Lithiase biliaire et imagerie

2021-2022

- Lithiase vésiculaire
  - Diagnostic différentiel
- Lithiase des voies biliaires
  - Dans la voie biliaire principale
  - En intra hépatique
- Cholécystite ou DD

Quelle(s) techniques d'imagerie en 1ère intention ?

Scénario

- Contexte
- Technique
- Conditions cliniques
  - Lithiase vésiculaire
    - Diagnostic différentiel
  - Lithiase des voies biliaires
    - Dans la voie biliaire principale
    - En intra hépatique

Pourquoi utiliser l'imagerie ?

- Bilan d'épisodes de douleur de l'HCD :
  - coliques hépatiques
- Cholécystite:
  - Suspicion
  - Bilan
- Bilan étiologique d'une pancréatite

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ACR Appropriateness Criteria Right Upper Quadrant Pain

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GUIDELINE SUMMARY NGC:010147 1996 (REVISED 2013)

ACR Appropriateness Criteria® right upper quadrant pain.

Developer

American College of Radiology

SUMMARY

- When AC is suspected in patients who have right upper quadrant pain, the diagnosis should be confirmed or excluded using ultrasound and/or cholescintigraphy.
- Ultrasound is preferred as the initial imaging test, with supplemental cholescintigraphy used in problematic cases, if the latter could potentially alter patient management.
- CT or MRI may be helpful in equivocal cases and can be used to identify complications of AC.
- If AC is excluded by ultrasound and/or scintigraphy, CT or MRI may be appropriate, depending on the clinical scenario.

J Am Coll Radiol. Author manuscript; available in PMC 2015 February 18.

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Journal of HBP Surgery

Diagnostic criteria and severity assessment of acute cholecystitis: Tokyo Guidelines

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J Hepatobiliary Pancreat Sci (2012) 19:578–585
DOI 10.1007/s00534-012-0548-0

ORIGINAL ARTICLE

New diagnostic criteria and severity assessment of acute cholecystitis in revised Tokyo guidelines

Masamichi Yokoe · Tadahiro Takada · Steven M. Strasberg · Joseph S. Solomkin · Toshihiko Mayumi · Harumi Gomi · Henry A. Pitt · Dirk J. Gouma · O. James Garden · Markus W. Büchler · Seiki Kiriyama · Yasutoshi Kimura · Toshio Tsuyuguchi · Takao Itoi · Masahiro Yoshida · Fumihiko Miura · Yuichi Yamashita · Kohji Okamoto · Toshifumi Gabata · Jiro Hata · Ryota Higuchi · John A. Windsor · Philippus C. Bornman · Sheung-Tat Fan · Harijt Singh · Eduardo de Santibanes · Shinya Kusachi · Atsuhiko Murata · Xiao-Ping Chen · Palepu Jagannath · SungGyu Lee · Robert Padbury · Miin-Fu Chen

Tokyo Criteria

Table 4 TG13 diagnostic criteria for acute cholecystitis

A. Local signs of inflammation, etc.:

(1) Murphy's sign, (2) RUQ mass/pain/tenderness

B. Systemic signs of inflammation, etc.:

(1) Fever, (2) elevated CRP, (3) elevated WBC count

Tokyo Criteria

Table 4 TG13 diagnostic criteria for acute cholecystitis

A. Local signs of inflammation, etc.:

(1) Murphy's sign, (2) RUQ mass/pain/tenderness

B. Systemic signs of inflammation, etc.:

(1) Fever, (2) elevated CRP, (3) elevated WBC count

C. Imaging findings:

Imaging findings characteristic of acute cholecystitis

Suspected diagnosis: One item in A + one item in B

Definite diagnosis: One item in A + one item in B + C

Imaging findings of acute cholecystitis

Ultrasonography findings (level 4)[2–5]

Sonographic Murphy sign (tenderness elicited by pressing the gallbladder with the ultrasound probe)

Thickened gallbladder wall (>4 mm; if the patient does not have chronic liver disease and/or ascites or right heart failure)

Enlarged gallbladder (long axis diameter >8 cm, short axis diameter >4 cm)

Incarcerated gallstone, debris echo, pericholecystic fluid collection

Sonolucent layer in the gallbladder wall, striated intramural lucencies, and Doppler signals.

Magnetic resonance imaging (MRI) findings (level 1b-4)[6–9]

Pericholecystic high signal

Enlarged gallbladder

Thickened gallbladder wall.

