:** without or associated with

- Means of repair:

- Tracheo-bronchial reconstructions: in the event of an associated tracheobronchial lesion. The 'Nelson's lobe' lung fills the mediastinum and obstructs the perforation or the suspicious tracheal area.
- Esophagoplasty: in the event of esophageal stenosis not accessible to endoscopy, consisting of reconstruction of the latter using long digestive transplants that can cross the thorax with an abdominal feeder pedicle:
  - Transverse colon: so-called isoperistaltic transverse Coloplast pedunculated on left superior colonic vessels, or by right retro-sternal ileocoloplasty.
  - Stomach: entirely respecting the continuity between the stomach and the esophagus in the peristaltic or aniso-peristaltic position, or by isoperistaltic or aniso-peristaltic gastric tubuloplasty.
- PEAN-type lower polar gastrectomy: in cases of antropyoloric stenosis.
- Pharyngoplasty: partial or total.

3. Esophageal dilatations:

In cases of caustic strictures of the esophagus, endoscopic dilation by Savary-Miller candle or balloon may be indicated, especially for short loose strictures, after the healing phase to avoid complications such as infection. or the iatrogenic perforation that can be caused by this technique.

Endoscopic dilation avoids the use of heavy surgical treatment, of its simplicity and effectiveness, however, certain predictive factors of its failure must be taken into account, namely the inflammatory nature of the mucosa and the short time before the first session. [37], [38]

The number and length of the stenosis are the major criteria for the indication of dilation, Mircea Chirica et al. In one of their studies, proceeded to dilation in the cases of less than 3 stenoses of length less than 5cm. If dilation fails after 5 dilation sessions or the stenosis is greater than 5cm or is multiple (>3), surgical reconstruction is possible. [39]

In our series, 6 patients with stenosis benefited from dilation sessions with a good evolution in 66.6% of the cases, only one case was complicated by a perforation due to the dilation which required surgical management by esophagoplasty and an another in whom dilation failed and died after a year of ingestion.

4. Psychiatric care:

The management of these patients must imperatively be supplemented by a psychiatric consultation and follow-up, especially for patients whose ingestion was intentional for autolysis.

In our study, all our patients were systematically referred to the psychiatry department after leaving the surgery department.

X. PLACE OF CT:

The contribution of CT as a predictive lesion assessment of complications is a new approach that is still debated, few studies so far have been able to address the subject. Its interest lies mainly in the diagnosis of trans-parietal necrosis, but studies have been able to demonstrate its ability to predict stenosis, morbidity, and mortality of patients. However, the results remain conflicting.

Auteur	Année	Type d'étude	Effectif	Résultats
Lurie et al.	2013	Rétrospective	23 adultes	- Tous ont bénéficié d'un scanner et une endoscopie dans les 48H. - Pas de preuve anatomo-pathologique de la nécrose. - Discordance TDM- Endoscopie : 66,6% des patients avec grade 3 de Zargar avaient un grade 1 scannographique (œdème sous-muqueux). - Concordance TDM – Endoscopie : chez 21,7% des cas. - Mortalité : CT (VPP 66% ; VPN 86% ; Ss 40% ; Sp 94%) / Endoscopie (VPP 21% ; VPN 100% ; Ss 100% ; Sp 38%). - Prédiction laparotomie : CT (VPP 66% ; VPN 75% ; Ss 28% ; Sp 93%) / Endoscopie (VPP 28% ; VPN 85% ; Ss 80% ; Sp 37%).
Mircea Chirica et al.	2000-2007	Rétrospective	125 adultes	- Prise en charge avec Endoscopie seule : grade 3b - Absence d'histologie. - Concordance TDM/histologie concernant la nécrose trans-pariétale chez 12 patients sur 17 (70.6%). - Réduction de la mortalité de 16% à 7% avec prise en charge scannographique. - Réduction de la morbidité de 66% à 55% après prise en charge scannographique. - Réduction du cout de soin et durée d'hospitalisation.
	2007-2012	Prospective	72 adultes	- Prise en charge avec Endoscopie grade 3b et scanner
Mircea Chirica et al.	2013-2014	Prospective	120 adultes	- Concordance TDM-Endoscopie / Nécrose : chez 24 patients (20%) dont 66% (n=16) confirmée par l'histologie après résection. - Concordance TDM-Endoscopie / Absence de nécrose : 77 patients (64%) avec prise en charge conservatrice. - Discordance chez 19 patients (16%) chez qui la TDM ne montrait pas de nécrose : prise en charge conservatrice.
Ryu et al.	2010	Rétrospective	49 adultes	- Tous ont bénéficié d'un scanner et une endoscopie dans les 72H. - La sensibilité et spécificité du score scannographique étaient modérément plus élevées que ceux de l'endoscopie par rapport à la prédiction de la sténose (CT : Ss 81.4% ; sp 95.6%) (Endoscopie : Ss 62.8% ; Sp 84.8%).
Matthieu Bruzzi et al.	2007-2014	Prospective	152	- Tous les patients ont bénéficié d'une endoscopie digestive et un examen scannographique. - La TDM a surpassé l'endoscopie dans la prédiction de la sténose à 120 jours de l'ingestion de l'agent caustique [CT= (VPP 76.1% ; VPN 85.4% ; Ss 86.1% ; Sp 75%)}. - A 1 an de l'ingestion, la TDM a surpassé l'endoscopie dans la prédiction de la nécessité d'une reconstruction chirurgicale.

