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Adhesive small bowel obstruction in children

THESIS

PRESENTED AND DEFENDED PUBLICLY THE 01/10/2019

BY

Mr.YOUNESS EL-KHADIR

Born on the 27 September 1993 in Kasba Tadla

TO OBTAIN THE DEGREE OF DOCTOR OF MEDECINE

KEYWORDS :

Adhesions – Adhesive small bowel obstruction – ASBO – Children – Pediatric – conservative management – Water-soluble contrast – Gastrographin

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بسم الله الرحمن الرحيم


وقل رب زدني علما

Hippocratic Oath

I swear to fulfill, to the best of my ability and judgment, this covenant:

I will respect the hard-won scientific gains of those physiciansin whose steps I walk, and gladly share such knowledge as is mine with those who are to follow.

I will apply, for the benefit of the sick, all measures [that] are required, avoiding those twin traps of overtreatment and therapeutic nihilism.

I will remember that there is art to medicine as well as science, and that warmth, sympathy, and understanding may outweigh the surgeon's knife or the chemist's drug.

I will not be ashamed to say "I know not," nor will I fail to call in my colleagues when the skills of another are needed for a patient's recovery.

I will respect the privacy of my patients, for their problems are not disclosed to me that the world may know. Most especially must I tread with care in matters of life and death. If it is given me to save a life, all thanks. But it may also be within my power to take a life; this awesome responsibility must be faced with great humbleness and awareness of my own frailty. Above all, I must not play at God.

I will remember that I do not treat a fever chart, a cancerous growth, but a sick human being, whose illness may affect the person's family and economic stability. My responsibility includesthese related problems, if I am to care adequately for the sick.

I will prevent disease whenever I can, for prevention is preferable to cure.

I will remember that I remain a member of society, with special obligations to all my fellow human beings, those sound of mind and body as well as the infirm.

If I do not violate this oath, may I enjoy life and art, respected while I live and remembered with affection thereafter. May Ialways act so as to preserve the finest traditions of my calling and may I long experience the joy of healing those who seek my help.

Declaration of Geneva, 1948

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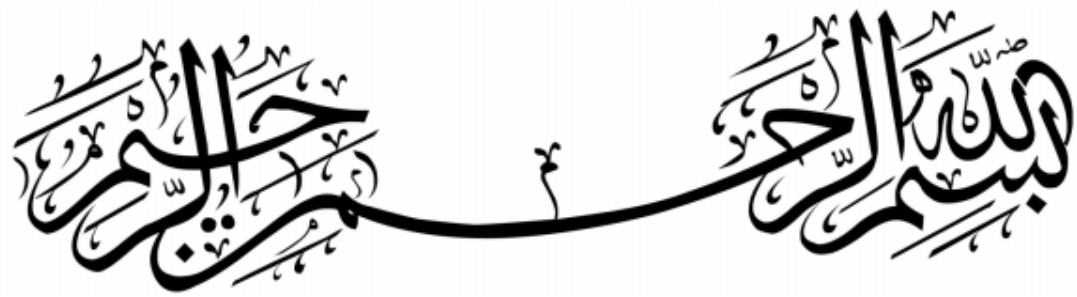

"رَبِّ أَوْزِعْنِي أَنْ أَشْكُرَ نِعْمَتَكَ الَّتِي

أَنْعَمْتَ عَلَيَّ وَعَلَى وَالِدَيَّ وَأَنْ أَعْمَلَ

صَالِحاً تَرْضَاهُ وَأَدْخِلْنِي بِرَحْمَتِكَ فِي

عِبَادِكَ الصَّالِحِينَ."

صدق الله العظيم

سورة النمل الآية 19

First and foremost praise is to ALLAH, the Almighty, the Greatest of all, on whom ultimately we depend for sustenance and guidance.

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My dear mom, I owe you everything in my life, and I hope that I have been up to your expectations and I have made you so proud as you have always been my source of pride and my example. May Almighty ALLAH protect you and grant you health, long life, and happiness.

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Please find this work, the testimony of my sincere gratitude ,and my deep appreciation and respect.

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List of abbreviations :

ASBO : Adhesive small bowel obstruction

AXR : Abdominal X-ray

NOM: Non operative management

AFL : air-fluid levels

DLB : Dilated loops of bowel

CT scan : Computed tomography scan

WSCA : Water-soluble constrast agent

CRP : C-Reactive Protein

BUN : Blood urea nitrogen

LDH :Lactate dehydrogenase

CK : Creatine kinase

OIB : Occlusion intestinale par brides

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INTRODUCTION

Adhesive small bowel obstruction (ASBO) is a mechanical disruption in the patency of the gastrointestinal tract, caused by peritoneal adhesions and resulting in a combination of emesis, obstipation, and abdominal pain.

Peritoneal adhesions can be defined as abnormal fibrous bands between organs or tissues or both in the abdominal cavity that are usually separated. They may be acquired or congenital. However, most are acquired as a result of abdominopelvic surgery, Less commonly, adhesions may form as the result of inflammatory conditions, intraperitoneal infection, radiation, or abdominal trauma [1]

Adhesive small bowel obstruction (ASBO) is very common in adults and considered the second cause of intestinal obstruction after obstructed abdominal wall hernia [2]. Moreover,it was reported that up to 25% of adult patients who have undergone abdominal surgery, subsequently developed ASBO [3]. Unfortunately, this condition does not spare children; the available data reported an incidence of ASBO in children that range between 1% and 9% [4-7].

ASBO should be suspected in any child with surgical history, who is presenting persistent vomiting, distension, and abdominal pain. Moreover, further investigations must be performed to confirm the diagnosisbecause delayed diagnosis and treatment can lead to devastating consequences. [8]

In adult patients with ASBO, conservative management found to be effective in a large proportion of cases [10-12], as well as operative management which can be effective and even essential in some cases, though it carries a risk of associated morbidity and mortality[9] . in children , there is a lack of consensus,or available guidelines, on the management of ASBO ; furthermore a small number of existing studies in the broader literature that have reviewed those therapeutic approaches in this group age .

The present study was designed to review the epidemiological, the clinical, and the paraclinical features, and mainly, tooutline and compare the different aspects of ASBO management in children.

PATIENTS & METHODS

I. TYPE OF STUDY :

This is a retrospective study, that involved 61 cases of adhesive small bowel obstruction (ASBO) in 57 children admitted in the pediatric surgery department of the Mohamed VI Teaching Hospital of Marrakech, within a period of 7 years, between January 2012 and December 2018

II. PURPOSE OF THE STUDY

This study aimed to review the epidemiological, the clinical, and the paraclinical features in children with ASBO .and also to outline and compare the existing therapeutic approaches for managing ASBO in pediatric patients .(conservative management Vs. operative management), and review their outcomes

III. PATIENTS

1. INCLUSION CRITERIA

Our study involved children under 15 years old, who were admitted in the pediatric surgery department of the Mohamed VI Teaching Hospital of Marrakech, and they were diagnosed with adhesive small bowel obstruction, basing on the past medical history, the clinical presentation and the abdominal X-ray

2. EXCLUSION CRITERIA

Patients diagnosed with other causes of small bowel obstruction were excluded.

IV. METHODS

to make this work, we followed those steps :

- **Step1 :**

Using the registry of our pediatric surgery department, we searched for medical records of children with ASBO that fit our criteria

- **Step2 :**

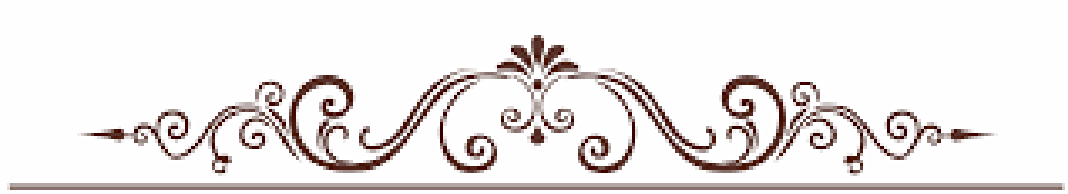
We made a medical summary sheet (see appendix I) which contains different parts for our studied parameters including ; Identity, past medical history, history of the present complain, clinical and preclinical features, management, outcome, and evolution of the patient.

- **Step 3 :**

we collected the essentialdata from medical records then we gathered them in the summery medical sheets.

- **step 4 :**

the gathered data were entered into an Excel database then organized and analyzed



RESULTS

I. EPIDEMIOLOGICAL DATA

1. Incidence :

During a 7 years period, between January 2012 and December 2018. We have identified 61 episodes of ASBO in 57 patientsadmitted in our pediatric surgery department.

Table I: Distribution of the number of Episodes of ASBO by years

Year	Number of episodes of ASBO
2012	7
2013	4
2014	11
2015	4
2016	13
2017	12
2018	10
Total	61

2. Gender

Out of a total of 57 patients included in our study, 46 (80.7%) were males, and 11 (19.3%) were females. the sex ratio was 4.18

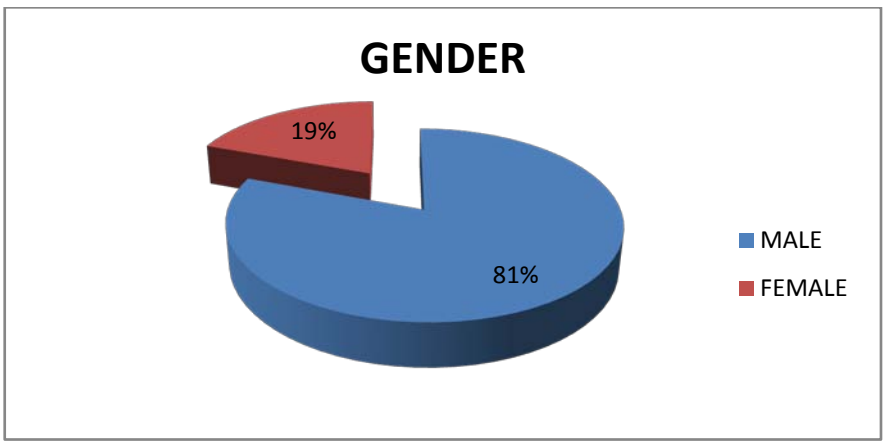


Figure 01: Gender distribution of the studied group

3. Age :

The mean age of our patients was 7 years and 5 months . and the age extremes ranged between 8 days and 15 years.

Table II: distribution ofpatients by age groups

Age group	Number (N)	%
Newborn (0 - 1 month)	1	1.75%
infant+ toddler (> 1 month - 3 years)	9	15.79%
preschooler child (> 3 - 6 years)	12	21.05%
school age child (> 6 - 12 years)	23	47.37%
Adolescent (> 12 years)	6	14.04%

II. PAST MEDICAL HISTORY

1. Medical history

Only one patient of our studied group had a medical condition, he was treated for abdominal tuberculosis, and he had a peritoneal biopsy.

2. Surgical history

3 out of our 57 patients had neither medical nor surgical history. The remaining 54 patients were operated on for different surgical conditions.

36 of the 54 patients were operated on for appendicitis (63.16%). their surgical records have shown, that 27 patients havehad perforated appendicitis (47.37%) and 9 patients with no perforation (15.79%). Four patients were operated on for Intussusception (7.02%). 3 had perforated bowel after abdominal trauma (7.02%). 3 patients had midgut volvulus (5.26%) . two patients had small bowel atresia (3.51%) . two others had omphalocele (3.51%). Diaphragmatic hernia (1.75%), sigmoid volvulus (1.75%) and peritoneal biopsy (1.75%) were found one time for each one. (the results are shown in the figure 03 bellow).

In our studied group, all patients who had surgical history have been operated on by laparotomy.

53 of 54 surgical procedures were performed in infracolic compartment while only one was performed in supracolic compartment for diaphragmatic hernia cure

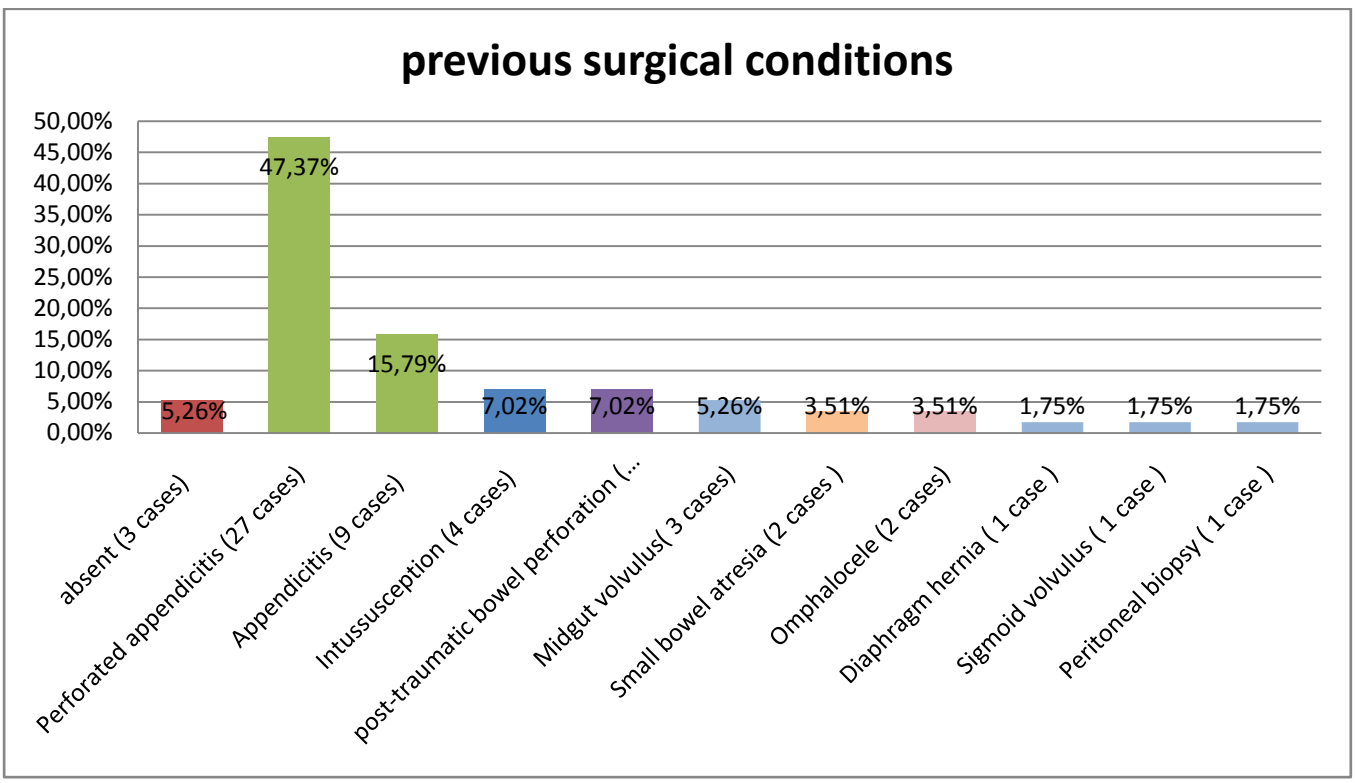


Figure02: distribution of the previous surgical conditions leading to ASBO

III. History of presenting Illness

1. Symptoms :

Table III: distribution of ASBO symptoms in our patients

Symptoms	Number of ASBO episodes	Percentage
➢ **Abdominal pain**	61	100.00%
• diffused pain	56	92 %
• Umbilical	5	7 %
➢ **Vomiting**	61	100.00%
• Bilious vomiting	37	61 %
• Nonbilious vomiting	24	39 %
➢ **Obstipation**	59	97 %

2. the onset of symptoms :

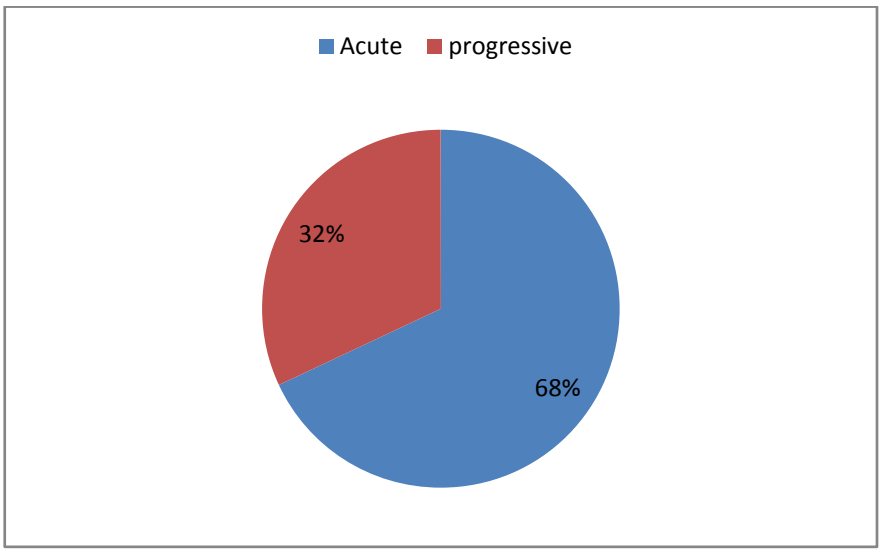


Figure 03: Distribution of the onset of symptoms

3. Patient delay:

it ranged from 6 hours to 7 days, and the mean delay was 54 hours.