Computed tomography (CT) findings (level 3b)[10]

Thickened gallbladder wall

Pericholecystic fluid collection

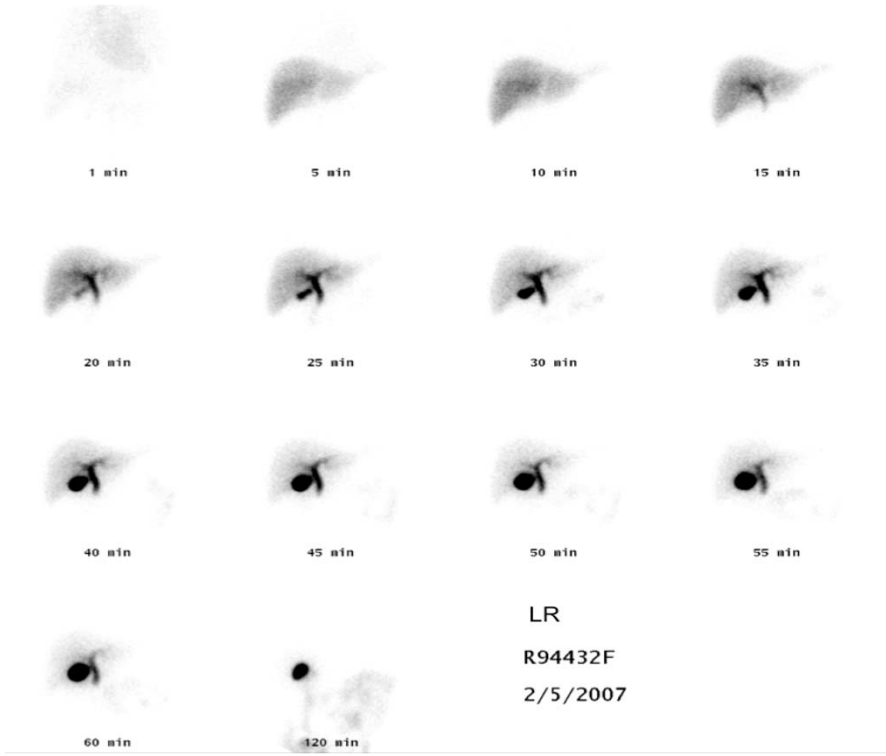
Enlarged gallbladder

Linear high-density areas in the pericholecystic fat tissue.

Tc-HIDA scans (level 4)[11,12]

Non-visualized gallbladder with normal uptake and excretion of radioactivity

Rim sign (augmentation of radioactivity around the gallbladder fossa).



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Pericholecystic fluid collection

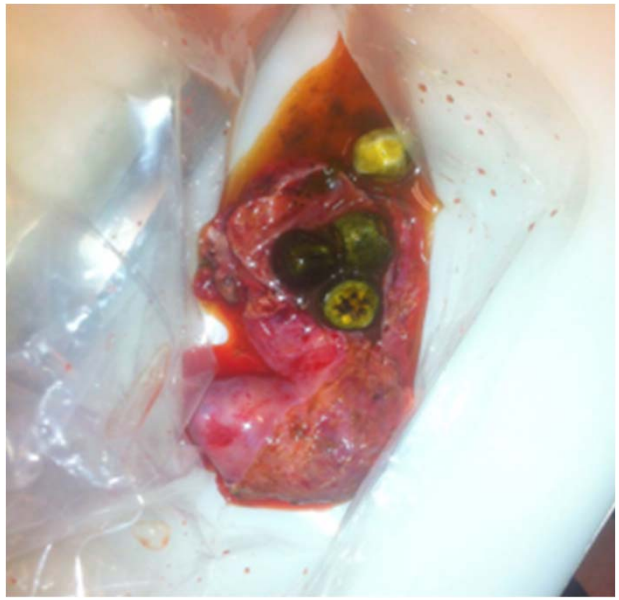
Enlarged gallbladder

Linear high-density areas in the pericholecystic fat tissue.