Author	Year	Study	Patients	Results	
Lurie et al.	2013	retrospective	23	All patients: all patients received a CT scan and an endoscopy within 48 hours - no antomo-pathological evidence of necrosis -CT discrepancy: endoscopy: 66.6% (Zargar grade 3 had CT grade 1) - CT concordance: endoscopy: 21.7% -mortality: CT (PPV 66%, PNV 86%, Ss 40%, Sp 94%) / endoscopy (PPV 21%, PNV 100%, Ss 100%, Sp 38%) - CT laparotomy prediction (PPV 66%, PNV 75%, Ss 28%, Sp 93%) / endoscopy (PPV 28%, PNV 85%, Ss 80%, Sp 37%)	
Mircea chirica et all .	2000-2007	retrospective	125	Only endoscopy : grade 3b	- concordance between CT and histology concerning transparietal necrosis in 12 cases out of 17 (70.6%) -reduction of mortality from 16% to 7% -reduction of morbidity from 66% to 55% -reduction of the cost of care and duration of hospitalization
	2007-2012	prospective	72	Endoscopy grade 3b and CT	
Mircea chirica et all .	2013-2014	prospective	120	- concordance :CT – endoscopy : necrosis in 24 cases (20 %) 66 % of them histologically confirmed - Concordance CT- endoscopy : no necrosis in 77 patients (64%) - Discordance in 19 cases (16%)	
Ryu et al	2010	retrospective	49	All patients: all patients received a CT scan and an endoscopy within 72 hours - the sensitivity and specificity of the CT score were moderately higher than those of endoscopy with respect to the prediction of stenosis 120 days after ingestion of the caustic agent (CT = PPV 76.1%, NPV 85, 4% , Ss 86%, Sp 75%) - 1 year after ingestion, CT outperformed endoscopy in predicting the need for surgical reconstruction	

Table 29: Interest of the scanner in the management of cases of ingestion of caustic agents.

1. Diagnosis of necrosis:

In the study conducted by Y. LURIE, the CT assessment tends to underestimate the lesions 66.7% of the patients included, with a Zargar grade 3 had had a Grade I CT scan (submucosal edema), while only 16.6% had grade 3 (trans-parietal necrosis). CT then had a good PPV against a bad NPV compared to endoscopy, with better sensitivity concerning the detection of lesions and less specificity. Y. LURIE concluded that CT scans are poorly sensitive and specific compared to endoscopy, in terms of predicting the prognosis of lesions in the acute phase, the latter requires the comparison of the results between the two examinations, radiological and endoscopic, to guide surgical management, giving an inevitable place to endoscopy on admission. [25] But this study is limited by the small number of patients included (23 cases), with the retrospective nature which limits certain parameters, including the absence of histological data.

For its part, MIRCEA CHIRICA is carrying out several exhaustive studies with its team, one of which aims to study the place of CT in the evaluation of esophageal necrosis in two phases, retrospective from 2000 to 2007 and prospective from 2007 until 2012, out of a sample of 197 patients, all classified as Zargar grade 3b, of whom 125 underwent endoscopy only (2000-2007) and 72 underwent both endoscopic and CT examinations (2007-2012).