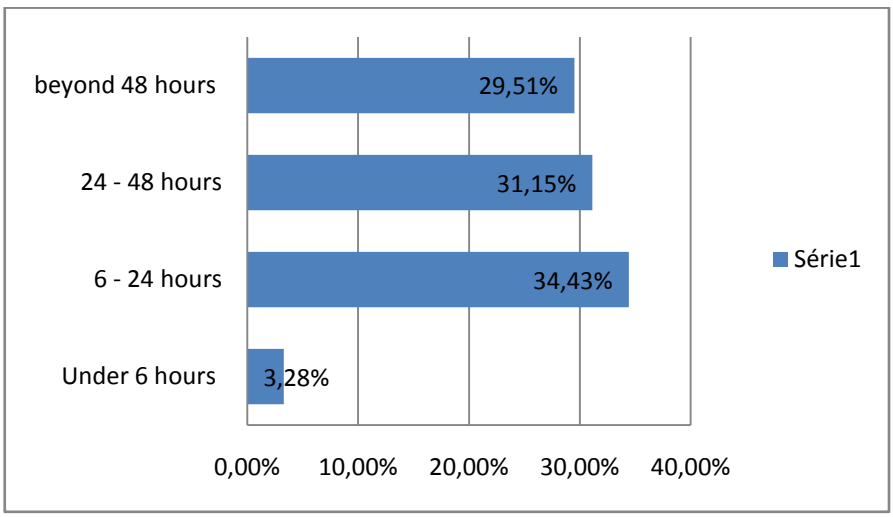


Figure04: Distribution of the time interval between the symptom's beginning and doctor visit

4. the Time period between the previous surgery and development of ASBO symptoms

The mean time period for the development of ASBO symptoms was 1 year. the extremes ranged between 7 days and 10 years

Table IV: Distribution of the time period between the surgery and the development of symptoms

Time period	Number of patients	percentage
[0 – 6 weeks]	13	24.07%
]6 weeks – 3 months]	16	29.63%
]3 months – 12 months]	16	29.63%
beyond 1 year	9	16.67%

IV. PHYSICAL EXAMINATION

1. General examination

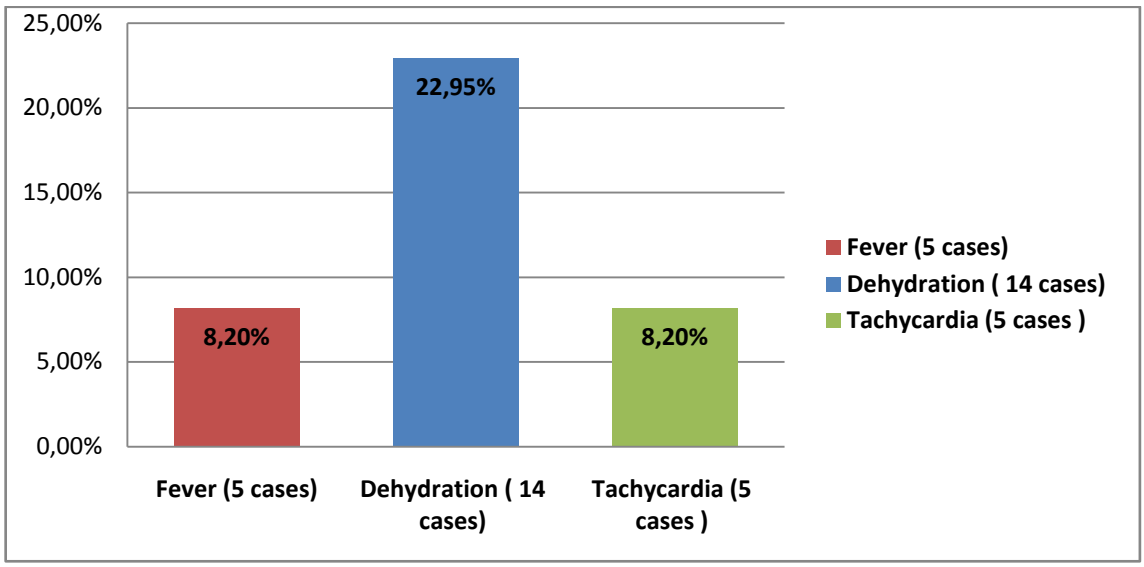


Figure 05: Distribution of physical signs found in the general examination

2. Abdominal examination :

Table V: Distribution of the abdominal examination findings

Physical signs	Number of ASBO episodes	Percentage
➢ **Abdominal distension**	35	57 %
➢ **Abdominal tenderness** • Diffused • Umbilical • Right Iliac region	61 - 49 - 9 - 3	100% - 80 % - 15 % - 5 %
➢ **Abdominal guarding**	3	5%

V. PARACLINICAL INVESTIGATIONS

1. Radiological investigations

1.1. Abdominal X-ray (AXR)

The AXR has been performed in all patients of our studied group, and it showed distended loops of bowels and air-fluid levels (AFL) mostly central.

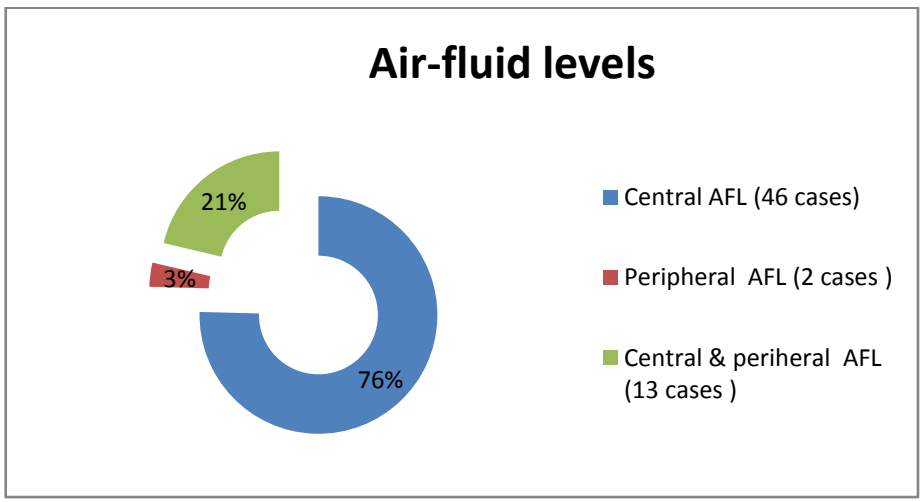


Figure 06: Distribution of the Air-fluid levels types found in our studied group

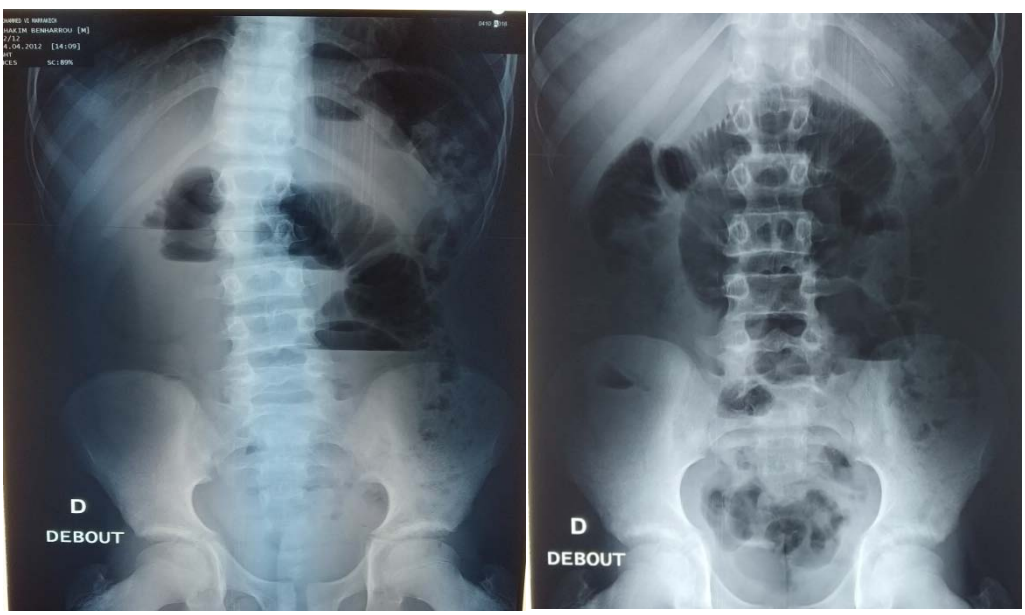


Figure 07: AXR of a 13 years old child showing central Air-fluid levels and dilated loops of bowel

Figure 08: AXR of a 7 years old showing peripheral Air-fluid levels and DLB

Figure 09: AXR of 12 years old child showing both central and peripheral Air-fluid levels

1.2.Abdominal ultrasonography

The abdominal ultrasonography was performed in 9 patients. It showed dilated loops of bowel in all of them,and peritoneal effusion in 6 patients.

2. Biological tests

9 patients have Hyperleucocytosis, one patient had severe anemia that needed a blood transfusion, and 14 patients have electrolytes imbalances including hyponatremia, hypochloremia and hypokalemia.

VI. MANAGEMENT

out of 61 ASBO cases, 11 cases (18%) underwent immediate surgery without any previous conservative therapy trial . 50 cases (82 %) were treated conservatively, and out of those patients, 16 cases (26%) underwent delayed surgical exploration after unsuccessful conservative management.

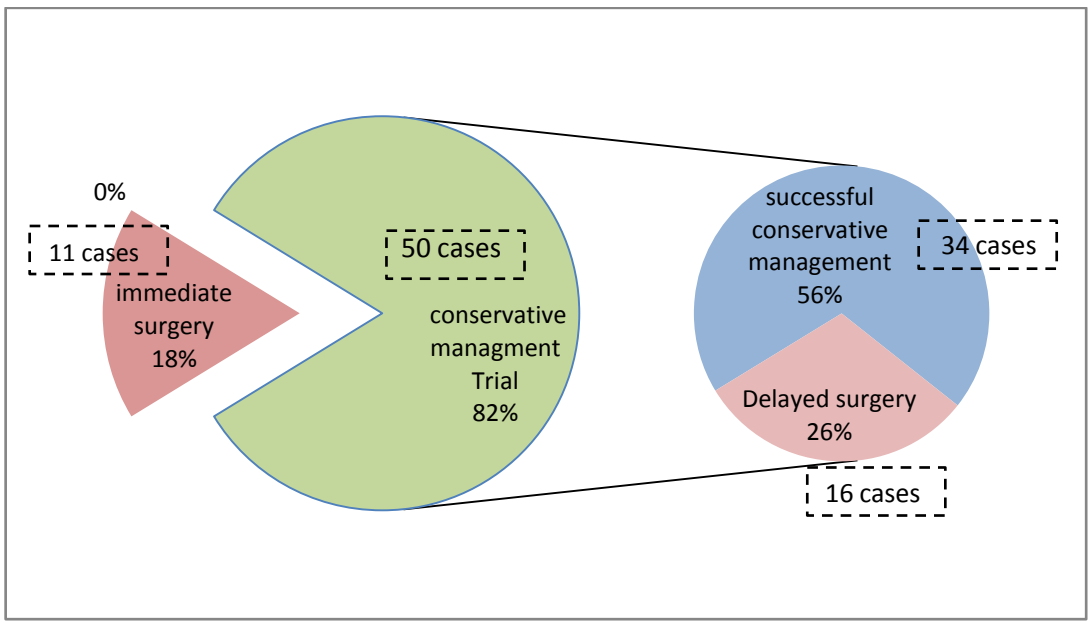


Figure 10: distribution of the types of management in our studied group

1. Conservative management

In all the 50 episodes where the patients have been managed conservatively, the patients were treated according to the standard decompressive therapy which included nasogastric decompression, exclusion of oral intake, intravenous fluid, correction of electrolytes imbalance, analgesics, monitoring and frequent reassessment. 33 patients among them received water-soluble contrast agent (Gastrographin) just after their admission along with the decompressive therapy, without waiting for a 48 hours delay.

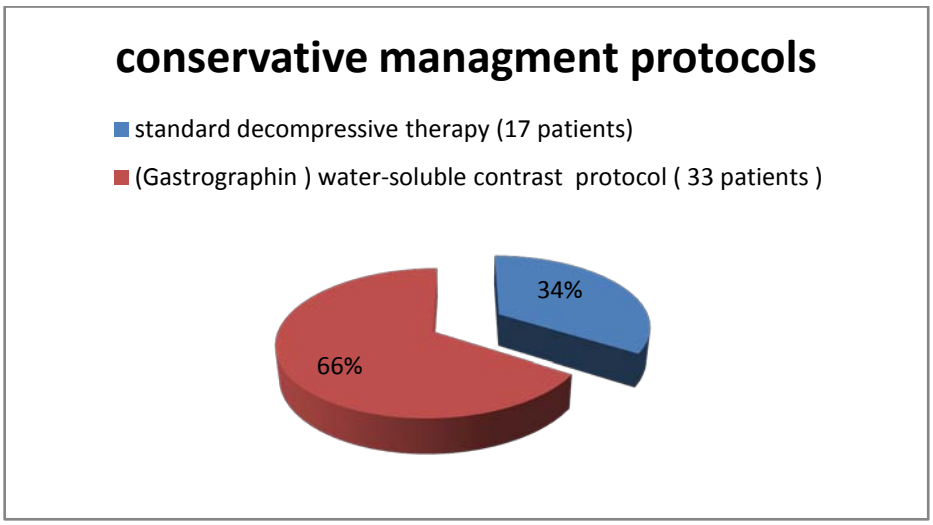


Figure 11: distribution of conservative management protocols performed in our patients

1.1. Duration of the conservative management trial

Table VI: minimum, maximum, and the mean duration of the conservative management trial

Treatment	Duration		
	min	max	mean
conservative management Trial	1 day	8 days	2.7 days
• **Standard decompressive therapy**	1 day	7 days	2.1 days
• **Gastrographin protocol**	1 day	8 days	3 days

1.2. The outcome of the conservative management

Table VII: Distribution of conservative management outcomes

Treatment	Number of episodes		success rate
	Successful outcome	**Unsuccessful outcome**	
conservative management trial	34/50	16/50	68 %
• **Standard decompressive therapy**	13 /17	4/17	76 %
• **Gastrographin prtocol**	21 / 33	12 /33	64 %

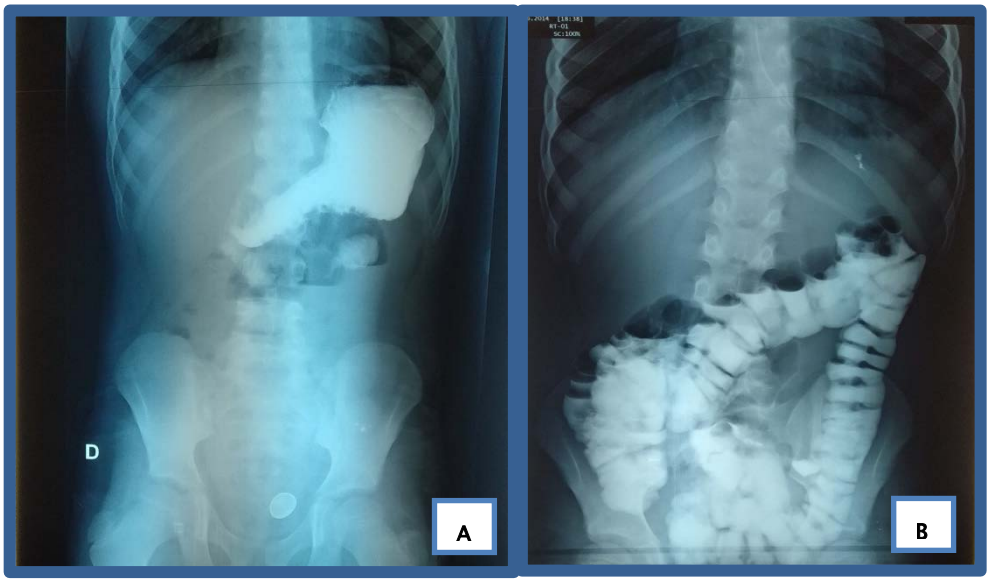


Figure 12: Abdominal Radiographs of a 13 years old male patient. A- AXR obtained 3 hours after Gastrographin administration showing contrast in the stomach. B- AXR obtained 12 hours after Gastrographin administration showing the presence of the contrast in the large bowel

Figure 13 : Abdominal Radiographs of a 9 years old male patient , A- AXR showing Air-fluid levels and dilated loops of bowel . B- AXR obtained 12 hours after Gastrographin administration. C- AXR Obtained 18 hours after Gastrographin administration showing the contrast in the large bowel and the rectum .

2. Operative management

2.1. Timing

27 patients (44 %) out of 61caseshave been managed operatively, eleven (18 %) of them have had immediate surgical exploration after their admission, the remaining 16 patients (26 %) underwent delayed surgery after unsuccessful conservative management trial.

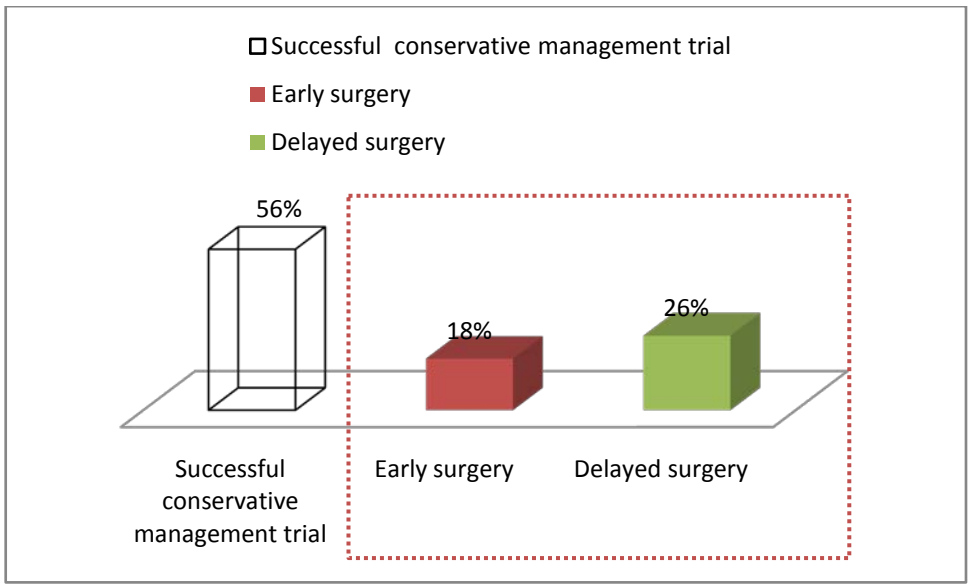


Figure 14: distribution of the operative management timing

2.2. Surgical approach

All operative managements in our studied group have been performed using Laparotomy

2.3. Surgical exploration

Table VIII: distribution of the surgical exploration findings

Surgical exploration findings	Number of patients	Percentage
Single bowel band	15	56%
Multiple bowel bands	12	44%
Adhesions	17	63%
Volvulus	6	24%
Gangrenous bowel	2	8%

2.4. Surgical procedure

Table IX: Distribution of the surgical procedures performed in our studied group

Surgical procedure	Number of patients	Percentage
Adhesiolysis	25	93%
Retrograde decompression (Milking of the intestinal content)	25	93%
Intestinal resection and anastomosis	2	7%
Perforation repair	0	0%
Decompressive enterotomy	0	0%

The intestinal resection was performed in two patients . for the first patient, it took away 25 cm of the ileum length and left out 7 cm of terminal ileum downstream . for the second, the resection took away 15 cm of the ileumlength and left away the last 10 cm of the terminal ileum.

2.5. Post-operative outcomes

One patient had postoperative sepsis that needed a transfert to critical care departement .

VII. Evolution :

1. Length of hospital stay :

Table X: Distribution of the length of hospital stay by therapeutic groups

Group	length of hospital stay		
	min	max	mean
➢ **Conservatively managed group**	1 day	8 days	3 days
• **Standard decompressive therapy**	1 day	7 days	2.1 days
• **Gatrographin protocol**	1 day	8 days	3.5 days
➢ **Operatively managed group**	4 days	13 days	6.1 days
➢ **All the studied group**	1 day	13 days	4.4 days

2. Recurrence

There have been 4 cases (6.5 %) of ASBO recurrence, 3 of them were previously managed conservatively (2 patients were managed with standard decompressive therapy and one received WSCA), the remaining case was managed operatively. During the recurrent episodes, the 3 patients who have been previously managed non operatively underwent surgery after unsuccessful conservative trial, while the patient who had been previously managed operatively was successfully managed non operatively.

Table XI : Features of the recurrent cases of ASBO

Cases / Features	Case 1	Case 2	Case 3	Case4
Gender	Male	Male	Male	Male
Age	11 years	12 years	1.5 year	7 years
The Previous surgical condition	Perforated Appendicitis	Perforated Appendicitis	Intussusception	Intussusception
Management of the first episode of ASBO	Immediate surgery	Standard conservative management	Standard conservative management	Conservative management using Gastrographin
Time of recurrence	4 months	6 months	3 days	6 days
Management of the recurrent episode of ASBO	Standard conservative management	Delayed surgery (after failure of conservative management)	Delayed surgery (after failure of conservative management)	Delayed surgery (after failure of conservative management)

3. Mortality

Two cases of death (3.5%) were registered in our studied group.One of themdied after 24 hours of conservative management, from a sudden cardiopulmonary arrest secondary to massive inhalation of gastric content. The second one was managed operatively after 72 hours of unsuccessful conservative management; he diedin the critical care unit from postoperative sepsis.