La lithiase bilaire

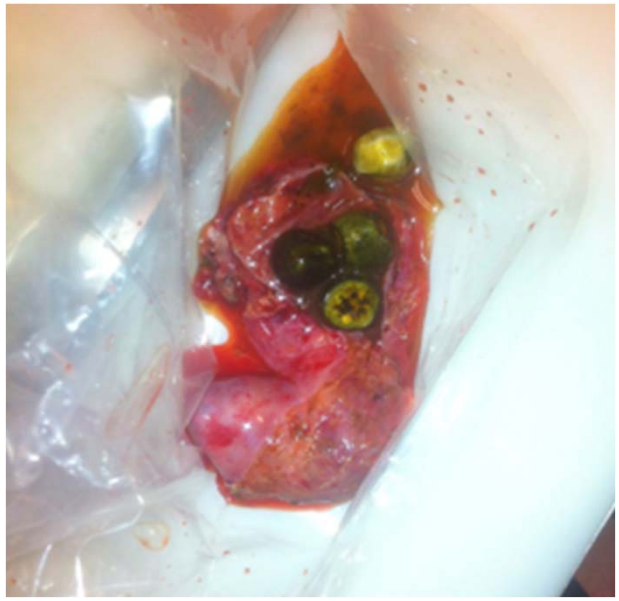
présence de calculs dans les voies biliaires

facteurs favorisants :
sexe féminin , âge , obésité , DNID , sédentarité
hémopathie chez l'enfant (drépanocytose)



La lithiase vésiculaire

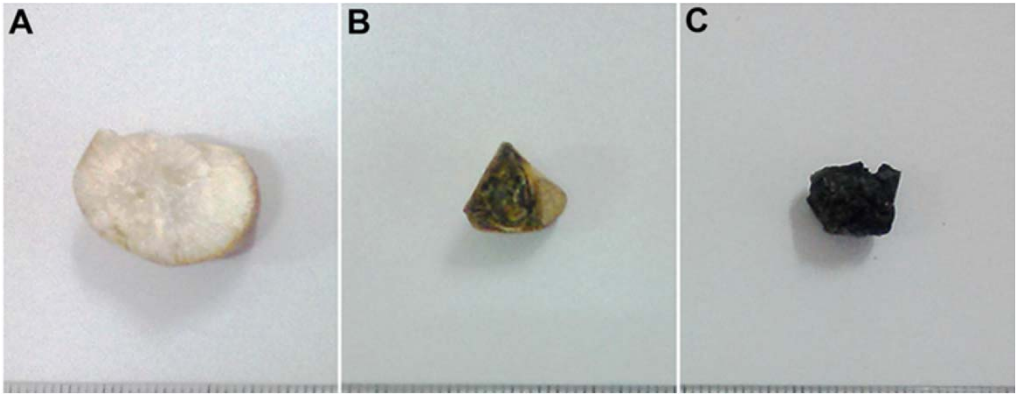
- 10 à 20 % des occidentaux
- 5 millions de français
- 80 000 cholecystectomies / an



La lithiase vésiculaire

conditions de formation
concentration ++ cholestérol / sels biliaires
bile lithogénique
cristallisation dans la couche de mucus
microcristaux sédimentaires : sludge
hypomobilité , stase : phénomène d'agglomération