In 2007 the results of the scan performed systematically were the basis on which the discussion of the surgical indication is based, thus transmural necrosis on CT led to an esophagectomy.

The team compared the histological data of the post-operative specimens with CT results in patients classified as Grade 3b endoscopy, which clarified the place that CT may have in the detection of transmural necrosis.

Among the patients who underwent CT, 35% (N=25) underwent surgery, of which 17 indicated based on the CT scan result, histology confirmed transmural necrosis in 12 patients out of 17, i.e. 70%, the 8 other patients were operated on as indicated by the surgeons correlated with the endoscopic result, and in whom the histology was not in favor of any transmural necrosis. Thus the indication for surgery was markedly reduced to a small number, compared to the group of patients managed by endoscopy alone (before 2007) where 124 patients underwent surgery versus a single patient treated conservatively who survived with treated stenosis by dilation.

Consequently, given the impotence of fibroscopy to determine trans-parietal involvement, an esophagectomy could be performed in excess, the scanner then made it possible to spare excessive esophagectomy. [40]

The same type of prospective study was conducted by V. Gault et al. By comparing the preoperative CT data of 14 patients with endoscopic grade IIIb, with histological results. The latter concluded that CT has good specificity and sensitivity in predicting trans-parietal necrosis reaching 80.8% and 81.2% respectively, with a PPV of 72.2% and VPN of 87.5. % [26], this study is limited by the small number of its population.

In our work, out of a series of 28 patients, 23 benefited from both an endoscopy and a CT scan exploring the esophagus, while in 22 patients only the stomach could be explored by these two examinations simultaneously. We compared the results of CT and endoscopy looking for their concordance concerning the presence of necrosis, considering that trans-parietal necrosis is likely to occur in patients with endoscopic stage 3b. Thus at the level of the esophagus, in 19 patients out of 23, the CT and endoscopy agree on the absence of trans-parietal necrosis and in one patient on its presence, while the results of only 6 patients were discordant.

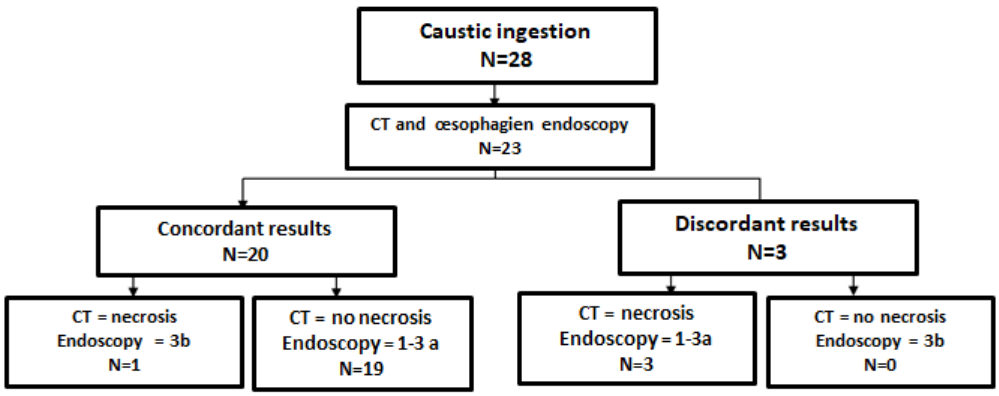


Figure 40: Diagram summarizing the concordance between endoscopic and computed tomography results in the esophagus concerning transmural necrosis.

At the level of the stomach, in 18 cases the results were concordant, including 16 on the absence of trans-parietal necrosis and 2 on its presence. In 4 patients the results were discordant.