DISCUSSION

I. Epidemiological data

1. GENDER

Table XII: the sex ratio reported by authors

Author	number of cases with ASBO	sex ratio (M/F)
J. Janik & al ; Canada ; 1981 [13]	131	1,2
Lautz TB & al ; USA 2011 [14]	2089	1,7
Al-Salem ; Saudi Arabia 2012 [15]	44	1,7
A. Eeson & al ; Canada 2010 [16]	151	2,2
Nasir & al ; Nigeria 2013 [17]	618	1,8
Akgur & al ; Turkey 2017 [18]	181	2
CY Lee & al ; Taiwan 2014 [19]	33	1.4
F. Linden; USA 2019 [20]	12	5
Z. Bouhnoun ; Morocco (Fes) 2016 [21]	43	1.2
our study	**61**	**4.1**

Studies conducted in adults, noted that gender was not a risk factor of ASBO . some studies reported males predominance **[22-24]**, while others recorded predominance of females **[25,26]**

in pediatric patients, all the reviewed studies reported a predominance of males with various sex-ratio.

2. AGE

Table XIII : the mean age and age extremes reported by authors

Author	Number of cases with ASBO	mean age	age extremes
A. Eeson & al ; Canada 2010 [16]	151	6.6 years	2 weeks - 17 years
Nasir & al ; Nigeria 2013 [17]	618	4.5 years	3 months - 15 years
Bonnard & al ; France 2010 [27]	8	7 years	4.4 years - 13.1 years
Al-Salem ; Saudi Arabia 2012 [15]	44	5.4 years	1 month - 12 years
C Festen; Netherland 1982 [7]	31	5,2 years	3 weeks – 16 years
LB Chirdan ; Nigeria 2011 [29]	22	7 years	21 days - 14 years
N. Erturk& al ; Turkey 2017 [28]	45	7.75 years	6 days - 14 years
Lautz TB & al ; USA 2011 [14]	2089	12,6 ans	6,8 years - 18,4 years
J. Janik & al ; Canada ; 1981 [13]	131	6 years	1 month - 17,9 years
Z. Bouhnoun; Morocco Fes 2016 [21]	43	9.5 years	9 months - 15 years
our study	61	7.5 years	8 days - 15 years

Adhesive small bowel obstruction can occur at any age [30]. Young et al.[31] reported a higher incidence of ASBO in infants under 1 year of age, irrespective of the initial site or indication for laparotomy. Conversely, the study conducted by N. Erturk et al [28] showed that 82.5% of ASBO occurred among children older than one year, while only 17.7% occurred in infant under 1 year . in the other hand Grant et al [6] found that there is no difference in readmission rates for ASBO between younger and older children when comparing the organ on which surgery was initially performed.

In our study, the mean age of the children with ASBO was 7.5 years, though some studies reported a mean age older or younger than our patients. Nasir et al [17] reported a mean age of 4.5 years, and in the series of Lautz [14] the mean age was 12.6 years.

3. PAST MEDICAL HISTORY

3.1. Medical history

Fewer are the studies that reported the existence of aprevious medical condition in their patients with ASBO. XY Cao [32] Reported 51 cases of ASBOin patients that were being treated for abdominal tuberculosis,and he also reported that Abdominal tuberculosis is an important and increasingly common cause of acute bowel obstruction.

In our study, only one child among our young patients had a previous medical condition; he was treated for abdominal tuberculosis. The same patient also had a peritoneal biopsy two months before the beginning of ASBO symptoms, which would most likely be the cause of those adhesions.

3.2. Surgical history

Table XIV: the mean previous surgical conditions leading to ASBO in children reported by authors

Author	Previous surgical conditions
Akgur& al ; turkey 1991 [18]	1/ Appendicitis = 28.73% 2/ Abdominal trauma = 19.3 % 3/intussusception = 11.6%
Feigin& al ; 2010 [33]	1/Appendicitis= 15.5 % 2/ Congenital bowel defect = 13.8% 3/ Abdominal tumor = 13.2 %
Nassir & al ; Nigeria 2013 [17]	1/ typhoid intestinal perforation= 24.1% 2/Intussusception= 20.7 % 3/ volvulus and malrotation = 17.2%
N. Erturk& al ; Turkey 2017 [28]	1/ Appendicitis *(without perforation) = 15.5% *(with perforation) = 20% 2/ megacolon + ARM = 15.5% 3/ small bowel atresia = 11%
Young & al ; USA 2007 [31]	1/ Appendicitis (without perforation)= 3% *(with perforation) = 13% 2/ Malrotation = 8% 3/ Hirschsprung disease = 6.5%
C.-Y. Lee & al ; Taiwan 2014 [19]	1/ Appendicitis = 37.5 % 2/ Hirschsprung disease = 12.5% 3/ Intussusception= 12.5%
Z. BOUHNOUN ; Morocco (fes) 2016 [21]	1/ Appendicitis * (without perforation) = 11.5% *(with perforation) = 60.5 % 2/ Intussusception = 13 % 3/ Volvulus and malrotation = 5%
Our study	1/ Appendicitis *(without perforation)= 15.79 % *(with perforation) =47.37 % 2/ Intussusception = 7 % 3 / abdominal trauma = 7% 4/ volvulus and malrotation = 5%

Various previous surgical conditions have been reported in children with ASBO. Vijay **[34]** found that Hirshprung disease and Intussusception were associated with high rates of ASBO . in Nasir's Study**[17]** typhoid intestinal perforation was the frequent previous condition.

Appendicitis was reported by the majority of authors [18,33,28,19,21] as the commonest previous condition associated with ASBO in children. Moreover the incidence of ASBO was higher after perforated appendicitis than non perforated one . some studies [13, 31] conducted in children to assess the risk after appendectomy , they noted a low incidence of ASBO after non perorated appendicitis ,elseways the incidence after perforated appendicitis was relatively higher , Young & al [31]reported a rate of 0.3% after non perforated appendicitis versus 3% after perforated appendicitis . this can be explained by the high level of inflammation that occurs during perorated appendicitis and the large laparotomy incision that is performed in this case, which raises the risk of adhesions formation [1]

In our study, the majority of the children with ASBO have had appendicitis.47.37% of cases have had perforated appendicitis and 15.79 % without perforation.

Many studies conducted in African countries [17,21, 34] reported Intussusception as the second previous condition in children with ASBO. Nasir & al [17]reported that 20.6% of the children with ASBO have had previous Intussusception . in our study intussusception was noted in 7 % of the children.

Grant et al [6] found that operation on the ileum of children is followed by a high risk of readmission for adhesionrelated problems. This risk is much higherand it can reach up to 25% when Ileostomy formation and closure is performed, that goes further to confirm our findings of high incidence of ASBO in children with previous midgut malrotation or small bowel perforation resulting of abdominal trauma, their incidence in our study were respectively 5% and 7% . those findings are consistent with other studies reports[17,18,21,31]

Aguayo & al [4] conducted a study on the risk of bowel obstruction after treatment of abdominal tumours; he concluded that Bowel obstruction is relatively uncommon after intra-abdominal malignancies in children, with an overall incidence of 3.7 %. However, Wilms ' tumour, rhabdomyosarcoma and Burkitt' s lymphoma appear to be associated with the highest risk of bowel obstruction in comparison with other intra-abdominal malignancies.

Tree children (5%) enrolled in our study have no medical nor surgical history, and though they presented ASBO . all of them underwent surgical exploration which has confirmed the presence of congenital adhesions. The incidence of adhesions without previous surgery has been reported to range between 3% and 28 % as determined by autopsy [36,37,38]. Congenital adhesion band is a very rare condition[38] which make a few existing studies that reviewed small bowel obstruction by Congenital adhesions. KH Yang [38] reported that 5.9 % of the patients with ASBO included in his research had congenital adhesions, including 2/3 of them that were children. And unlike adult patients, pediatric patients with congenital ASBO showed a high proportion of early operation and bowel resection [38].

3.3. The time interval between the previous surgery and the development of ASBO

Obstruction can occur anytime from the early post-operative period to many decades later after the surgical procedure [39], a Swedish study conducted by RE. Anderson in adult patients showed that ASBO can occur even 30 years after laparotomy[40]. In pediatric patients, most of ASBO occur within the first year after surgery [31,34,41,42]. Akgur & al [18] reported that most of the ASBO occurred less than 3 months after laparotomy, and those episodes were associated with a high success rate of conservative management which was 81% versus 59 % when the time elapsed after the laparotomy is more than 18 months.

In our study, 53.7% of ASBO episodes occurred in less than three months after the surgical procedures, and about 80.3% occurred in less than one year, which is consistent with the previous findings in the literature.

II. PATHOPHYSIOLOGY

1. Etiopathogenesis of adhesions

1.1. Post-operative adhesions formation [43,35,45,48]

Peritoneal adhesions may be acquired or congenital; however, most are acquired as a result of peritoneal injury, which occurs by inflammatory conditions, intraperitoneal infection, abdominal trauma, or most commonly abdominopelvic surgery.

The mechanisms of post-operativeadhesion-genesis are common to all acquired peritoneal adhesions. They involve mesothelial surface disruption with subsequent fibrino-coagulative and inflammatory signalling processes.

When peritoneal serous is injured, the process of repair or healing occurs in two ways; the first one is theusual way which leads to the resolution of the injury and peritoneal repair. The second way is pathological and it leads to persistent adhesions formation.

Immediately after injury, there is bleeding and an increase in vascular permeability with fluid leakage from injured surfaces. Simultaneously, a posttraumatic inflammatory response occurs, with infiltration of inflammatory cells, the release of pro-inflammatory cytokines and activation of the complement and coagulation cascades. The activation of the coagulation cascade results in the formation of thrombin, which is necessary for the conversion of fibrinogen to fibrin, which functions to restore injured tissues and, once generated, is deposited along peritoneal surfaces. Fibrin is a tacky substance and causes adjacent organs or injured serosal surfaces to coalesce.

Under normal circumstances, the formation of a fibrin matrix during wound healing is only Temporary and degradation of these filmy fibrinous adhesions by locally released proteases of the fibrinolytic system occurs within 72 hours of injury. Fibrinolysis allows mesothelial cells to proliferate and the peritoneal defect to be restored within 4 to 5 days, preventing the permanent attachment of adjacent surfaces. This process has the key role in tissue remodelling and repair.

If fibrinolysis does not occur within 5 to 7 days of peritoneal injury, or if the local fibrinolytic activity is reduced, the fibrin matrix persists.If this occurs, the temporary fibrin matrix gradually becomes more organized as collagen-secreting fibroblasts and other reparative cells infiltrate the matrix.The organization of fibrin bands over time and their transformation into mature fibrous adhesions is what enables them to persist These“mature” adhesions are not simply composed of connective tissue; studies have demonstrated that, over time, they become highly organized cellular structures that contain arterioles, venules, capillaries and nerve fibres in addition to collagen.

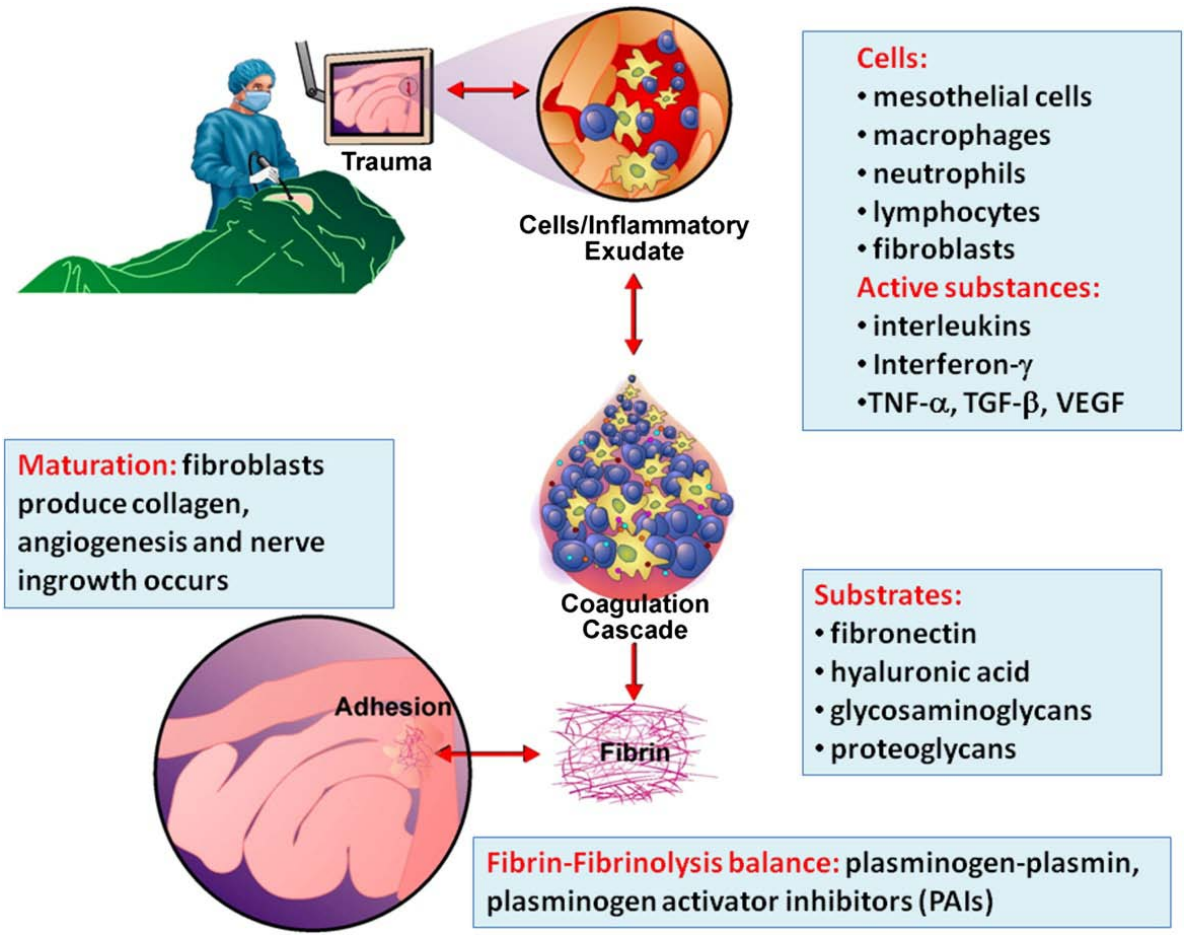


Figure 15: Illustration image summarizes the process of adhesion formation [108]

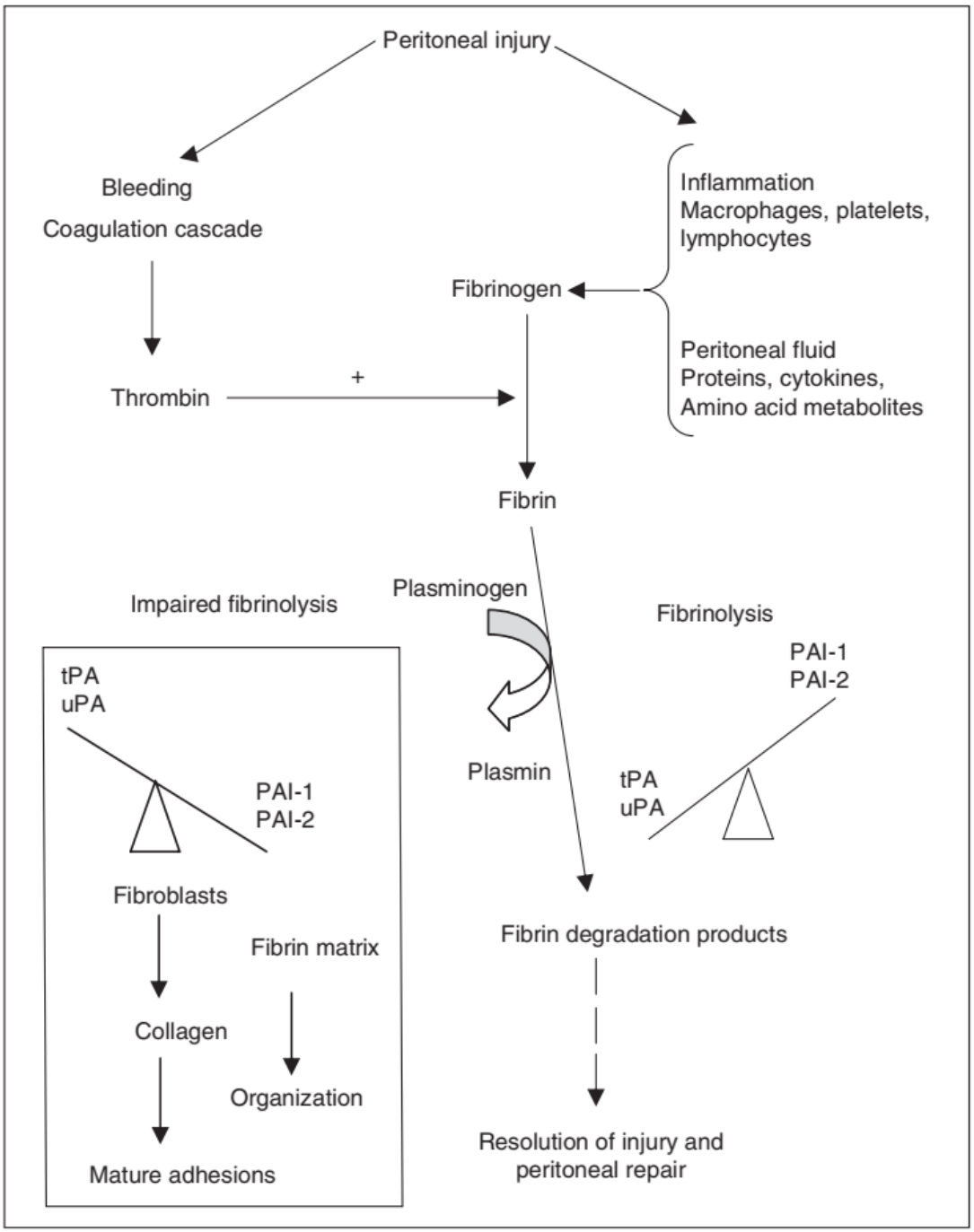


FIG. 1. Biological events involved in peritoneal tissue repair and adhesion formation. PAI-1 = plasminogen activator inhibitors group 1; tPA = tissue plasminogen activator; uPA= urokinase-like plasminogen activator.

Figure 16: Biological events involved in peritoneal tissue repair and adhesion formation[43]

1.2. Congenital Adhesions formation[38,47]

The congenital adhesions andbands have no relation to an intra-abdominal process (previous laparotomy, inflammatory diseases, peritonitis, embryogenic remnants, etc.), they are considered to have embryologic bases, such as incomplete regression of the fetal vitelline circulation, or a genetic defects that impair embryogenesis. Furthermore, congenital adhesions might also be a result of intrauterine exposure to certain infectious agents or ischemic events.