Composition



Weerakoon et al

Cholesterol pur

Cholesterol mixte

Calcul pigmentaire

UCL

La lithiase biliaire & vésiculaire

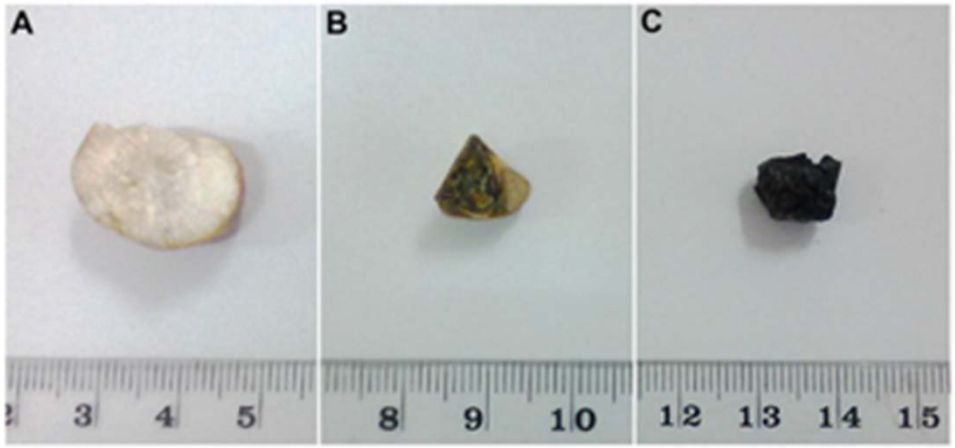


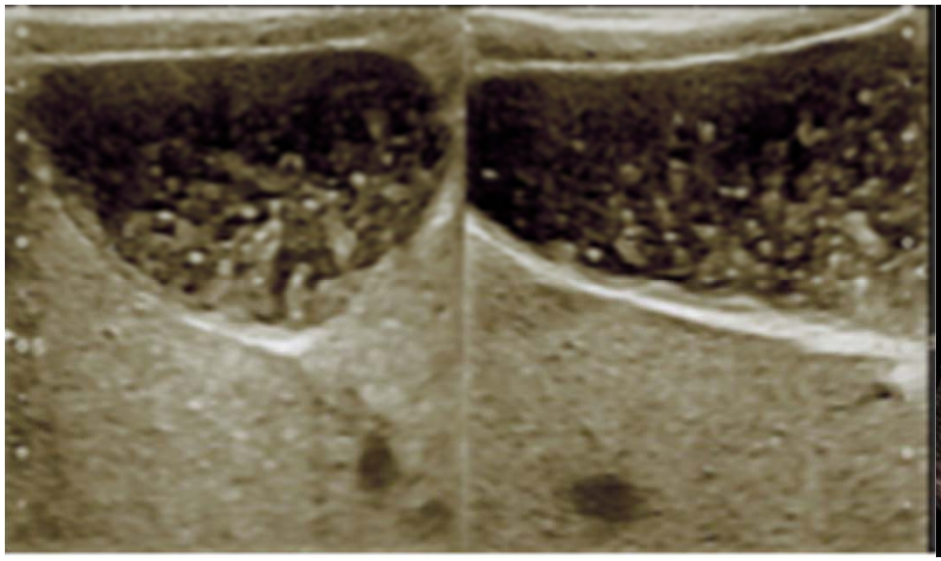
Table 1. Chemical composition of gallstones as revealed by FTIR.

Type of GS	Chemical composition	Frequency *n* (%)
Pure cholesterol	Cholesterol	10 (09)
Mixed cholesterol	Cholesterol, calcium bilirubinate, calcium carbonate, calcium phosphate	38 (37)
Pigment	Calcium bilirubinate	23 (23)
	Calcium bilirubinate, calcium carbonate, calcium phosphate	25 (25)
	Calcium bilirubinate and calcium palmitate	06 (06)