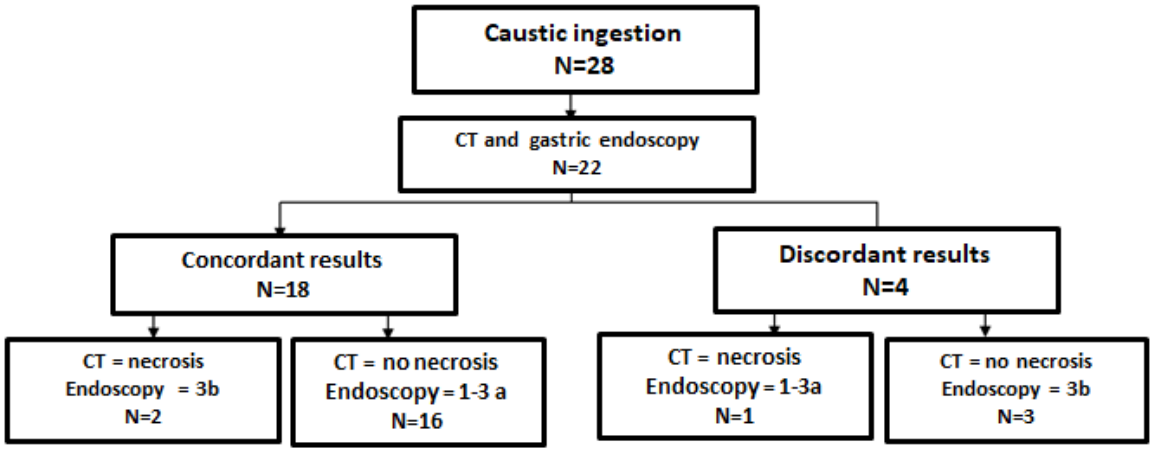


Figure 41: Diagram summarizing the concordance between endoscopic and CT scan results in the stomach.

Concordance was then found in 87% at the level of the esophagus and in 82% at the level of the stomach. These results converge with those obtained by Chirica et al. In another study where concordance was found in 101 out of 120 patients following the same reasoning. [23]

The limited number of patients included and the digestive resections carried out, as well as the absence of histological data, were limitations in our study, which did not allow us to confirm trans-parietal necrosis.

2. Stenosis prediction:

Stenosis is the leading complication in patients who have survived the acute phase of caustic ingestion, whose management is currently complicated by the resources and the time it requires, for a functional prognosis that can be disappointing. [41]

In another study, Matthieu Bruzzi and M. Chirica followed the evolution of 152 patients who had benefited from conservative management, over a period stretching from 2007 to 2014, to establish a correlation between CT classification and the prediction of esophageal stenoses compared to endoscopy, allowing them to be monitored appropriately for patients at risk, in a possible preventive context.

The risk of stricture formation was 0, 28, 50, and 76% for Zargar endoscopic grades 1, 2a, 2b, 3a, and 3b, respectively, compared to 0, 17, 83% for CT grades I, IIa and IIb according to the classification they have established for this specific purpose (see p.108-109). Their results demonstrate that the CT score outperforms endoscopy in predicting stricture formation after 120 days of ingestion, as long as the combination of the two examinations does not show any deviation from use. scanner alone at this stage. Moreover, 1 year after ingestion, a CT scan alone outperformed endoscopy in predicting the need for esophageal reconstruction. [2]

In another South Korean study published in 2009, HH RYU compared the usefulness of CT versus endoscopy in predicting caustic stenosis in 49 patients, CT was found to have better sensitivity and specificity. [32]

About our study, the results obtained are similar to those found in the literature the CT scores from IIa to III were sensitive at 84.6% with a positive predictive value of 73.3% and a negative predictive value of 75% compared to the occurrence of a stenosis, but with poor specificity, however without exceeding endoscopy which has a sensitivity of 92.9%, with a negative predictive value of 85.7% and positive of 76.5 %. Our results remain limited by the small number of patients included in our study compared to other studies with missing data.

The scanner then seems to be a promising means for the prediction of the stenosis, which can make it possible to take preventive measures and to spread out the follow-up of the patient, however, a confrontation with endoscopic data is necessary.

3. 3-Surgery, mortality, and morbidity:

The new management established by Mircea Chirica et al, made it possible to avoid esophagectomy in 2/3 of patients classified as Zargar grade 3b, having performed a CT scan coupled with endoscopy. No death was described in these patients benefiting from a conservative attitude. Thus, mortality fell from 16% to 7% while morbidity from 66% to 53% by integrating the scanner into the therapeutic decision in their study, this led the team of gastroenterologists from the same center to follow an approach based essentially on the scanner.