2. Mechanisms of obstruction [44,45,46]

The ASBO is a mechanical obstruction, the mechanism of this obstruction can occur either by direct extrinsic compression . or by volvulus, during which, the loop of intestine twists around itself and the mesentery that supports it, resulting in bowel obstruction and later on ischemia and gangrene due to arterial occlusion

3. Consequences of intestinal obstruction [43,8,47,48]

The pathophysiologic alterations produced by the intestinal obstruction are both local and general. With mechanical occlusion of the lumen,the intestine above becomes distended, resulting from an accumulation of gas and fluid within its lumen. This distention is enhanced by swallowed air, diffusion of gas via the bloodstream, multiplication of bacteria within the stagnant intestinal contents, and hypersecretion of the injured gut.

When intraluminal pressure exceeds capillary and venous pressure in the bowel wall, the intestinal absorption and lymphatic drainage decrease, the bowel becomes ischemic when capillary blood flow stops, allowing bacteria to pass into the peritoneum; from that point, it passes into the bloodstream, leading to septicemia by a process known as bacterial translocation. The peritoneal fluid is continuously secreted by the visceral peritoneum and absorbed by the parietal peritoneum, mostly in the diaphragmatic abdominal surface, where the diaphragmatic pores can be distended as much as 3 times their normal size to allow the passage of bacteria. The colonized fluid is then transported via the lymphatic channels into the thoracic duct, which drains in the jugulosubclavian angle, allowing bacteria to enter the circulation and causing septicemia.

Perforation can develop as the ischemia leads to bowel necrosis. First, lymphatic obstruction occurs because of the lesser pressure in these vessels. This is followed by a venous obstruction, which accelerates the edema process because blood enters the affected bowel segment but does not have a drainage route. Finally, the continuous increase in the bowel wall pressure blocks the arterial vessels, leading to ischemic necrosis and perforation.

The massive third spacing of fluids results from peritoneal reflex hypersecretion. Rapidly leads to shock, contributing to morbidity and mortality. This sequence may occur more quickly in a closed-loop obstruction with no proximal escape for bowel contents.

In addition to the local effects produced by obstruction, most profound systemic alterations occur. These may be summarized as a rapidly occurring dehydration with oliguria, hemoconcentration, hypochloremia, hypopotassemia, alkalosis, retention of certain nitrogenous substances, and alterations of the serum proteins.

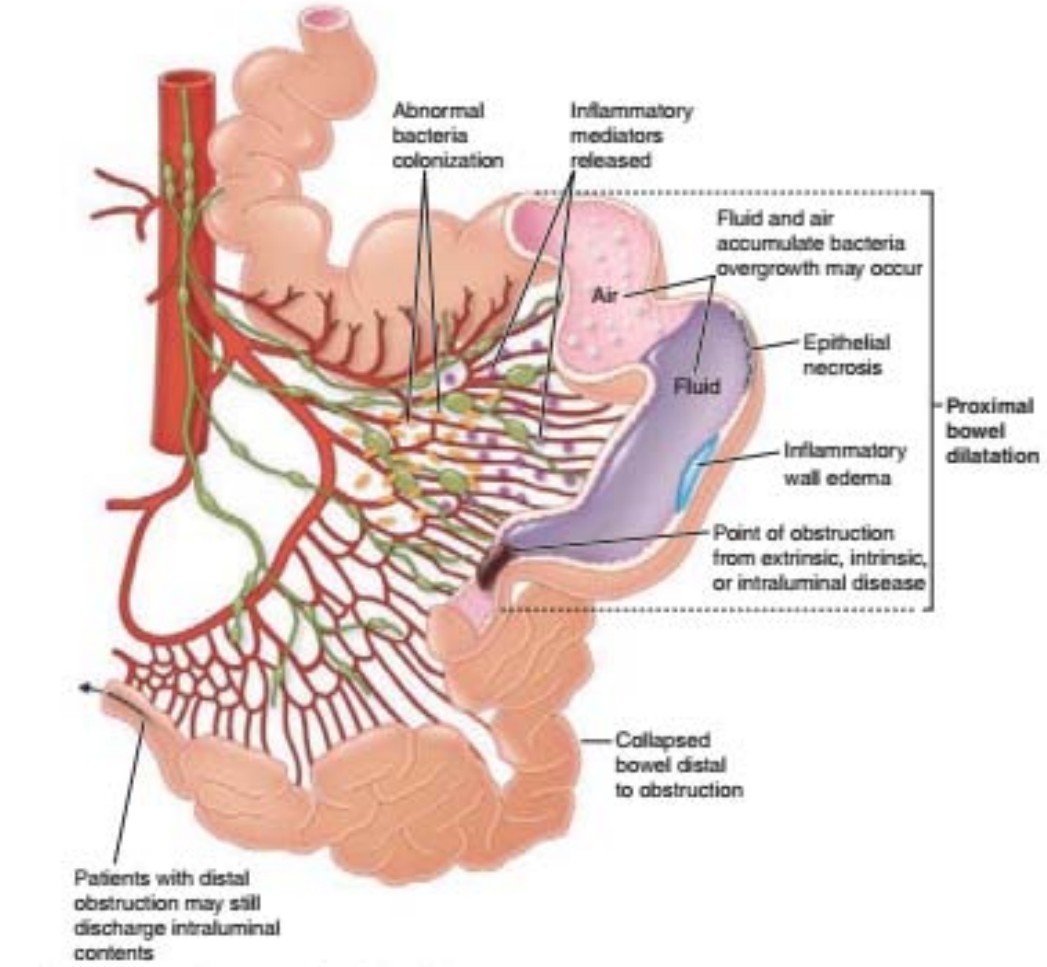


Figure 17: illustration of pathophysiological changes of small bowel obstruction [49]

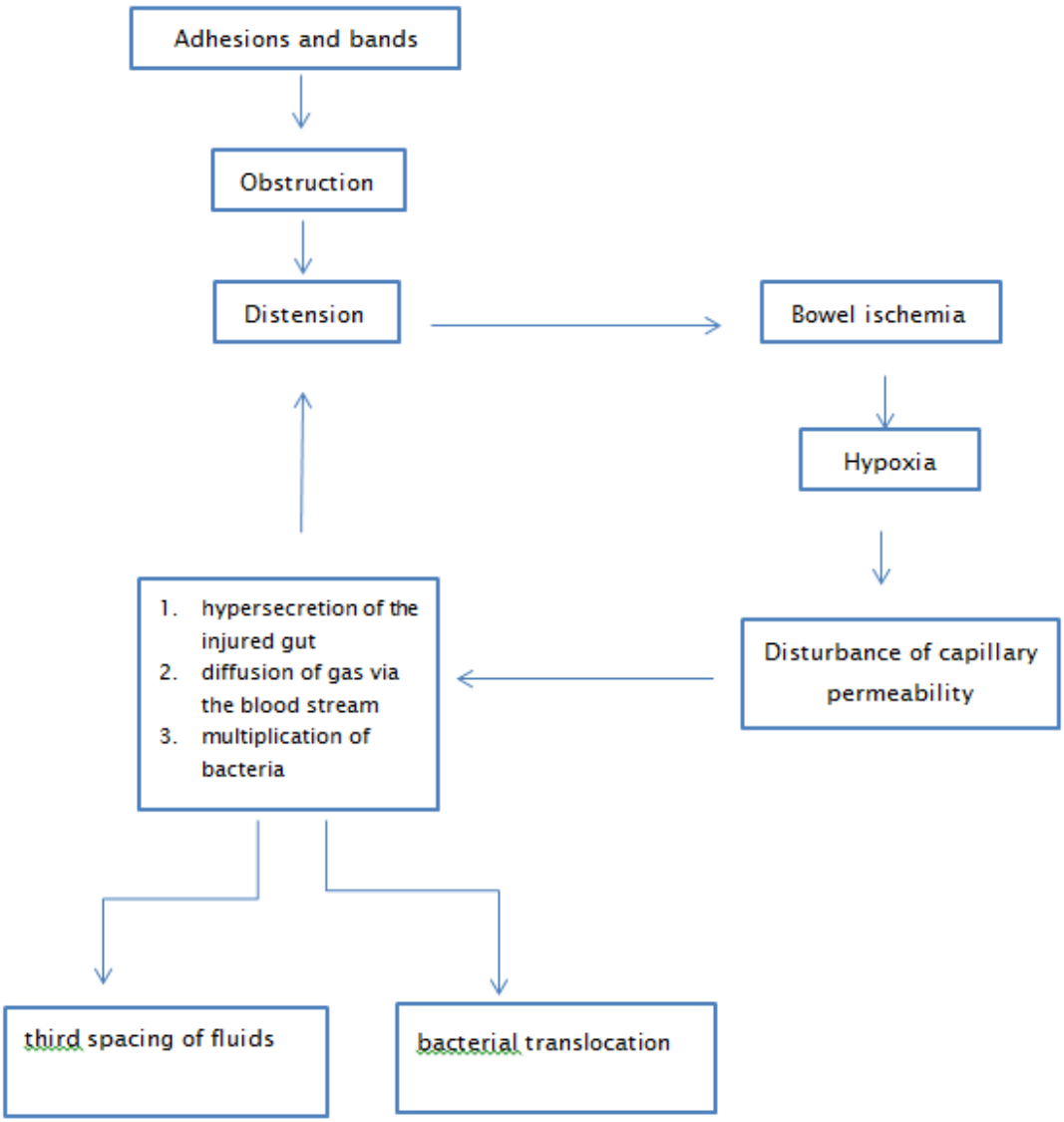


Figure 18: consequences of small bowel obstruction

III. CLINICAL FEATURES [8,44,45,46]

1. SYMPTOMS

1.1. Abdominal pain

Pain is the earliest symptom to appear; it can be acute or progressive . and most commonly of a colicky character. Furthermore, in children, the pain is associated With motor agitation and crying, and as the obstruction progress, The child becomes increasingly lethargic.

Neither the site nor the type of pain can determine the exact location of the obstruction.

1.2. Vomiting

Vomiting is as early as the site of obstruction is more proximal; initially, the emesis may be nonbilious, but with time it progresses to bilious or event feculent emesis . in 30% of cases the vomiting is associated with relief of abdominal pain.

1.3. Obstipation

With partial obstruction, there still be a continuous passage of flatus and small stool. But in children with complete obstruction both of flatus and stool ceases. Interestingly, in early stages, diarrhea may be present and might confuse the clinical picture.

In our study, abdominal pain and vomiting were present in all children (100%), and the obstipation was found in 59/61 patients (97%)

2. Physical signs

2.1. Systemic signs

In the early stage, children with SBO could present Tachycardia along with palor and motor agitation, which can be related to high pain intensity. Later on, when the patients are seen in a late-stage, they can present tachycardia, Fever, hypotension, and dehydration, those signs indicate commonly circulatory collapse and bowel gangrene.

In our study fever was found in 5/57 patient (8%), dehydration in 12/57 patients (24.5%) and the tachycardia in 5 patients (8%)

2.2. Abdominal examination

- **Inspection**

The inspection of the abdomen notices the amount of distension which is greater in case of lower small bowel or colonic obstruction. While in proximal bowel obstruction it might not be so marked .furthermore visible peristalsis under the abdominal wall can be seen . and also abdominal scars related to previous surgery but in case of congenital ASBO abdominal scar is missing.

- **Palpation**

Palpation usually finds abdominal tenderness, diffused or localized . Less commonly, abdominal guarding can be found, which highly refers to bowel necrosis or event peritonitis in case of diffused abdominal guarding.

- **Percussion**

Is of a little value except for tympanic resonance that confirms abdominal distension or dullness over the flanks which suggestive of peritoneal effusion.

- **Auscultation**

Also, it is of a very little help. Initially, it noticed hyperactive bowel sounds due to hyperperistalsis then decrease.

2.3. Rectal examination

A rectal exam must be performed. This gives important information, including the absence of stool, and possibly blood.

IV. PARACLINICAL FEATURES

1. Radiological investigations

Radiologic imaging plays a crucial role in the diagnosis and management of patients with suspected SBO. A myriad of radiologic investigations are available, and the evaluating physician must choose in a reliable, cost-effective way. [46]

1.1. Abdominal X-ray [44, 50,51,52,53]

After a past medical history and physical exam, the initial radiographic study would be plain abdominal radiographs in orthostatic position .even though an upright film is optimal, lateral decubitus or cross-table lateral films could be acceptable substitutes in bedridden patients.

In ASBO, the abdominal radiographs can classically show the following features.:

- dilated loops of small bowel proximal to the obstruction >2.5-3 cm
- predominantly central dilated loops
- valvulae conniventes are visible
- air-fluid levels in erect view study, especially suspicious if >2.5 cm in length and in the same loop of bowel but at different heights
- free intraperitoneal air can be found in case of bowel perforation

Obviously, any or all of these findings may be present, but the skilled clinician should be able to correlate these radiograph findings with the clinical picture. A recent study evaluating plain radiographs in patients with suspected SBO demonstrated good accuracy, with a mean sensitivity of 83% for correctly identifying a SBO[51]. while it has a low specificity in diagnosing an SBO since both mechanical obstruction and functional bowel disorders may appear identical [46]

1.2. Abdominal Ultrasonography [52,54,55]

Ultrasound is a Bedside test also an alternate modality, but really of limited use, especially when intestinal dilation is present and air-fluid levels limit the effectiveness of the study. Adhesions and band are not seen in ultrasound images, but it shows many suggestive findings of small bowel obstruction :

- dilated bowel loop (diameter >2.5 cm)
- ineffective peristalsis (results in "whirling" appearance of intra-luminal contents)
- the prominence of the valvulae conniventes (present in dilated jejunal loops)

Furthermore, ultrasound can help to find suggestive signs of bowel ischemia/ infarction that will need urgent surgical evaluation :

- extra-luminal free fluid (the "pointy" triangular appearance of inter-loop free fluid is often referred to as the tanga sign)
- loss of peristalsis
- bowel wall thickening >3 mm (with effacement of mural architecture)
- intramural bowel gas

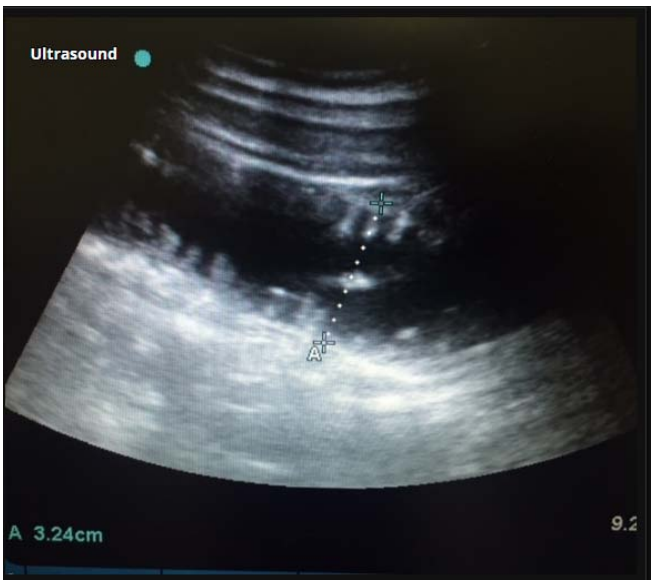


Figure 19: abdominal ultrasound image showing, dilated small bowel loop, with diameter 3.24 cm (> 2.5 cm) [104]

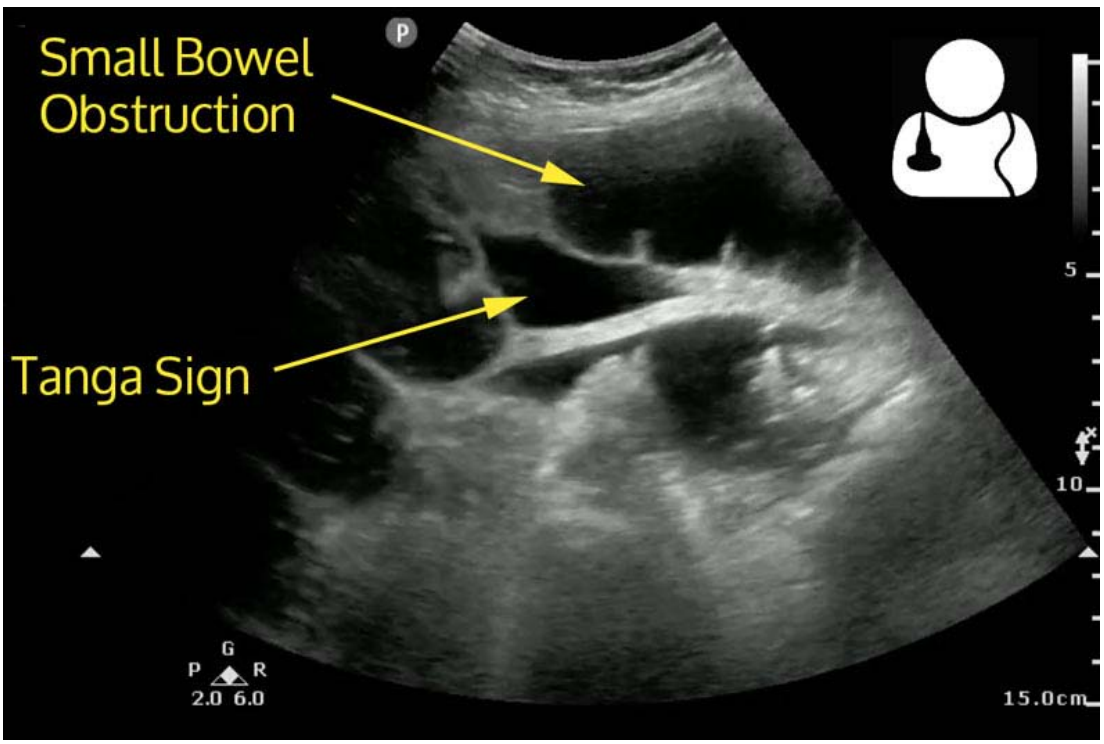


Figure 20: Ultrasound image showing a dilated loop of bowel and extra-luminal free fluid known as "Tanga sign "**[56]**

1.3. Abdominal CT scan [52,57]

Abdominal CT scan, especially with administration of oral or intravenous contrast, performs better than plain Xray in finding the transition point, evaluating the severity of obstruction, identifying the cause of obstruction, and recognizing complications such as ischemia, necrosis, and perforation **[58]**. The sensitivity, specificity, and accuracy of CT scans for ASBO diagnosis are very high; they are, respectively, from 90% to 94%, 96%, and 95% **[59]**.The ASBO Features on CT may include:

- dilated small bowel loops >2.5–3 cm
- normal caliber or collapsed loops distally
- small bowel feces signwhich can be defined by the presence of feculent matter mingled with gas bubbles in the lumen of dilated loops

Figure 21: Axial abdominal CT showing the transition point between dilated bowel loop and collapsed loop (red arrows) **[61]**