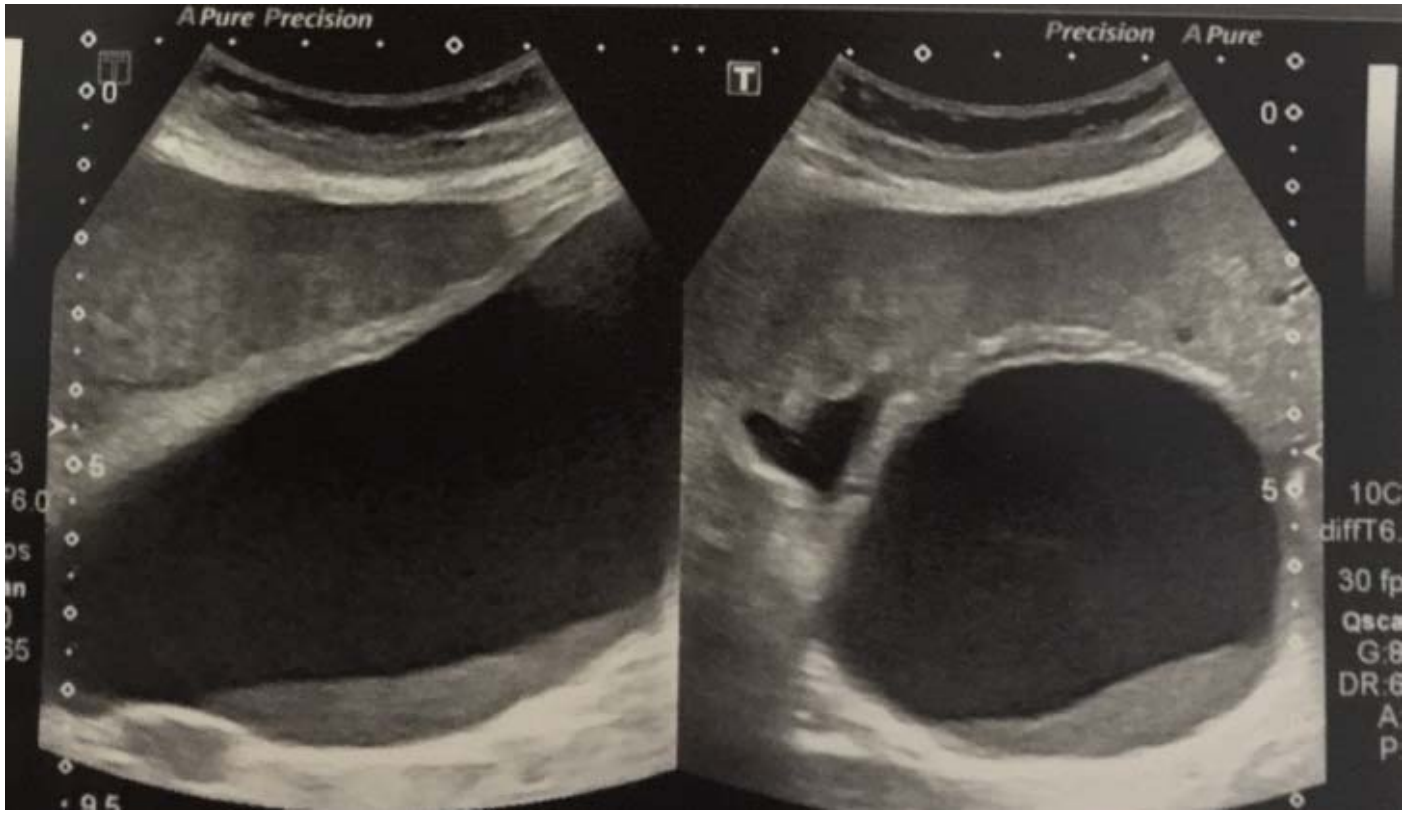
doi:10.1371/journal.pone.0121537.t001

La lithiase biliaire & vésiculaire

Sludge
Microlithiase
Lithiase

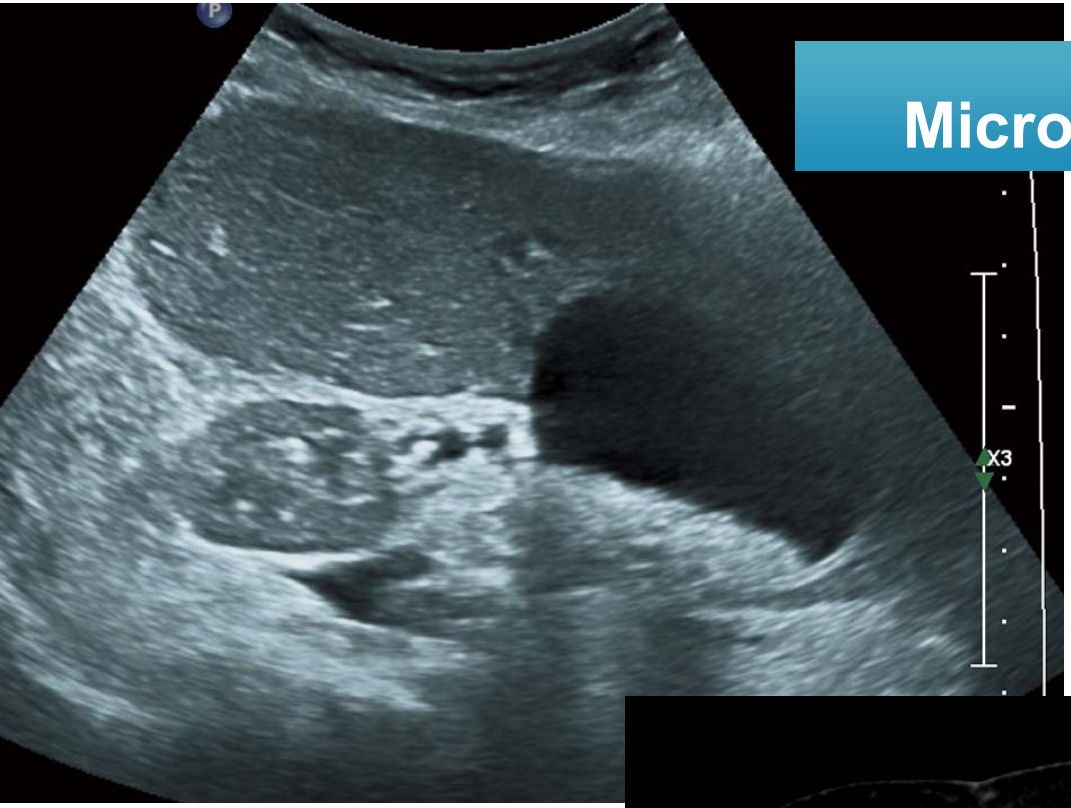