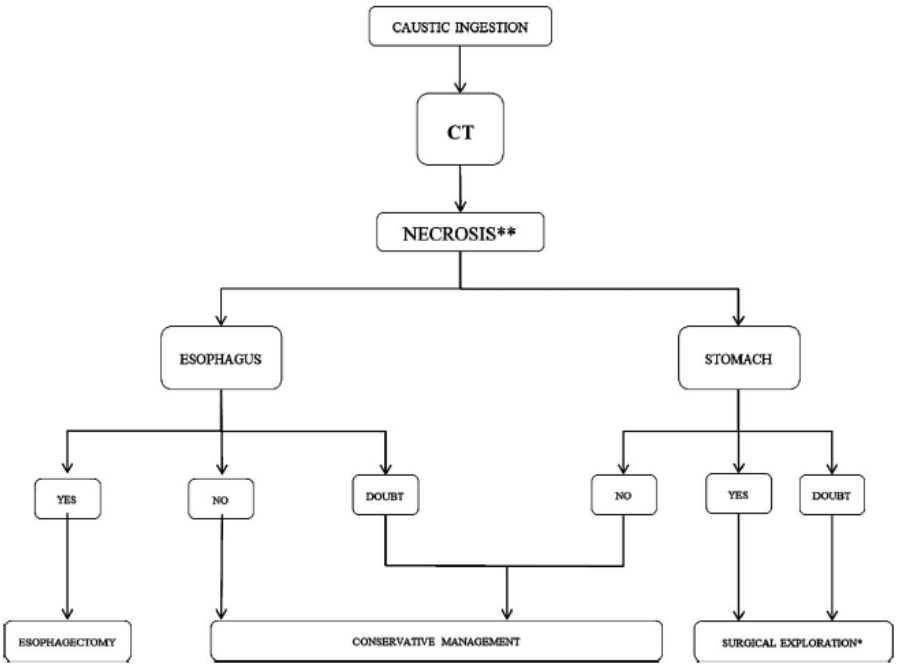


Figure 42: Algorithme de prise en charge des ingestions de caustiques proposé par Chirica et al.

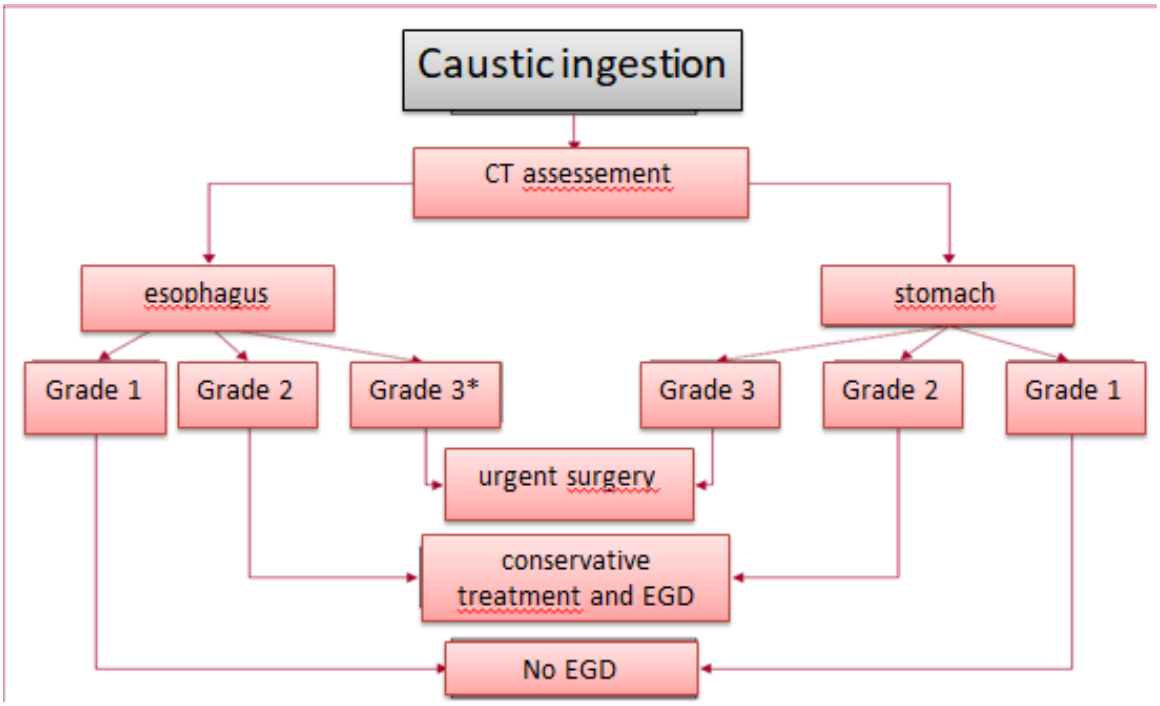


Figure 43: Management algorithm proposed by the gastroenterology team based on that of Chirica et al. of the Saint-Louis Hospital (Paris).