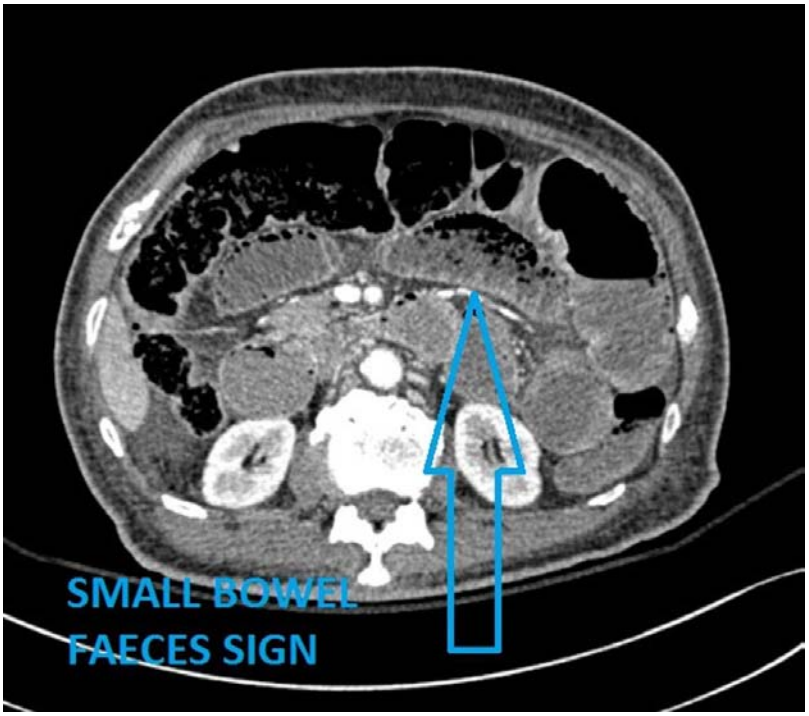


Figure 22: Axial abdominal CT showing small bowel feces sign (blue arrow) [62]

CT scan can also show characteristic features of Closed-loop obstruction (when a bowel loop of variable length is occluded at two adjacent points along its course) :

- radially distributed, C or U-shaped small bowel loops
- " Fat notch sign":reflects insinuation of mesenteric fat at an area of adhesions with focal caliber change
- “beak sign” corresponding abrupt luminal transition and luminal constriction
- two adjacent collapsed loops of bowel

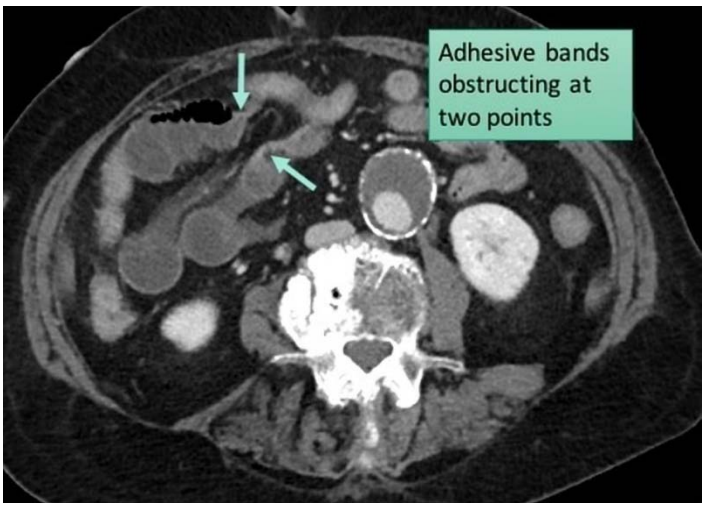


Figure 23: Axial abdominal CT of an adult patient showing closed-loop obstruction caused by adhesive bands**[63]**

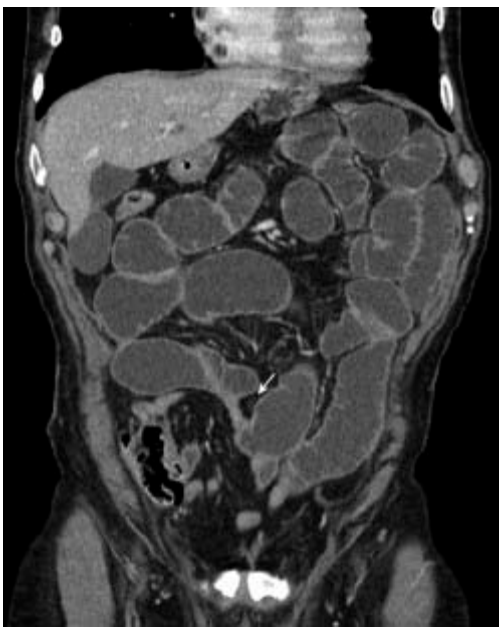


Figure 24:coronal reconstruction of an abdominal CT scan showing the "Fat notch sign" (white arrow) **[64]**

Figure 25 : coronal reconstruction of an abdominal CT scan showing the "beak sign" (double arrow)**[64]**

When the diagnosis of closed-loop obstruction is delayed, it may be associated with intestinal ischemia which is known as bowel strangulation. It is considered as a dangerous complication since it is associated with high mortality. Strangulation presents with non-specific features ,it include**[60]**:

- thickened and increased attenuation of the bowel wall
- halo or target sign
- pneumatosis intestinalis: gas in the intestinal wall
- pneumatosis portalis: gas in the portal vein or in the mesenteric vein
- localized fluid or hemorrhage in the mesentery
- variable amounts of peritoneal fluid

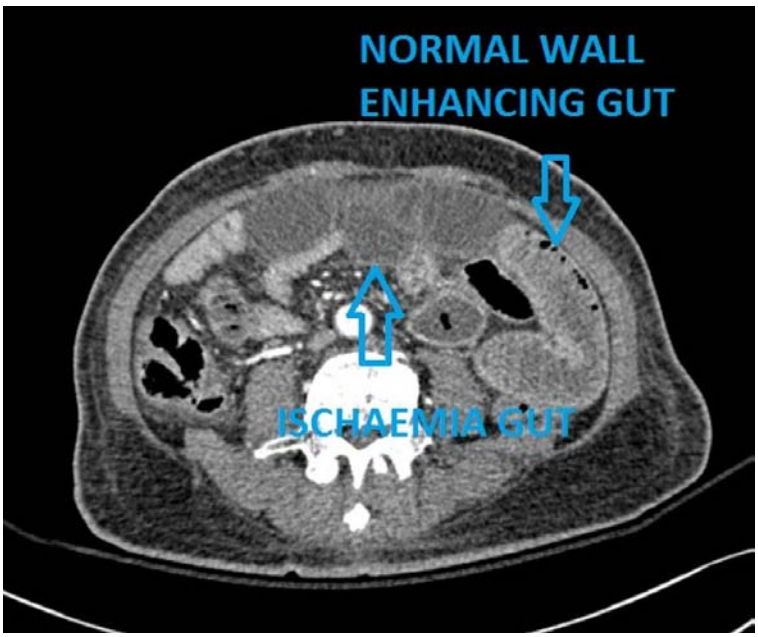


Figure 26: Axial Abdominal CT showing the difference between normal bowel wall enhancement and ischemic bowel wall enhancement **[62]**

Figure 27: Axial abdominal CT showing Intramural bowel gas or pneumatosis intestinali**[65]**

However, abdominal CT scan should not be routinely implemented in the diagnosis making process except when clinical history, physical examination, and plain film were not convincing for ASBO diagnosis, since these are readily available, less expensive, expose the patient to less radiation, and may highlight the need for abdominal CT in some patients . [66]

2. Biological tests

The minimum of laboratory tests includes; blood cell count, lactate, electrolytes, CRP, and BUN/creatinine. Currently, available laboratory tests do not contribute to the diagnosis of SBO, but they can confirm the clinical suspicion of volume depletion, guide volume resuscitation, and assist recognition of bowel ischemia. [67]

Elevated Leukocytosis count and CRP has been implicated as a marker of strangulation, although sensitivity and specificity of these tests are relatively low [68]

Electrolytes are often disturbed in patients with a bowel obstruction; in particular, hypochloremia, hypokalemia, and metabolic alkalosis are frequently found.Dehydration and Hypovolemia may manifest as azotemia with an elevated blood urea nitrogen (BUN) to creatinine ratio (BUN/creatinine), and it could result in acute kidney injury [71]

L-lactate, lactate dehydrogenase (LDH) and creatine kinase (CK) can rise due to hypoperfusion of the intestinal tissue. L-lactate level increases sensitivity for detecting bowel ischemia and is considered a reliable indicator for emergency surgical intervention. In contrast, LDH and CK raised levels are therefore unspecific[69,70]

VI. MANAGEMENT

1. The Goals of management [44,46,47,71]

The management of Adhesive small bowel obstruction in children consists of the following goals :

- ✓ Resuscitation, restoring of normal fluid volume and the correction of electrolytes imbalances
- ✓ Relief of pain and discomfort
- ✓ Achieving resolution of the obstruction and minimize morbidity and mortality

2. Therapeutic approaches and Indications

2.1. Conservative management

2.1.1. Standard conservative management without WSCA

The cornerstone of non-operative management is gastro-intestinal decompression with a nasogastric tube and bowel rest by nil peros. Moreover it consists of, intravenous fluid hydration, correction of electrolytes imbalances, nutritional support, Strict input and output measurements, symptomatic treatment, and Prophylactic antibiotics that should be administered as the clinical situation dictates .in addition to that patients should be Frequently reassessed to make sure that the patient is not developing suggestive signs of complications . [71,72]

Table XV: Distribution of ASBO's therapeutic approaches and the success rate of conservative management according to studies reports

Author	number of patients with ASBO	number of ASBO episodes	number of episodes underwent immediate surgery	Number of episodes managed conservatively		the success rate of conservative management	
Akgür (1991) [18]	181	230	81 (35 %)	149 (65 %)		73,8 % (110)	
Vijay (2005) [34]	69	74	5 (7 %)	69 (93 %)		52,2 % (36)	
Eeson (2010) [16]	151	165	32 (19 %)	133 (81 %)		16 % (26)	
Osifo (2010) [73]	21	21	0 (0%)	21 (100%)		0% (0)	
Feigin (2010) [33]	128	174	0 (0%)	174 (100%)		63% (109)	
Bonnard (2010) [27]	24	24	0 (0%)	24 (100 %)		58 %	
				* without WSCA 16	* with WSCA 8	* without WSCA 50 % (8)	*with WSCA 75 % (6)
Nasir (2013) [17]	29	34	0 (0%)	34 (100%)		47% (16)	
Bouhnoun (2016) [21]	43	49	1 (2 %)	48 (98 %)		18 % (9)	
our study	**57**	**61**	**11** (18 %)	**50** (82 %)		**68%** (34)	
				*** without WSCA** **17** (28%)	* **with WSCA** **33** (54%)	*** Without WSCA** **76 %** (13)	***With WSCA** **64 %** (21)

The paradigm for the initial management of children with ASBO is similar to that for adults and has not changed appreciably in the last two decades [39]. The last update of guideline that was written by Broek et al under the auspices of the World Society of Emergency Surgery recommends that Non-operative management should always be tried in patients with adhesive small bowel obstruction, Unless there are signs of peritonitis, strangulation, or bowel ischemia [72]

Akgur et al [18] suggested the use of a conservative trial for managing the patients in the absence of fever, leukocytosis, localized tenderness, and complete obstruction. And Stewardson [74] suggested immediate surgery for patients with any of 4 those classic predictors (fever, tachycardia, leukocytosis, localized tenderness).

Inconsistently Eason reported in his study that 46% of patients who resolved without surgery, had at least one of the classic predictors at presentation. According to him, those clinical criteria promote an overly aggressive surgical management, and only fever and leukocytosis could predict the need for operative management. [16]

There is no evidence for the optimal duration of non-operative treatment, but most authors consider a 72-h period as safe and appropriate [72,75,76,77,78]. Conservative treatment can be continued for more than 72 h in cases with persistent high output from a decompression tube, but no other signs of clinical deterioration. Lautz et al [39] recommended an observation period of 24 to 48 hours in stable infants/ young children less than 1-2 years , and 48 hours for children greater than 1-2 years of age . in the absence of clinical indicators suggesting improvement, observation periods greater than 48 hours carry a higher risk of bowel ischemia and necrosis, and the likelihood of resolution of the ASBO is extremely low.

The objective criteria that identify those patients who are likely to respond to conservative treatment does not exist. Eason [16] suggested that the absence of leukocytosis and older age are predictors of successful conservative management. Some authors[71,75]proposed the following as strong predictors of NOM failure:

- ✓ The presence of ascites
- ✓ complete ASBO (no evidence of air within the large bowel),
- ✓ increased serum creatine phosphokinase
- ✓ ≥ 500 mL from the nasogastric tube on the third NOM day.

The success rate of the usual conservative management without WSCA ranged from 0% to 73.8 %. four of those studies included in our review [18,27,33,34]reported that conservative treatment was successful in more than 50% of cases . on the other hand , it was successful in none of the cases in the study reported by Osifo [73], however the author acknowledged that the study was conducted in a poor resource country that lacks adequate facilities and manpower which appears to have limited the capacity for comprehensive conservative treatment and monitoring . Despite the presence of resource, Eeson et al [16]reported a much lower rate of conservative treatment success (16%) than reported in the other studies. The reason for this much lower rate of treatment success is not apparent, but would suggest that children in that study may have had more severe ASBO episodes than those in the other studies. Further studies are needed to identify the characteristics of children with ASBO who are most likely to respond positively to conservative treatment.

In the present study, the success of the standard conservative management without WSCA was observed in 13 cases among 17 cases that were treated according to the standard method, with a success rate of 76 %. which is consistent with the published reports

2.1.2. Conservative management with Water-soluble contrast agent

Water-soluble contrast agent has a diagnostic and therapeutic role [71,79]. Gastrografin is the most commonly utilized contrast medium. It is a mixture of sodium diatrizoate and megluminediatrizoate. Its osmolality is 2150 mOsm/L. Which is higher than the osmolalityof extracellular fluid. It promotes shifting of fluid into the bowel lumen and increases the pressure gradient across an obstructive site. The bowel content is diluted, and in the presence of the wetting agent,the passage of bowel contents through a narrowed lumen is facilitated[79]. Gastrografin also decreases edema of the small bowel wall, and it may also enhance smooth muscle contractile activity that can generate effective peristalsis and overcome the obstruction

[71]. Gastrografin is water-soluble and relatively safe even if the obstruction is complicated by perforation. Complications from the use of Gastrografin in small bowel obstruction are rare, although anaphylactic reactions and lethal aspiration have been described[80,81]

Barium has also been used to evaluate adhesive small bowel obstruction; it is not as easily diluted by the enteric fluid as Gastrografin and provides a better mucosal image on radiography. However,a barium study can be risky because it may become inspissated and completely obstruct the bowel. Barium may spread into the peritoneal cavity if perforation occurs, a condition that is potentially lethal[79]

Contrast agent administration should be preceded by infusion of intravenous Ringer solution or Isotonic saline which would be administered first as boluses based on urine output then as a maintenance infusion based on body weight[27] . to guard against dehydration caused by the effect of the contrast medium. The patients should be monitored, and their electrolytes imbalances corrected prior to using Gastrografin . after dilution with an equal volume of isotonic solution, The prepared mixture is given according to the age 30–50 mL for under 10 years, and the other patients receive 100 mL. Through the nasogastric tube and kept closed for 3 h and then reopened. [19,27]

Patients should then have serial plain orthostatic abdominal radiographs at 3, 6, 12, and 24 h after contrast administration. after Gastrografin administration. the Management is considered successful when the contrast is visualized in the cecum and a clinical improvement is noted. Once successful management is achieved, the nasogastric tube is removed and feeding is initiated. If the contrast agent failed to reach the cecum, complete obstruction is diagnosed and patients will be scheduled for surgical intervention[19,20,27]

Figure 28: A-Abdominal radiograph of one of our patients ,obtained 12 hours after Gastrographin administration showing dilated bowel loop and Air-fluid levels filed with Gastrographin. B- Abdominal radiograph obtained 24 hours after Gastrografin administration showing that the contrast had reached the large bowel (haustras).

Many studies have been performed to evaluate the role of water-soluble contrast in the management of ASBO in adult patients**[82,83,84]**. They concluded that the contrast agent has great diagnostic benefit in assessing the indication for surgical intervention and the timing of surgery. The use of Gastrografin in ASBO patients helps to promote an earlier resolution of obstruction and also reduces the length of hospital stay compared with standard conservative management. However its role in reducing the rate of laparotomies remain inconclusive . in the other side, other studies that were conducted by Assalia **[85]** and, Di Savario **[86]** they found that Gastrografin decreased the fraction requiring surgery. Branco et al **[87]** and Abbas et al **[83]** reported that the appearance of water-soluble contrast in the colon within 24 hours after administration could accurately predict the resolution of ASBO with a sensitivity and specificity that reach 96 %. Atahan et al. **[88]** concluded that conservative treatment was recommended for

patients in whichcontrast medium is observed in the right colon within 8 hours followingadministration, regardless of the presence of obstruction signs.

Table XVI:the success rate of WSCA protocol according to studies reports

Author	Number of ASBO cases	Number of cases managed conservatively using WSCA	the success rate of conservative management using WSCA
Bonnard (2010) [27]	8	8	6 /8 (75%)
Chee-Yew Lee (2014)[19]	33	19	16/19 (84%)
Abdelkader (2011) [89]	35	12	8/12 (66.6 %)
AF. Linden (2019)[20]	12	12	10/12 (83 %)
Our study	**61**	**33**	**21/33 (64 %)**

Reviewing the literature. Wefound 4 studies which have been conducted to evaluate the Role of WSCA in the management of ASBO in young patients. In all those studies included in our review, the success rate of this approach was high, and it ranged from 66.6% to 84 %.

All authors[19,20,27,89] reported the valuable role of WSCA in ASBO management. Bonnard [27] and Linden[20] administrated the water-soluble contrast early after admission, conversely to the other related studies which delayed the administration until the fail of the 48 hours trial decompressive therapy [19,89]. Those studies conducted by Bonnard and Lindon were the only studies which have directly compared standard GI decompression versus the use of water-soluble contrast in the pediatric population they reported that the rate of success was relatively high in the water-soluble group compared to those treated with standard decompressive therapy , their success rate were accordantly 75% and 83% for WSCA groups versus 50% and 55% for the usual decompressive therapy groups . Furthermore, the length of hospital stay was also shorter in the water-soluble groups.

In our study, the success rate in the group treated with WSCA was 64 %, which is consistent with the studies reviewed. However when compared to our standard decompressive therapy group, this rate was relatively low (64% versus 75%), this result can be related to many limitations including the small number of patients, the difference in numbers in the two compared groups, and the variety of the previous surgical conditions leading to ASBO. Moreover, there was no standard time for the administration of the water-soluble contrast in our studied patients and in some cases a delay of administration, which may have influenced the therapeutic effect.

2.2. Operative management

2.2.1. Timing of surgery

The operative management of ASBO can be performed early or delayed after unsuccessful conservative management.There is an old dogma "Never let the sunrise or set on a small bowel obstruction" was based on the concern that delay in operation was associated with an increased risk of bowel ischemia, the need for bowel resection, and subsequent complications.**[39,]**

Emergent operative management is indicated for patients presenting with signs of bowel ischemia, which may include localized tenderness on exam, tachycardia, fever, laboratory studies indicating leukocytosis **[39,18,74]**. Eason **[16]** reported that only Fever and Leukocytosis were the only reliables that predicted the need for immediate surgery. The rate of patients who underwent immediate surgery ranged between 5% and 35 % **[16,18, 34,89]**. in our study 18 % of the patients were operated on immediately after their admission which is in accordance with the literature .