***Grade 3:** Trans-parietal necrosis. EOGD = Esophagogastroduodenal endoscopy.

In ours, the action to be taken was essentially conservative, with surgical exploration in patients admitted in a table of perforation (N=4) diagnosed by CT scan when endoscopy was contraindicated.

A jejunostomy was necessary only in patients who developed complications during their hospitalization (N=7) or required reconstructive surgery after failure or complication of endoscopic dilation (N=2).

The results of the two examinations were closely related to the prediction of morbidity.

A radiological stage at IIb or higher was predictive of morbidity at 88.2% with a sensitivity of 79% and specificity of 75%, while the presence of an anomaly on the scanner is predictive at 81.8% of the occurrence of a short or long-term complication, with a VPN of 80%, a sensitivity of 94.7% and specificity of 50%. On the endoscopy side, a Zargar stage ≥ 2b, is like CT, predictive of a complication in 88.2%, with a better sensitivity of 93.8% (VPN and specificity of endoscopy respectively at 85,7% and 75%).

As for mortality, the scanner surpassed the endoscopy in its prediction, mainly related to acute complications, in particular pneumopathy (N=9), digestive perforation (N=4), and hemorrhages (N=6). Sensitivity, specificity, PPV and NPV of CT stage IV in predicting mortality were 50%, 100%, 100% and 82.6%, respectively, compared to 37.5%, 85%, 50% and 77.3% for Zargar endoscopic stage IIIb.

XI. . LIMITATIONS OF THE STUDY:

Our study was limited by several constraints, in particular, the number of patients included, which contributed little statistically, due to a lack of radiological or endoscopic data with a large number of incomplete files.

The lack of histological data in operated patients and concrete proof of trans-parietal necrosis has constrained our work in terms of necrosis prediction to only a descriptive approach.

The retrospective nature of the study did not allow us to control the radiological parameters, by choosing a unified optimal protocol for all our patients, which reduced the quality of the interpretation, especially regarding the exploration of esophageal enhancement due to non-optimal injection time.

XII. RECOMMENDATIONS:

With approximately close results between endoscopy and CT in terms of prediction of trans parietal necrosis, complications, and mortality, we propose the integration of CT as a simple and rapid means of the first intention, in the algorithm of initial management. in the acute phase of patients who have been ingested caustics, while reducing the risk associated with endoscopy and the anesthesia required to perform it [Figure 42].

- A patient with a CT scan without abnormalities classified as Stage I may benefit from monitoring and symptomatic treatment.
- A patient with CT stage IIa will require remote endoscopy for the first few hours with conservative medical management.
- In Patients with a stage IIb, III scanner or doubtful result, an endoscopic confrontation will be necessary after which they may benefit from surgery if the result is in favor of trans-parietal necrosis, or monitoring and treatment conservative in case the examinations are discordant.
- A radiological Stage IV patient should benefit from urgent exploratory surgery with resuscitation measures.

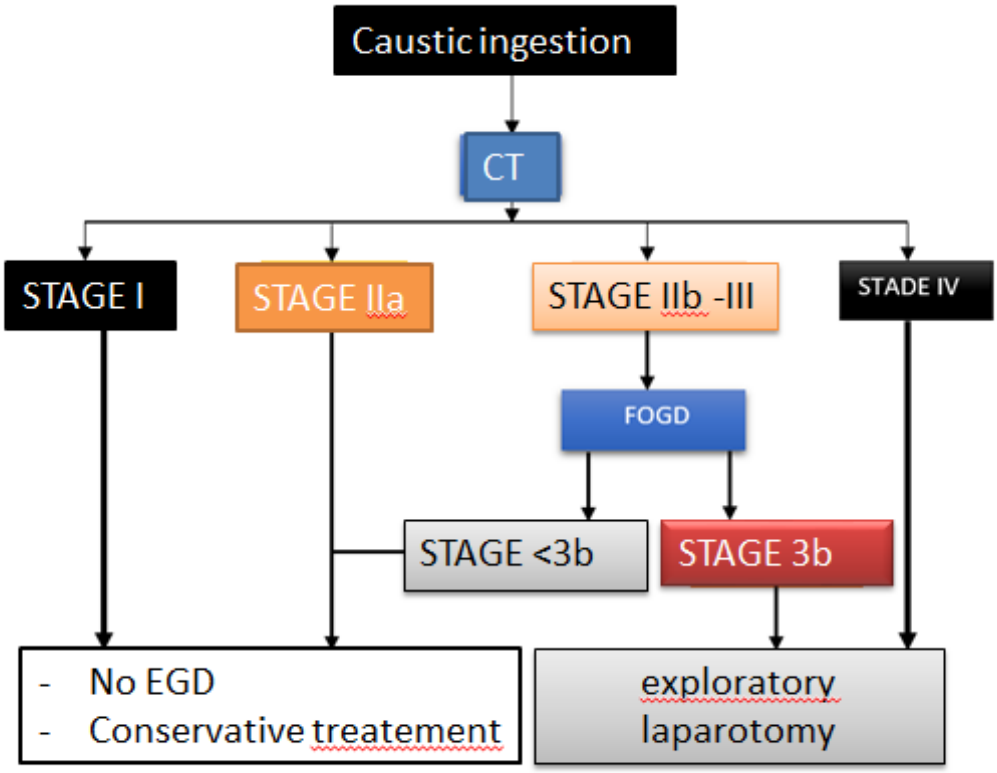


Figure 44: Recommended support algorithm.

Stage I: normal; Stage IIa: submucosal edema; Stage IIb: submucosal edema without mucosal enhancement with fat infiltration; Stage III: Trans-mural necrosis; Stage IV: Complication. EGDF: Esophagogastroduodenal fibroscopy.

Conclusion

In conclusion, CT seems to be a promising means to the diagnosis of necrosis, the establishment of vital prognosis during the acute phase, and the prediction of stenosis in association with endoscopy, allowing to guide the management and patient follow-up. However, our essentially descriptive study had several limitations, including the retrospective nature with a small number of patients and the lack of histological data.

We, therefore, recommend the integration of the scanner into the patient management algorithm and encourage other prospective and multicenter studies to confirm the results found in the literature to support the possibility of using the scanner alone as a means of exploration of patients who have been victims of caustic ingestion to minimize endoscopic examinations which can prove to be dangerous and lead to excessive surgeries.

Abstract

ABSTRACT:

Title: Computed tomography of caustic ingestion: the experience of department of radiology emergency (about 28 cases).

Author: Essaber Hatim

Keywords: computed tomography – endoscopy – corrosive ingestion.

Caustic ingestion is a common incidental condition, involving the vital and the functional prognosis by the existence of acute complications or stenosis creating a therapeutic challenge.

Until today, endoscopy is still the principal tool for diagnosis and management. Original studies are discussing the CT as a favorable alternative to endoscopy and proved the danger of this invasive assessment, and also the excessive exeresis operations depended on its outcomes.

Our retrospective study included 28 patients who were victims of corrosive product ingestion, having carried out a CT scan of which 24 underwent an endoscopy investigation, over 3 years.

According to the CT score and the ZARGAR classification, the two exams were performed at 87% in the esophagus and 82% in the stomach on the presence or absence of trans-parietal necrosis. Digestive Harm saw Radiologically on 81.8% predictive of the occurrence of a complication, with an NPV of 80%, a sensitivity of 94.7%, and a specificity of 50%. A scenographic score between IIa and III was anticipating stenosis in 73.3% of cases, with an NPV of 75% and sensitivity of 84.6%, at the same time endoscopy had a rather better NPV, PPV, and sensitivity. A scannographic IV score was 100% predictive of mortality with an NPV of 82.6%. The CT scan permitted the diagnosis of complications and postoperative follow-up.

The place of CT is indisputable and should be integrated into the management, however, a confrontation with endoscopic data is stilled unavoidable.

RÉSUMÉ :

Titre : Tomodensitométrie dans l'ingestion caustique : l'expérience du service de radiologie des urgences (à propos de 28 cas).