On the other hand, delayed surgery is performed after the failure of the conservative management . in the reviewed studies the rates of patients who underwent delayed surgery after failure of non-operative management varies between 26% and 100% **[18,33,73]**. In our studied group, 32% of the patients underwent delayed surgery, similarly to the rate reported by Fegin **[33]** which was 33 %.

Some authors indicate that there is a correlation between the timing of surgery and the risk of bowel ischemia. Feigin [33] reported that 31% of the patients managed operatively underwent bowel resection , and none of the patients who were operated on before the 16th hours needed bowel resection , he also reported that the rate of bowel resection in patients who were operated on in the first 48 hours was 12 %, which was lower compared with a rate of 27 % of bowel resection encountered in patients operated on lately after the 48 hours . in light of those findings, feigin [33] concluded that the index of suspicion for compromised bowel rises after 16 hours, and the decision for surgery should be made at approximately 48 hours; moreover the observation should probably not be prolonged more than 72 hours .

2.2.2. Surgical approach

Abdominal exploration through laparotomy has been the standard treatment for adhesive small bowel obstruction. In recent years, however, laparoscopic surgery for ASBO has been introduced[72]. The potential benefits of laparoscopy include less extensive adhesion reformation, earlier return of bowel movements, reduced post-operative pain, and shorter length of stay [90,91,92]

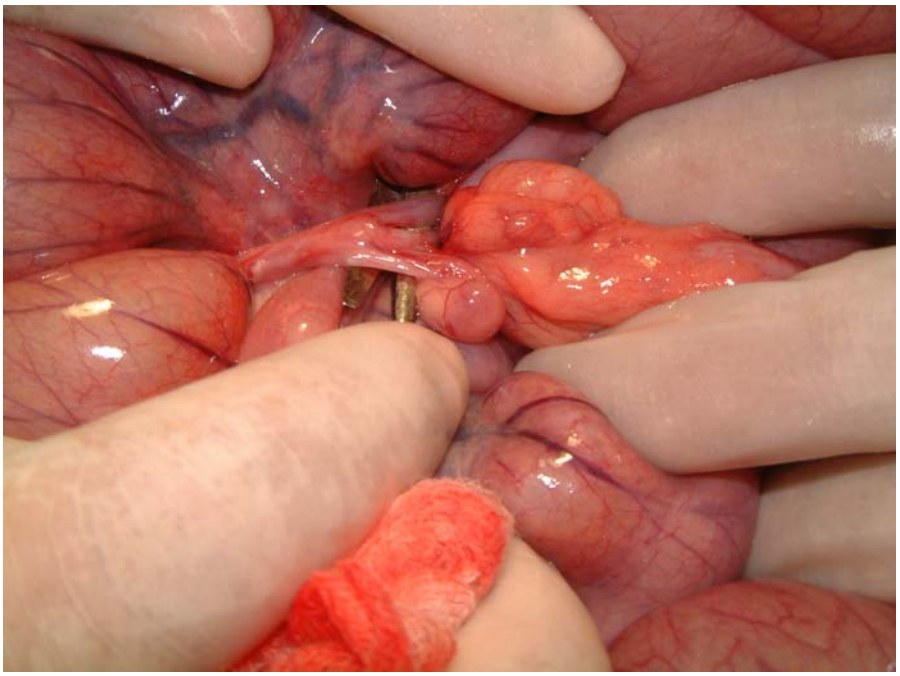
Aguayo et al[4] reviewed an experience with the laparoscopic approach for SBO in 34 children They found that the laparoscopic approach was safe, but that 32% required conversion to a laparotomy, Which can be due to an inability to find transition point, inability to achieve pneumoperitoneum, dense adhesions and gangrenous intestine [93].

The risk of bowel injuries is higher in laparoscopic surgery for ASBO and bowel resections are significantly more frequent in laparoscopic surgery [72] . in a study the Incidence of bowel resection was 53.5 versus 43.4% in laparoscopic versus open procedures [94]. Some authors concluded that laparoscopic approach is safer than the open procedure, but only in the hands of experienced laparoscopic surgeons and in selected patients[95]

Farinella et al reported that predictors for a successful laparoscopic treatment of ASBO are the following: ≤ 2 laparotomies in history, appendectomy as the operation in history, noprevious median laparotomy incision, unique band adhesion as pathogenic mechanism of small bowel obstruction , early laparoscopic management within 24 h from the onset of symptoms , and no signs of peritonitis on physical examination, and experience of the surgeon [96]

2.2.3. Surgical exploration

Surgical exploration tends to search for the site and the cause of obstruction, which can be determined by exploring the transition point between the distended and the flat bowel. Festen [7] reported that in more than 70% of the children with ASBO included in his study, single adhesion was the cause of obstruction , howevermulti-adhesions, volvulus and compressing abscess were also reported to be foundduring exploration .the process of exploration is meant to assess the bowel viability which will further guide to choose the right surgical procedure .



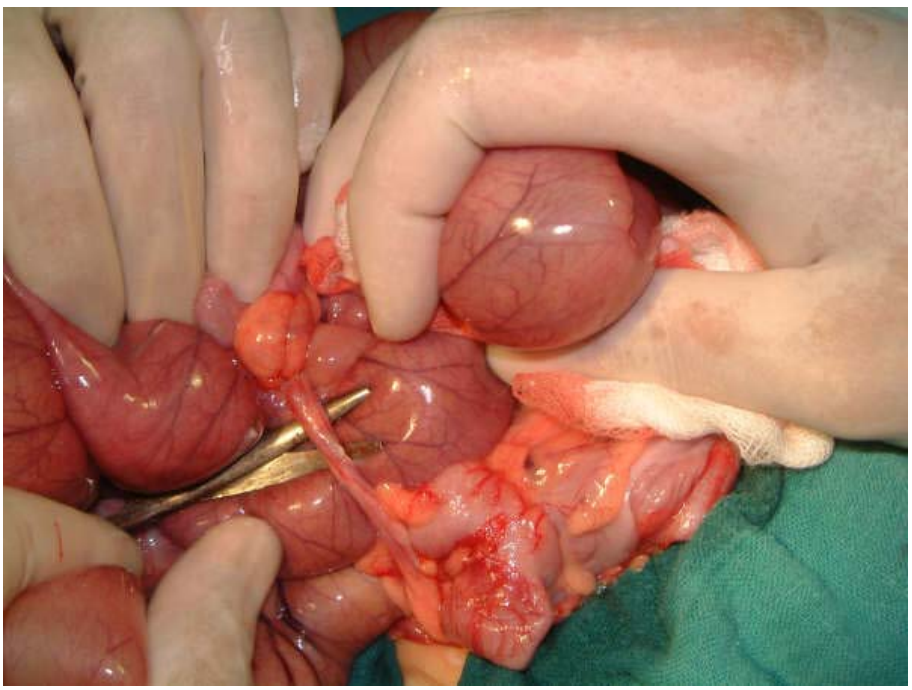


Figure 29: Perioperative picturesof one our patients , showing a congenital adhesive band obstructing the small bowel

2.2.4. Assessment of bowel viability [97]

It is a critical process. The evaluation of bowel viability must be done after the removing of the obstruction (adhesiolysis or untwisting bowel in case of volvulus). Based on several signs this process helps to know if the intestine is going to survive or not :

Bowel is viable if:(1) its surface is glistening,(2) its colour is pinkish, or only slightly blue, (3) it feels resilient like normal bowel,(4) it contracts sluggishly (like a worm) when you pinch it, (5) pulsations are seen in the vessels which run over the junction between it and its mesentery

Bowel is not viable if:(1) it tends to dry out and its surface is no longerglistening, (2) it is greyish purple, dark purplish-red or even black,(3) it feels like blotting paper, (4) it does not contract when you pinch it,(5) its blood vessels are not pulsating or are clotted.

If the process of assessment is inconclusive, remove the cause of thestrangulation, apply a warm, moist pack to it, and wait10mins. If it is viable, its color will change from dusky to normal pink. If this happens, it is alive even if youcannot feel the pulsations of the mesenteric vessels.It may be viable if some areas remain purplish because ofbruising. But if these areas are large, or do not improve incolor, consider all the discolored bowel to benon-viable

Figure 30: perioperative picture showing bowel ischemia

2.2.5. Surgical procedures

- **Adhesiolysis [97,98]**

surgical adhesiolysis is the process of freeing adhesions, it is the current method of managing adhesions .during this process there is a great danger of bowel perforation. and the safest way to separate adhesions is to use the 'push and spread technique preferably with Metzenbaum's or McIndoe's scissors, Using the outer sides of the blades to spread the tissues which are not so blunt .

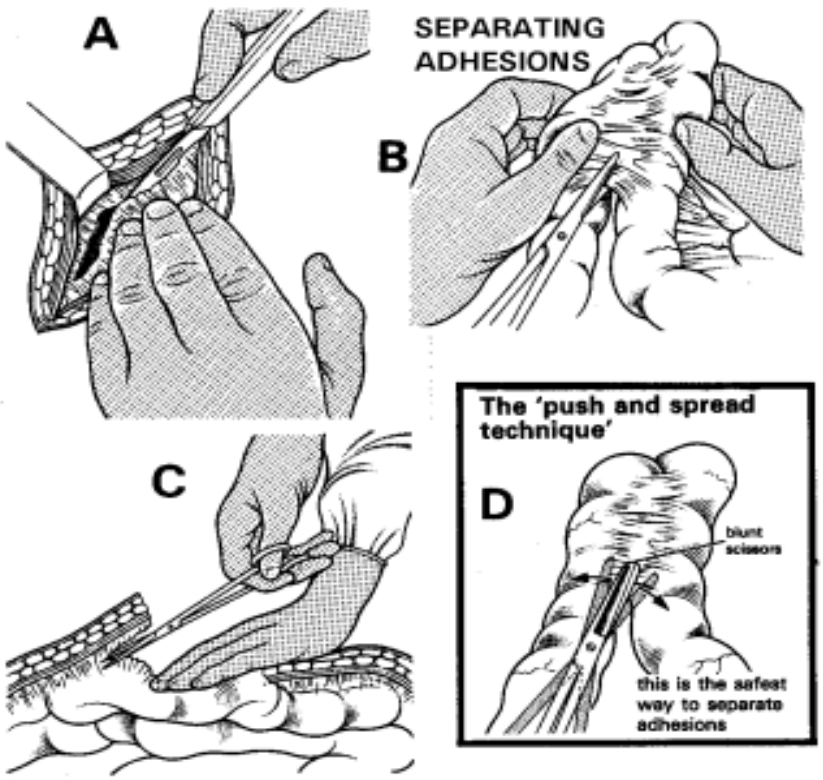


Figure 31: illustration images of adhesiolysis [97]

- **Retrograde decompression [97,99]**

Is the method of choice, provided the bowel is not too oedematous and friable. It is useful for the entire small bowel. It consist of milking the contents of the entire small bowel between the index and the middle fingers, starting from the jejuno-ileum junction and progressing proximally into the stomach . as the decompression is in process the gastric fluid need to be aspirated through a large bore nasogastric tube working properly to avoid fluid spill which may cause aspiration .

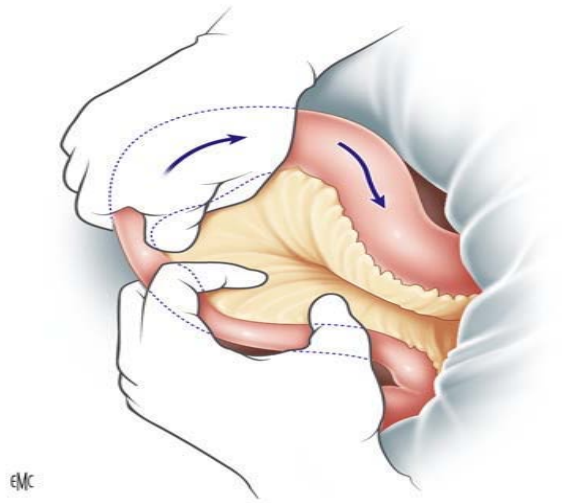


Figure 32: image illustrating the process of retrograde decompression**[99]**

- **Enterotomy decompression [97]**

It consists of making a longitudinal opening in the anti-mesenteric border of the bowel, then inserting a decompressor and sucking out gas and fluid. this method is rarely used it can be the best method in case of bowel resection, during which bowel content is drained from the open ends of the bowel before proceeding to resection.

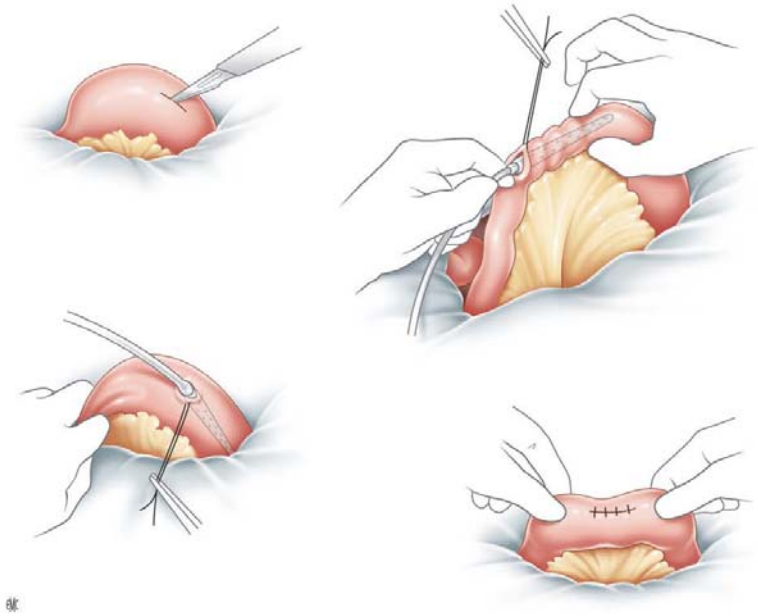


Figure 33: illustration image of enterotomy decompression**[97]**

- **Bowel resection and anastomosis [97]**

If a section of bowel is not viable, it must be resected and the two ends of the bowel joined by anastomosis. Though those ends which will be joined must be viable pink bleeding ends, and the bowel must be emptied before anastomosis

usually ends-to-ends anastomosis is performed, joining their serosal surfaces together by 2 layers with an interrupted or continuous suture. At the end of the procedure, the bowel is washed with warm fluid and gently squeezed on either side to test the anastomosis leak.When it is not safe to make an ends-to-ends anastomosis; a stoma is performed.

Table XVII:the surgical procedures performed by differentauthors

Surgical procédures / Author	Adhesiolysis	Retrograde decompression	Resection – Anastomosis	Enterotomy
N. Erturk [28]	84.5%	not reported	13.3%	2.2
Akgur [18]	82.5%	-	14.6%	2.5
Vijay**[34]	66%	-	33%	27
Eason**[16]	88%	-	9.4%	2.2
our study	93%	93%	7%	0

** Excluding patients managed with immediate surgery

2.2.6. Post-operative outcomes

Many studies have reported the occurrence of postoperative complications [16,17,18].Eeson reported a complication rate of 12% [16]. The most common complications observed were surgical site infection, wound dehiscence, prolonged ileus, sepsis and peritonitis after anastomosis leakage, and pneumonia.

We have registred one case of sepsis and peritonitis that occurred after operative management .

VII. EVOLUTION

1. Length of hospital stay

Table XVIII: the mean length of hospital stay reported by authors

Author	the mean time of hospital stay (days)		
	standard conservative management	conservative management with WSCA	operative management
Eeson (2010) [16]	6.4	-	immediate = 10.4 Vs Delayed= 14
Feigin [33]	4.2	-	10.2
Nasir [17]	5	-	13
Bonnard [27]	6.5	3	-
Abdelkader [89]	-	3.6	-
F Linden[20]	6.2	3.6	-
our study	**2.1**	**3.5**	**6.1**

The length of hospital stay depends on the therapeutic approach and the complications encountered. The studies reviewed reported a length of hospital stay that ranges from 4.2 to 6.5 days for the patients who were managed conservatively without using WSCA, while this length was shorter for the patients who were managed by WSCA, it ranged between 3 and 3.6 days . in the other hand the operative management required much longer time of hospital stay in comparison to the conservative management , the length of stay for patients treated operatively ranged between 6.1 and 14 days . Nasir [17], Feigin [33] and Eason[16] concluded that conservative management is associated with a shorter length of hospital stay than the operative management. Moreover, the administration of WSCA reduces the length of hospital stay significantly compared to the standard conservative management.

Our study reported a shorter length of hospital stay for the patients managed conservatively than those managed operatively, which is consistent with the literature. Conversely, the length of stay for patients managed with WSCA was a little longer compared to the standard protocol which can be explained by the small number of the patients, the inequality of number between the two groups, and the heterogeneity of the previous surgical conditions.

2. Recurrence

Recurrent ASBO continues to be a concern long after the initial treatment of obstruction, with areported range of 16-53% **[18,31,100,101]**. Although surgical treatment of ASBO may lead to the additional formation of adhesions,and recurrent episodes of ASBO. The recurrence is slightly higher for patients treated conservatively than those managed operatively; moreover this rate rises for both approaches after a 5 years period**[72].** Akgur**[18]**reported a double recurrence rate after conservative management compared with the operative management (which was 36.47% vs 18.75%), he also reported that recurrent ASBO episodes could be treated conservatively and the previous treatment has no effect on the rate of success of this management.

No agreement exists about the possibility to predict the recurrence risk**[71]** .though; some authors reported that Factors associated with a higher risk of recurrence are age < 40 years, matted adhesion and postoperative surgical complications**[102]**, moreover, multiple previous operations were associated with increased risk of ASBO recurrence. Lautz et al**[39]** reported that patients with 3 or more previous operations had a relative recurrence risk of 3.2

In our study, we encountered 4 cases (6.5%) of ASBO recurrence, 3 were previously managed conservatively, and only one had undergone surgery, which is accordant with studies reports.

3. Mortality

Akgur et al [18] reported a mortality rate of 1.1% (two deads), one occurred after preoperative perforation complicated to advanced peritonitis and the other after sepsis caused by peritonitis resulting from leakage.

In Festen's study [7]death was encountered in two cases (6.6%), one child died of peritonitis and sepsis caused by an intestinal fistula after decompressive enterotomy, the other one died of sepsis after mesenteric thrombosis.

In our studied group, we have registred two deads (3.5%). One of them died after 24 hours of conservative management, from a sudden cardiopulmonary arrest secondary to massive inhalation of gastric content. The second one was managed operatively after 72 hours of unsuccessful conservative management; he died from sepsis after being transferred to pediatric critical care medicine.