Auteur : Essaber Hatim

Mots clés : Agents Caustiques – Scanner – Endoscopie.

L'ingestion de produits caustiques est une condition momentanée fréquente dans le monde, mettant en jeule pronostic vital et fonctionnel par la survenue de sténoses présentan un challenge thérapeutique.

Jusqu'au aux nos jours, l'endoscopie garde une gande place autant que Gold-standard dans diagnostic et la prise en charge. De recentes études discutant le role du scanner comme moyen prometteur et concurrent de l'endoscopie, dévoilant le risque que celui-ci porte, avec le grand nombre de chirurgie d'exérèse excessive justifiée sur ses résultats.

Notre étude rétrospective et analytique, est accomplie au sein du Centre Hospitalier Ibn-Sina, portée sur 28 patients victimes d'une ingestion de caustique , sur une période de 3 ans.

Selon le score scannographique établi et la classification de ZARGAR, les deux examens se sont attribués à 87% des cas au niveau de l'œsophage et 82% au niveau de l'estomac sur la présence ou non de nécrose trans-pariétale. L'atteinte digestive observée radiologiquement était pourvoyeuse à 81,8% de la survenue d'une complication, avec une VPN de 80% et une sensibilité de 94,7%. le score scannographique entre IIa et III est prédictif d'une sténose dans 73,3% des cas, avec une VPN de 75% et une sensibilité de 84,6%, alors que l'endoscopie a été un peu meilleure en VPN, VPP et sensibilité. le score IV scannographique était prédicteur de mortalité à 100% avec une VPN de 82,6%. La scanner a permis de diagnostiquer les complications et le contrôle post-operatoire.

Le role du scanner est assuré ,et il doit integrer systématiquement dans la prise en charge du malade , toutefois une confrontation aux données endoscopiques reste essentiel pour une meilleur décision thérapeutique.

الملخص

العنوان: التصوير المقطعي في حالات ابتلاع المواد الكاوية : تجربة قسم الأشعة الطارئة (حوالي 28 حالة).

المؤلف: الصابر حاتم

الكلمات المفتاحية: المواد الكاوية – التصوير المقطعي بالأشعة – المنظار

ابتلاع المواد الكاوية حالة مرضية ظرفية مترددة، تهدد حياة المريض في المرحلة الحادة و تمثل تحدي للعلاج على المدى البعيد بحدوث تضيقات في الجهاز الهضمي العلوي.

حتى يومنا هذا ، لا يزال المنظار الداخلي هو المعيار الأساسي للتشخيص و توجيه العلاج بينما دراسات جديدة تستخدم الماسح بالاشعة كوسيلة واعدة للفحص في هذه الحالات، منافسة للمنظار الذي قد يشكل خطرا بمضاعفة الإصابات أو التسبب في جراحات استئصال مفرطة.

تم إجراء دراستنا في المركز الإستشفائي الجامعي ابن سينا حول 28 مريضًا ضحية ابتلاع مواد كاوية ، الذين أجروا فحصًا بالأشعة المقطعية ، 24 منهم خضعوا للتنظير الداخلي ، على مدى فترة 3 سنوات.

بناء على التقييم بالأشعة من جهة و بالمنظار من جهة أخرى ، اجتمع الفحصان على نفس النتيجة عند 87 % من الحالات من حيث وجود تنخر في المريء وعند 82 % من الحالات على مستوى المعدة. استطاع الماسح بالأشعة كذلك التنبؤ بنسبة 81,8 % بحدوث مضاعفات و كانت قيمة تنبئه الإيجابية بحدوث تضيق ترتفع إلى 73,3% و السلبية إلى 75 % بحساسية 94,7 % ، بينما كان للمنظار قيمة متفوقة بتنبئه الإجابي و السلبي بالتضيق ، كما استطاع الفحص بالأشعة التنبؤ بالوفاة بنسبة 100 %. أتاح الفحص بالأشعة بواسطة التصوير المقطعي تشخيص المضاعفات وتتبع الحالات بعد إجراء عملية جراحية.

كخلاصة لدراستنا ، لدى الفحص بالأشعة مكانة لا يمكن إنكارها ، يوجب ادماجه ضمن العلاج، الا انه من الواجب

المقارنة بنتائج المنظار للحصول على بيانات شاملة تمكن من استنتاج أفضل القرارات للمريض.

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