VIII. PREVENTION

Enormous effort has been applied to reduce adhesion formation. There are various approaches to prevent those adhesions which can be separated to three main categories: adjusting surgical techniques, Pharmacological Adjuvant Therapy, Adjuvant Barrier Therapy between or over damaged surfaces, those last two approaches are most common and modern **[35].**

1. Surgical techniques :[35 , 1 , 71]

Postsurgical coalescing adhesions will form only when both contacting peritoneal surfaces have been traumatized during surgery. There are many effective measures, which minimize peritoneal trauma and lead to less adhesion formation; those measures include :

- pursuing general principles of atraumatic, gentle, and bloodless surgery during either laparoscopy or laparotomy
- careful and delicate handling of the bowel in order to reduce severe trauma, keeping tissues moist with irrigation, and avoiding large abdominal wounds and unnecessary dissection
- using micro and atraumatic instruments to reduce serosal injury
- non closure of the peritoneum during the closure of abdominal wounds, since peritoneal closure may induce ischemia and predisposing the site to decreased fibrinolytic activity and increased adhesion formation.
- Using polyglycolic acid derivatives and monofilament synthetics sutures which are associated with less tissue reaction, whereas catgut suture even rapidly absorbed it is highly reactive
- Areas at higher risk of adhesion development may be covered with omentum, peritoneal flaps, falciform ligament, and broad ligament
- Using powder-free gloves (talc and starch), and avoid contact of the peritoneum with other foreign materials such as gauze lint, sutures, and material extruded from digestive tract because they may be responsible for peritoneal inflammatory reaction which potentiates adhesion formation

- In the presence of intraperitoneal blood, it should be aspirated in irrigation solution and adequate hemostasis must be performed.
- Preferring minimally invasive/laparoscopic surgical techniques, since de novo adhesion formation occurs more frequently in patients undergoing laparotomy.

2. Pharmacological adjuvant therapy :[1,35,109]

A variety of pharmacological agents have been studied including steroidal, nonsteroidal anti-inflammatory drugs, heparin, antihistamines, antibiotics, antioxidants, antiangiogenics, and antifibrotics. Those Drugs may be administered systemically, or ideally, locally with minimal systemic effect, and they are directed against various causes and components of the inflammatory process and/or of adhesion formation.

unfortunately, even some of those drugs have been proven to be effective in animals models. There are obvious implications for biocompatibility and toxicity of any material used for adhesion prevention, furthermore there still no clinical trials that have been done

3. Adjuvant Barrier Therapy: [1,35 ,109,110,111]

Most clinical experience is with intraperitoneal adhesion barriers, applied at the end of surgery with the aim to separate injured peritoneal and serosal surfaces until complete adhesion free healing has occurred. Efficacy of anti-adhesion barriers in open surgery has been well established for reducing the incidence of adhesion formation.

- **Barrier solution :**

Hyaluronic acid has been combined with Phosphate-Buffered-Saline (HAPBS) into a macromolecular solution to prevent adhesion formation, marked as Separacoat ®. This solution is applied intraoperatively, prior to dissection, to protect peritoneal surfaces from indirect surgical trauma (e.g., abrasion and desiccation) rather than postoperatively to separate surfaces after they are traumatised.In both animals models and human studies, HAPBS solution was documented to have safely and significantly reduced serosal damage and decreased incidence, extent, and

severity of de novo adhesions.

Adept® is a liquid agent consists of 4% icodextrin in an iso-osmotic electrolyte solution. Icodextrin is high-molecular-weight glucose polymer which results in a prolonged intraperitoneal residence time of the solution allowing abdominal organs to be separated by flotation. The operative handling of Adept is simple as it is introduced via instillation into the peritoneal cavity following laparoscopy where it remains until absorption. It is important to carefully close the abdominal wounds subcutaneously to prevent post-operative leakage of the solution. For this reason, Adept is also not well suited for laparotomy. This solution has demonstrated efficacy in the secondary prevention of adhesions following laparoscopy for adhesiolysis. Unfortunately, Icodextrin has a minimal impact on new adhesion formation.

- **Solid synthetic barrier :**

Hyaluronic acid carboxymethylcellulose (HA/CMC) marketed as Seprafilm® is a solid synthetic barrier that prevents denuded epithelial tissues from touching. It has documented efficacy for abdominal and pelvic surgery, and It has been shown to reduce the incidence, extent, severity, and surface area of postsurgical adhesions, in addition to that it was approved by FDA for abdominal and pelvic laparotomy. beside the high price, the limitations of HA/CMC is that it is indicated for open procedures only and can be difficult to handle because the film is brittle and cracks easily. Also, it fixes upon contact with a moist tissue and cannot easily be moved after application which requires very careful handling. However, it is regularly used off-label in laparoscopic surgery using a cigar roll fashion that eases application without reducing its effectiveness.

CONCLUSION

At the end of this work, and in light of our findings and after analyzing the related studies, we come out with those conclusions :

Adhesive small bowel obstruction is one of the leading emergencies encountered in pediatric surgery that needs early and effective management.

The Peritoneal adhesions are a result of a pathological healing process that follows peritoneal injury, and they occur most commonly after abdominopelvic surgery. However, those adhesions can be congenital.

ASBO can occur at any age, and also after laparotomy for any surgical condition, however history of perforated appendicitis is associated with a high incidence of ASBO

Abdominal pain and vomiting are constant symptoms of ASBO, while obstipation and abdominal distension can be missing in some cases

Abdominal X-ray in orthostatic position plays a crucial role in small bowel obstruction diagnosis. Abdominal CT scan performs better than plain X-ray in identifying the cause of obstruction and recognising complications. Nonetheless, it should not be routinely implemented in the diagnosis making process. The biological tests help to confirm the clinical suspicion of volume depletion and electrolytes imbalances, and also assist recognition of bowel ischemia.

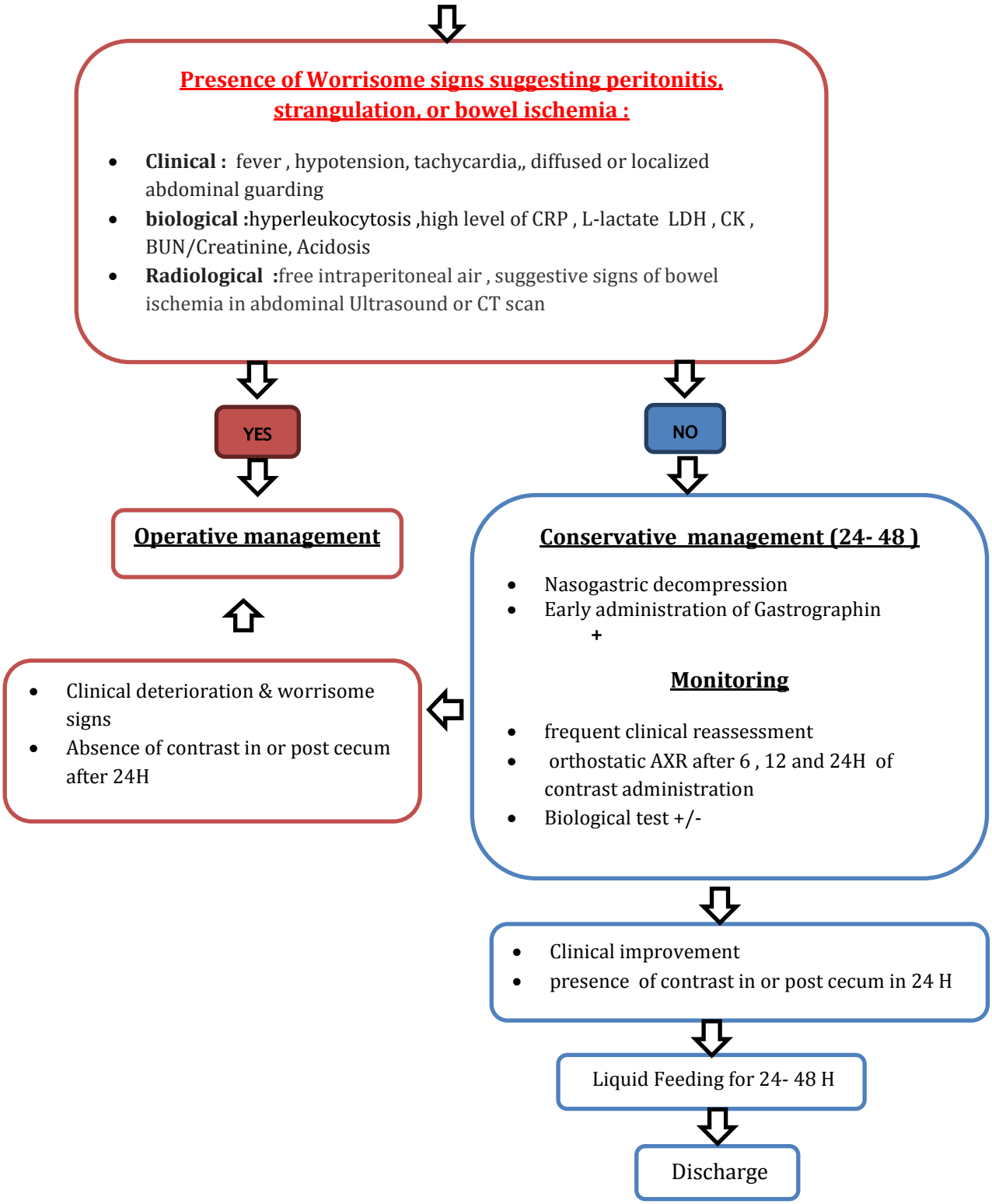
Conservative management, either with standard decompressive therapy or using water-soluble contrast agent, is a safe and effective approach in managing ASBO in selected children without predictor signs of strangulation and bowel ischemia. However, in the absence of clinical indicators suggesting improvement, this approach should not exceed 48 hours of trial.

Laparotomy is used for children with strangulating ASBO and after the failure of conservative management trial. Inappropriate patients, the laparoscopic approach can also be used for managing this condition.

Further prospective studies are needed to establish optimal management for ASBO in the pediatric population.

Algorithm for the management of ASBO in children :

Child admitted for ASBO


Presence of Worrisome signs suggesting peritonitis, strangulation, or bowel ischemia :
Clinical : fever , hypotension, tachycardia,, diffused or localized abdominal guarding
biological :hyperleukocytosis ,high level of CRP , L-lactate LDH , CK , BUN/Creatinine, Acidosis
Radiological :free intraperitoneal air , suggestive signs of bowel ischemia in abdominal Ultrasound or CT scan
YES
NO
Operative management
Clinical deterioration & worrisome signs
Absence of contrast in or post cecum after 24H
Conservative management (24- 48)
Nasogastric decompression
Early administration of Gastrographin
+
Monitoring
frequent clinical reassessment
orthostatic AXR after 6 , 12 and 24H of contrast administration
Biological test +/-
Clinical improvement
presence of contrast in or post cecum in 24 H
Liquid Feeding for 24- 48 H
Discharge

APPENDIX

Appendix I:

PATIENT'S MEDICAL SUMMARY

I- IDENTITY :

- Entry code : .....
- Full Name : .....
- Age : .....
- Gender : .....
- Date of admission : .....
- Date of discharge :.....

II- PAST MEDICAL HISTORY

1) MEDICAL HISTORY : NO ☐ YES ☐ (precise)

.....

.....

2) SURGICAL HISTORY : NO ☐ YES ☐ (precise)

A) The Condition

Appendicitis with perforation ☐ Appendicitis without perforation ☐

Intussusception☐ ☐ Malrotation ☐ Hirschprung's disease ☐

Other ☐ (precise) .....

B) type of intervention (Laparotomy ☐ Laparoscopy ☐)

C) site of intervention (supramesocolic space ☐ inframesocolic space ☐)

III- HISTORY OF PRESENTING COMPLAINT (HPC)

1) Symptoms

A- Abdominal pain : NO ☐ Yes ☐ (if so, precise the abdominal region)

(Right iliac ☐ Right lumbar ☐ left lumbar ☐ Epigastric ☐ Umbilical ☐

hypogastrium ☐ Right hypochondrium ☐ Left hypochondrium ☐ Diffuse ☐)

B- Vomiting : NO ☐ YES ☐

If so , precise the character (non-bilious ☐ Bilious ☐)

C- OBSTIPATION (Failure to pass flatus and stool): NO ☐ YES ☐

D- Other symptoms : .....

2) TIME TO ONSET OF SYMPTOMS AFTER THE FIRST SURGERY

A- 0 – 6 Weeks ☐ B- 7weeks – 3months ☐ C- 3months - 12 months

D- 13 months – 5 years ☐ E – more than 5 years ☐

3) TIME INTERVAL BETWEEN THE SYMPTOMS' BEGINNING AND THE DOCTOR'S VISIT

A- First 6H ☐ B – 6H – 12hours ☐ C – 12H- 24 hours

D- 24H – 48 Hours

IV- PHYSICAL EXAMINATION

1) GENERAL EXAMINATION

A- VITAL SIGNS : RR=…… PR =…….. …. . BP =……/…….. mmHg

T=…… **(FEVER)** NO ☐ YES ☐

B- Dehydration : NO ☐ Yes ☐

C- **General condition** Good ☐ deteriorated ☐

2) ABDOMINAL EXAMINATION

A- ABDOMINAL SCAR : NO ☐ YES ☐

B- ABDOMINAL DISTENSION : NO ☐ YES ☐

C- ABDOMINAL MASS : NO ☐ YES ☐

D- ABDOMINALTENDERNESSE : NO ☐ YES ☐

E- ABDOMINAL GUARDING : NO ☐ YES ☐

3) EXAMINATION OF THE REMAINING BODY SYSTEMS : NORMAL ☐ ABNORMAL ☐

(……

……..)

V- PARACLINICAL INVISTIGATIONS

1) RADIOLOGICAL

A- **ABDOMINAL X-RAY (AXR) :** air-fluid levels ☐ : distended loops of bowel ☐

…

B- ABDOMINAL ULTRASOUNOGRAPHY : ……

……

C- ABDOMINAL CT SCAN : ……

……

2) BIOCHEMICAL

A- CBC : Hb☐ (……) **WBC ☐** (……..)

PLT ☐ (……) Hte (……)

B- CRP : ☐ (……..)

C- Serum electrolytes : ☐ (……

……..)

3) Other

……

……

VI- TREATEMENT

1- CONSERVATIVE MANAGEMENT

A- Usual Conservative protocol NO ☐ YES ☐

Nasogatric aspiration ☐ Analgesics ☐ Antispamodics ☐
Antiemetics ☐ Antibiotics ☐

Resuscitation with intravenous fluids and electrolytes, ☐

a) Duration of the usual conservative protocol :……..

b) Treatement outcome : Successful ☐ Unsuccessful ☐
(……
……).

B- Water-soluble contrast (Gastrografin) protocole NO ☐ YES ☐

a) The outcome : Successful ☐ Unsuccessful ☐

2- OPERATIVE MANAGEMENT NO ☐ YES ☐

If so , precise :

A- Timing : **EARLY** (INITIALLY) ☐ **DELAYED**(after conservative management) ☐
If DELAYED ,pricise the time elapsed after the admission……..

B- Surgical Approach
Laparotomy ☐ laparoscopy ☐

C- Surgical exploration : ……..

D- Operative procedure
Adhesiolysis ☐ Retrograde decompression ☐ resection of bowel ☐
decompressive enterotomy ☐ perforation repair ☐
Other ☐ (……)

E- Postoperative outcomes

- Normal with No complication ☐
- Complication ☐ (wound abcess ☐ fistula ☐ ventral hernia ☐
  Dehiscence ☐ Pneumonia ☐ other ☐ (……
  ……)

VII- Evolution

1) Lenght of hospital staying ……days
2) Death NO ☐ YES ☐
3) Recurrence NO ☐ YES ☐

3

Appendix II:

Anatomical and physiological Reminder

1. Anatomical Reminder [104,105]

1.1. The small bowel

The small intestine is the longest part of the gastrointestinal tract with approximately 6 to 7 meter long .it extends from the pyloric orifice of the stomach to the ileocecal fold, and it consists of the duodenum, the jejunum, and the ileum .

1.1.1. Duodenum

Duodenum is first part of the small intestine, is about 20 – 25 cm long and its lumen is the widest of the small intestine . this C-shaped structure is retroperitoneal except for its beginning , and is adjacent to the head of the pancreas . it is divided into four parts and terminates at the duodenojejunal flexure.

The arterial supply to the duodenum includes superior pancreaticoduodenal branches of the gastroduodenal artery and the inferior pancreaticoduodenal branch of the superior mesenteric artery , those two arteries anastomose with each other .the veins draining the duodenum follow the arterial supply and terminate in the portal venous system.

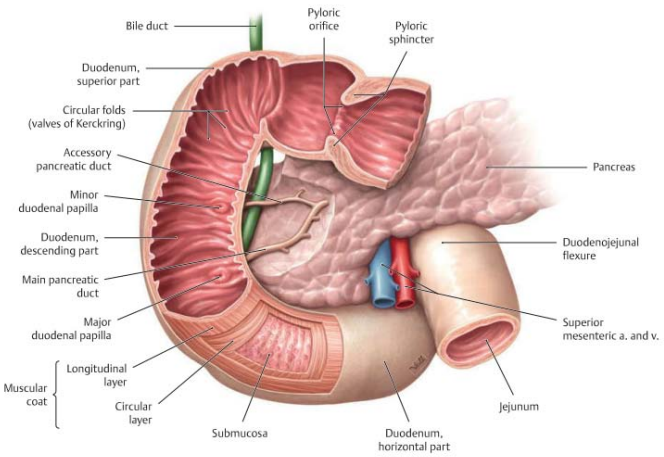


Figure 34 : anterior view of the duodenum with anterior wall opened

1.1.2. Jejunum

By convention, the proximal two-fifths represent the jejunum and the remainder the ileum. However, no precise anatomical feature marks the junction between the two, there being a gradual morphological transition along the whole length of the small intestine . the jejunum is larger in diameter and has a thicker wall than the ileum . in addition to that the inner mucosal lining of the jejunum is characterized by numerous prominent folds that circle the lumen (plicae circulares) but diminish in both size and number along the intestine so that in the distal ileum they are often absent .

The arterial supply to the jejunum includes jejunal arteries from the superior mesenteric artery.

1.1.3. Ileum

The ileum makes up the distal three-fifths of the small intestine . Compared to the jejunum, the ileum has thinner walls, fewer and less prominent mucosal folds (plicae circulares) and it contains lymphoid tissue arranged in discrete clumps (Peyer's patches) .The ileum opens into the large intestine where the cecum and ascending colon join together and it project two flaps into the lumen of the large intestine (the **ileocecal fold**) . Additionally Musculature from the ileum continues into each flap ,forming a sphincter. Which prevent reflux from the cecum to the ileum, and regulate the passage of contents from the ileum to the cecum.

The arterial supply of the ileum comes from ileal arteries of the superior mesenteric artery and an ileal branch of the ileocolic artery (from the superior mesenteric artery).

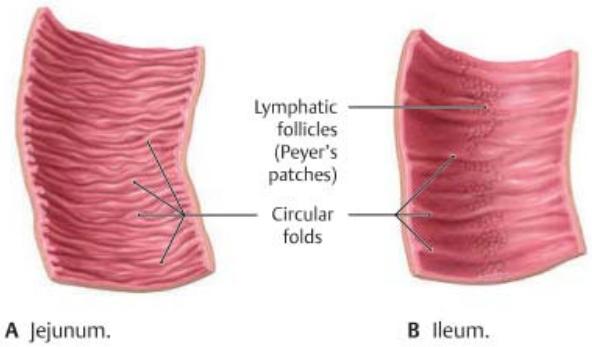


Figure 35: Wall structure of the jejunum and ileum

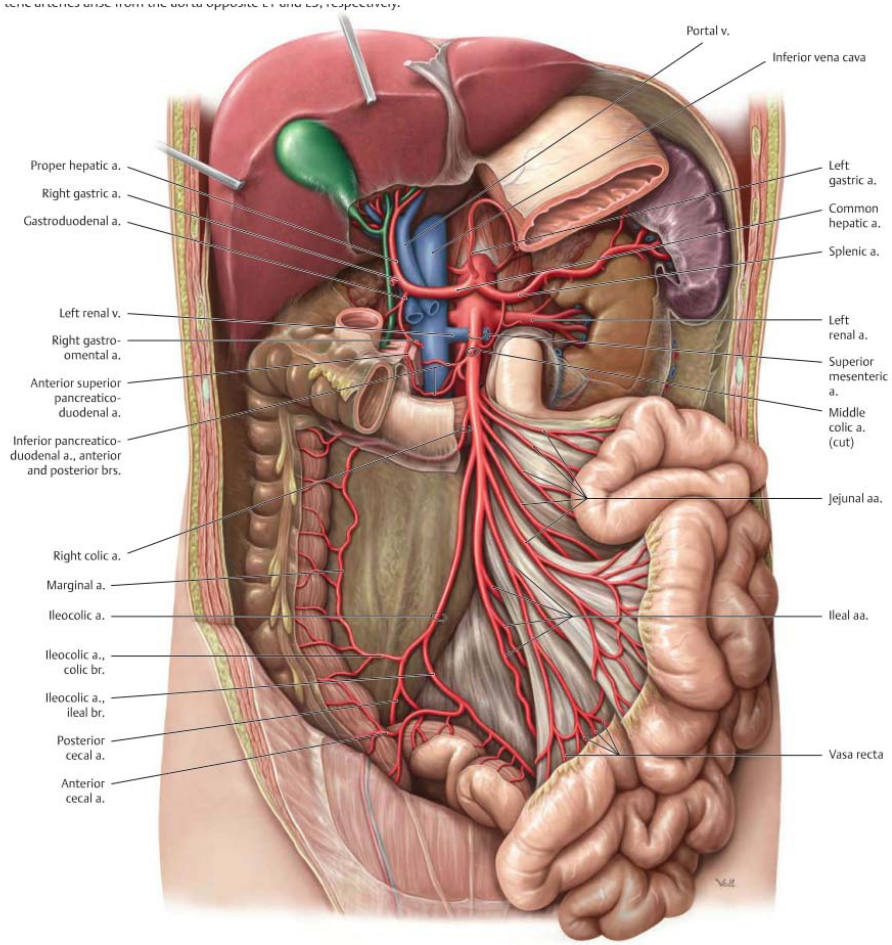


Figure 36: Anterior view showing the arterial supply of small bowel

1.2. The large intestine

The large intestine extends from the distal end of the ileum to the anus. It is approximately 1.5 m long in adult, and its lumen is more wide than the small intestine. It comprises the caecum, appendix, colon, rectum and anal canal. The caecum and appendix lie in the right iliac fossa, while the colon runs a circuitous course forming ascending colon, right colic flexure, transverse colon, left colic flexure, the descending colon and the sigmoid colon, before continuing on posterior wall the pelvic cavity as the rectum.

The large intestine contains Sacculations called (the haustra of the colon) and segregation of longitudinal muscle in its walls into three narrow bands (the taeniae coli).

The blood supply of the caecum, appendix,ascending colon and most of the transverse colon is provided by the superior mesenteric vessels. The remainder of the colon is supplied by the inferior mesenteric vessels. The veinous drainage follows the arterial supply and terminates in the portal venous system.

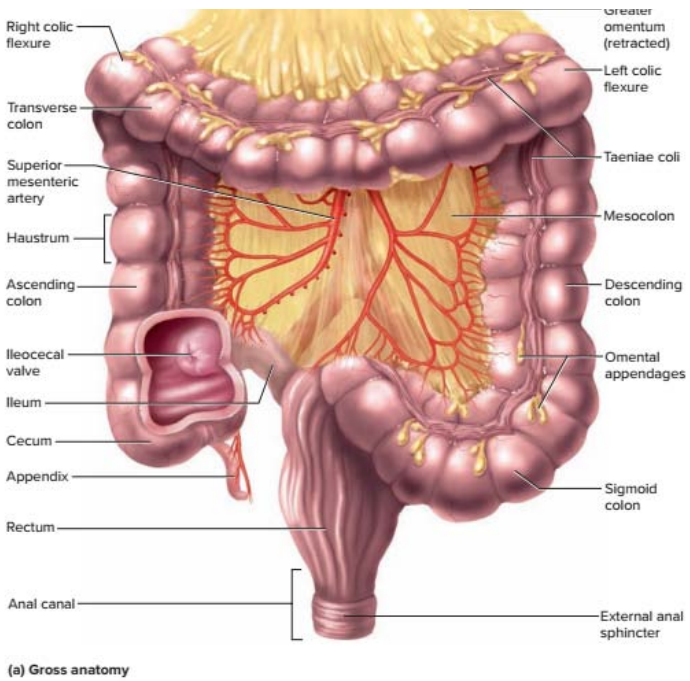


Figure 37: the arterial supply of the large bowel

1.3. The peritoneum and peritoneal cavity

The peritoneum is a serous membrane with parietal and visceral layers. It has almost the same surface area as the epidermis which is approximately between 1 to 2 m2. The parietal peritoneum lines the walls of the cavity and the visceral peritoneum covers the viscera.Between the two layers of peritoneum there is a potential space (the peritoneal cavity). Usually, this cavity contains only a few millilitres (mL) of serous fluid. It comprises the greater and lesser sacs. The greater sac is very extensive; it lies between the diaphragm above into the pelvic cavity below. While the lesser sac or the omental bursa is located in the upper part of the abdomen, behind the stomach and communicates with the greater sac through a narrow opening (the omental or epiploic foramen). The greater sac is divided by the transverse mesocolon into supracolic and infracolic compartments

The structure of the peritoneum consists of a single outer layer of mesothelial cells that are supported by a basement membrane and that detach readily with even the slightest trauma The submesothelial layer consists of components of the extracellular matrix, along with capillaries and lymphatics.Fluid resorption and diffusion occurs freely across these layers.The fluid in the peritoneal cavity contains several different cell types, including leukocytes and macrophages.These cells, along with the mesothelium, secrete various cellular mediators that have roles in peritoneal healing, enabling modulation of the inflammatory response over a large surface area.

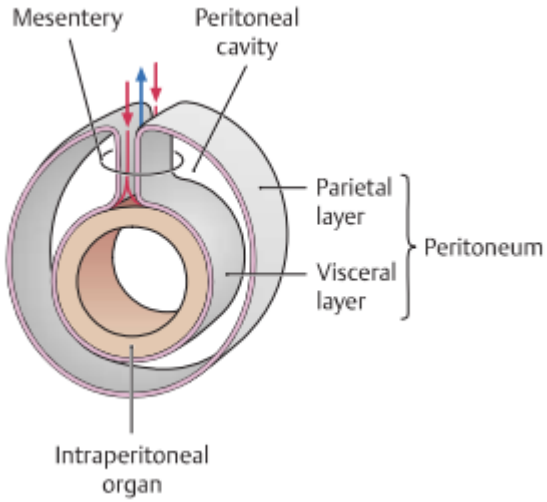


Figure 38: Illustration image showing the disposition of peritoneum layers in the peritoneal cavity

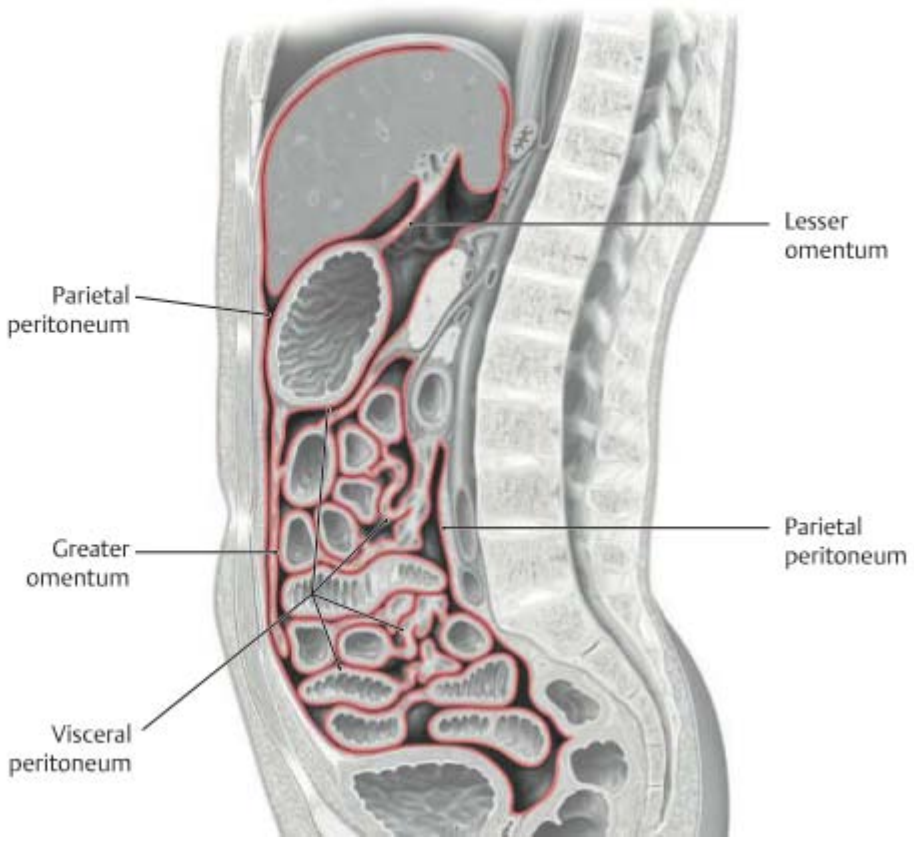


Figure 39: Midsagittal section of the abdominopelvic cavity showing the disposition of peritoneum

2. Physiological reminder [104,106,107]

2.1. Physiology of the digestive system

The primary function of the digestive systemis to transfer nutrients, water, and electrolytes from the food we eat into the body's internal environment. There are four basic digestive processes: motility, secretion, digestion, and absorption :

- **Motility**:refers to the muscular contractions that mix and move forward the contents of the digestive tract. It helps to mix food with the digestive juices, which promotes the digestion of the food and facilitates absorption by exposing all parts of the intestinal contents to the absorbing surfaces of the digestive tract.
- **Secretion**: Several digestive juices are secreted into the digestive tract lumen by exocrine glands along the route, each with its own specific secretory product. Theyconsist of water, electrolytes, and specific organic constituents important in the digestive process. Furthermore there are endocrine cells located in the digestive tract wall secrete gastrointestinal hormones into the blood that help control digestive motility and exocrine gland secretion
- **Digestion**: refers to the biochemical breakdown of the structurally complex foodstuffs of the diet into smaller, absorbable units by the enzymes produced within the digestive system :
    - ✓ Carbohydrates are degraded into simple sugars or monosaccharides ("one-sugar" molecules), such as glucose, fructose, and galactose. These monosaccharides are the absorbable units for carbohydrates.
    - ✓ Proteins are degraded primarily into their constituent amino acids as well as a few small polypeptides, both of which are the absorbable units for protein.
    - ✓ Triglycerides, which are neutral fats consisting of a glycerol with three fatty acid molecules attached. During digestion two of the fatty acidmolecules are split off, leaving a monoglyceride and free fatty acids which are the absorbable units of fat.

- **Absorption :** Through the process of absorption, the small absorbable units that result from digestion are transferred from the digestive tract lumen into the blood or lymph .water and all products of carbohydrate, protein, and fat digestion, as well as most of the ingested electrolytes, and vitamins are mostly absorbed in duodenum and jejunum, very little occurs in the ileum . Otherwise, vitamin B12 and bile salts are exceptionally absorbed in Ileum, because the specialized transport mechanisms for these two substances are located only in this region.

The colon extracts more H_2O and salt from the contents. What remains to be eliminated is known asfeces.

2.2. The functions of the peritoneum

- **Movement of viscera**: the serous fluid secreted by the peritoneum allows free movement of abdominal viscera and peristalsis of the stomach and intestine
- **Defense and Protection**: it guards against germs by phagocyte and plays a role in the immunological response by activating lymphocytes and monocytes .also the greater omentum has a particular power to move toward the site on infection .it plays the role of "abdominal policeman"
- **Absorption**: it absorbs fluid effusion to blood capillaries
- **Repair:** the mesothelial cells of the serosa synthesize inflammatory cytokines, growth factors, and extracellular matrix (ECM), which are actively involved in serosal wound healing and the regulation of peritoneal inflammation.

ABSTRACT

Abstract:

Adhesive small bowel obstruction (ASBO) is one of the leading causes of surgical emergencies. Unfortunately, it does not spare children.

This is a retrospective Study involved 61 cases of ASBO in 57 children, admitted in pediatric surgery department of the Mohamed VI Teaching Hospital of Marrakech, within a period of 7 years, between January 2012 and December 2018. It aims to review the epidemiological, clinical, andparaclinical features, and also outlines the therapeutic aspects of this condition.

A masculine predominance of 80.7% was found. The mean age of our patients was 7 years and 5 months with extremes ranging between 8 days and 15 years.Perforated appendicitis was the most common previous surgical condition leading to ASBO. Three patients (5.26%) have no medical nor surgical history, and one patient was treated for abdominal tuberculosis. Pain and vomiting were present in all children. Obstipation was found in97% of cases. abdominal radiographs showed air-fluid levels and distended loops of bowel in all the patients.

11 cases were operated-on immediately, and 50 cases have had non-operative management trial, using either standard nasogastric decompression or Water-soluble contrast agent, which was successful in thirty-four cases (68%), the remaining 14 cases with unsuccessful conservative management underwent delayed surgery. The surgical management consisted of adhesiolysis with retrograde decompression in 23 patients and bowel resection in 2 patients. The mean length of hospital stay was 3 days for the patients managed conservatively and 6.1 days for the patients who underwent surgery. Recurrence occurred in 4 patients. Mortality was encountered in two patients (8%). Conservative management is a safe and effective therapeutic approach for managing ASBO in selected childrenwithout predictor signs of strangulation and bowel ischemia.

Résumé :

L'occlusionintestinalepar bride (OIB) est l'une des principales urgences chirurgicales , malheureusement , elle n'épargne pas les enfants .

ceci est une étude rétrospective qui a inclus 61 cas de OIB chez des enfants qui ont été admis au service de chirurgie pédiatrique de l'hôpital Universitaire MED VI à Marrakech , sur une période de 7 ans , entre Janvier 2012 et Décembre 2018 . cette étude a pour objectif d'étudier les aspects épidémiologique , clinique et paraclinique et aussi de souligner les aspects thérapeutiques de cette pathologie .

Une prédominance masculine de 80.7% a été retrouvée chez nous patients . l'âge moyen a été 7ans et 5 mois avec des extrêmes entres 8 jours et 15 ans . l'appendicite perforée était l'antécédent chirurgical le plus fréquent . trois patients (5.26%) n'avaient aucun antécédents médicaux ou chirurgicaux et un patient a été suivi pour tuberculose abdominale . la douleur abdominale et les vomissement étaient présents chez tous nous patients ,etl'arrêt des matières et des gaz dans 97% des cas . les radiographies abdominale ont montré des niveau hydro-aérique et la dilatation des anses intestinales chez tous les patients .

11 cas ont été opéré immédiatement , et 50 cas ont bénéficiés de l'essai du traitement conservateur soit par l'aspiration nasogastrique standard ou l'administration deproduit de contraste hydrosoluble ,cette essai a été efficace chez 34 cas (68%) , les 14 cas restants dont le traitement conservateur a échoué ontsubit d'une exploration chirurgical retardée . le traitement chirurgical a consisté l'adhésiolyse avec l'entérovidange rétrograde chez 23 patients et la résection intestinale chez 2 patients . la moyenne de séjour a l'hopital était 3 jours pours les patients traité conservative mentet 6.1 jours pour les patients qui ont subit la chirurgie . la récurrence a été renregisté chez 4 patients . la mortalité est survenu chez deux patients (8%) . le traitement conservateur estun moyen therapeutique sure et efficace pour la prise en charge de OIB chez des enfants bien sélectionnés sans signes predicteurs destrangulation et d'ischemie intestinale .

ملخص

يعتبر انسداد المعي الدقيق بالالتصاقات احدا هما لمستعجلات الجراحية , للاسف هذه الحالة المرضية لا تعفي الاطفال .

هذه دراسة استرجاعية شملت 61 حالة لانسداد المعي الدقيق بالالتصاقات لدى 57 طفلا استقبلوا في مصلحة جراحة الاطفال بالمستشفى الجامعي محمد السادس بمراكش , خلال المدة 7 سنوات بين يناير 2012 ودجنبر 2018 . وترمي هذه الدراسة لمراجعة المميزات الوبائية ,والسريرية , والاسريرية ,وكذا الكبرازمختلف الاوجه العلاجية لهذه الحالة المرضية .

تم ملاحظة سيادة العنصر الذكوري (بنسبة 80.7 %) . متوسط العمر لدى مرضانا كان 7 سنوات و 5 اشهر ومختلف الاعمار من اصغرها الى اكبرها كان بين 8 ايام و 15 سنة . ثلاث مرضى (5.26%) لم تكن لديهم اية سوابق طبية او جراحية , مريض واحد كانت له سابقة علاج لمرض سل البطن .

مرض التهاب الزائدة الدودية كانت الحالة الجراحية السابقة المؤدية اكثر الى انسداد المعي الدقيق بالالتصاقات . الالم والتقيؤ كانا متواجدين عند كل الاطفال , في حين الامساك المعند وجد لدى 97% من الحالات . الاشعة السينية للبطن بينت وجود مستويات بين الهواء – السائل وكذا الكوجود عروات امعاء منتفخة عند كل المرضى .

11 حالة استدعت تدخل جراحي فوري , و 50 حالة استفادوا من المعالجة التحفظية التجريبية , باستعمال التخفيف الضغط الانفي المعدي المعيار ي او استعمال العامل تباين يذوب بالماء . المعالجة التحفظية كانت ناجحة عند اربعو ثلاثون حالة (68%) , الاربعة عشر حالة المتبقية التي لم تفلح معها المعالجة التحفظية خضعوا للجراحة المؤجلة .

المعالجة الجراحية تضمنت عملية فك الالتصاقات و تفريغ الامعاء الرجوعي لدى 23 مريض و عملية قطع الامعاء لدى مريضين . متوسط مدة الاقامة في المستشفى هو 3 ايام بالنسبة للمرضى المعالجين نوفقا لمعالجة التحفظية , و 6.1 يوم بالنسبة للمرضى الذين خضعوا للجراحة . ايبوبة المرض حدثت لدى 4 مرضى . الوفاة سجلت لدى مريضين (8%) .

المعالجة التحفظية هي طريقة امنة و فعالة لمعالجة انسداد المعي الدقيق بالالتصاقات عند بعض الاطفال المنتقين و نعلاماتتنبؤ باختناق و نقصارتواء الامعاء .

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قسم الطبيب

اقَسِمُ باللهِ العَظِيمْ

أن أراقبَ اللّه في مِهنَتِي.

وأن أصُونَ حياة الإنسان في كآفّةِ أطوَارهَا في كل الظروف والأحَوال

باذلا وسْعِي في استنقاذها مِن الهَلاكِ والمرَضِ والألَم والقَلق.

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والله على ما أقول شهيد

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