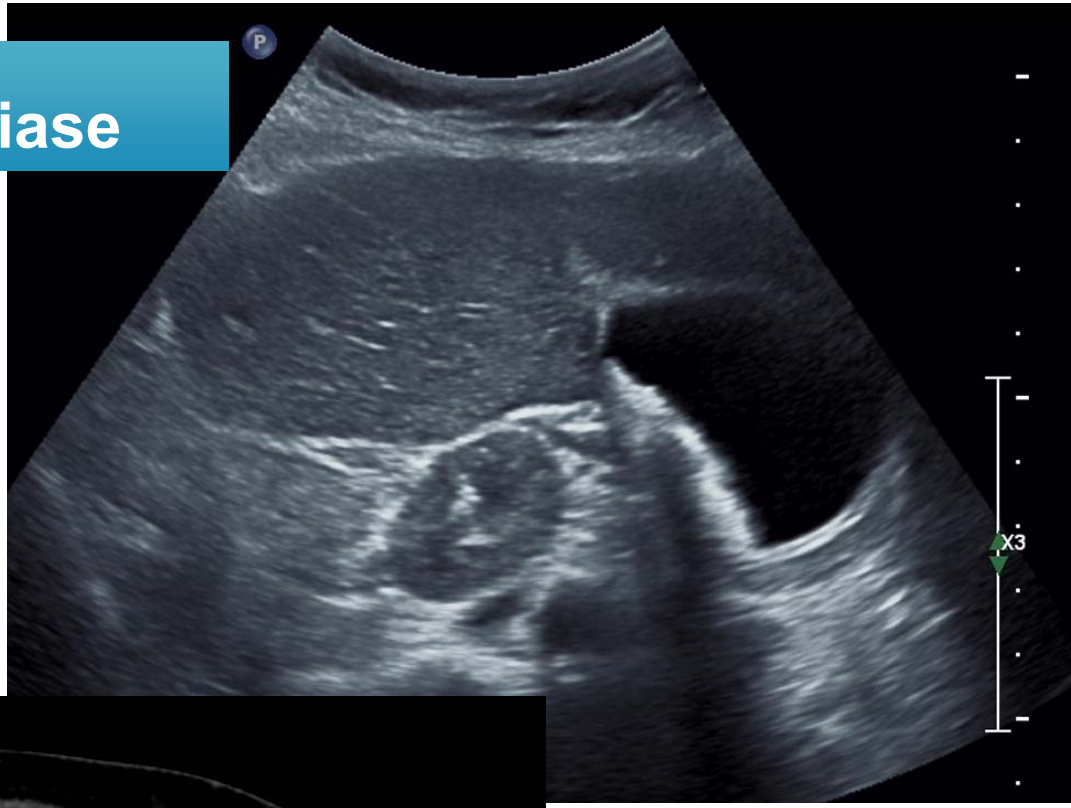
Sludge



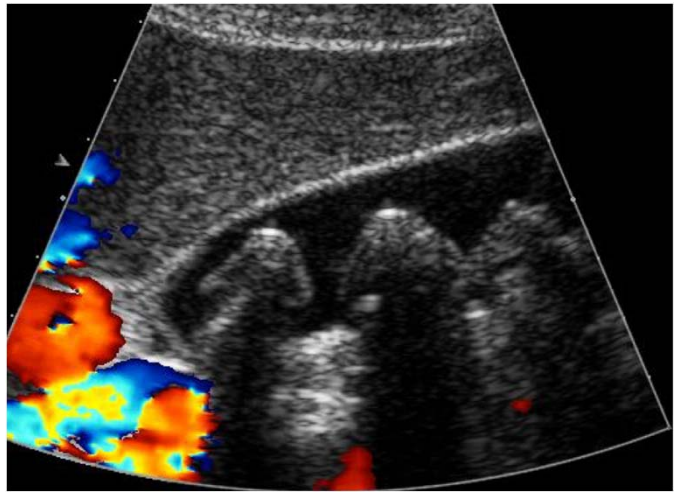
mélange de cristaux de cholestérol, sels calciques et mucus biliaire

Microlithiase





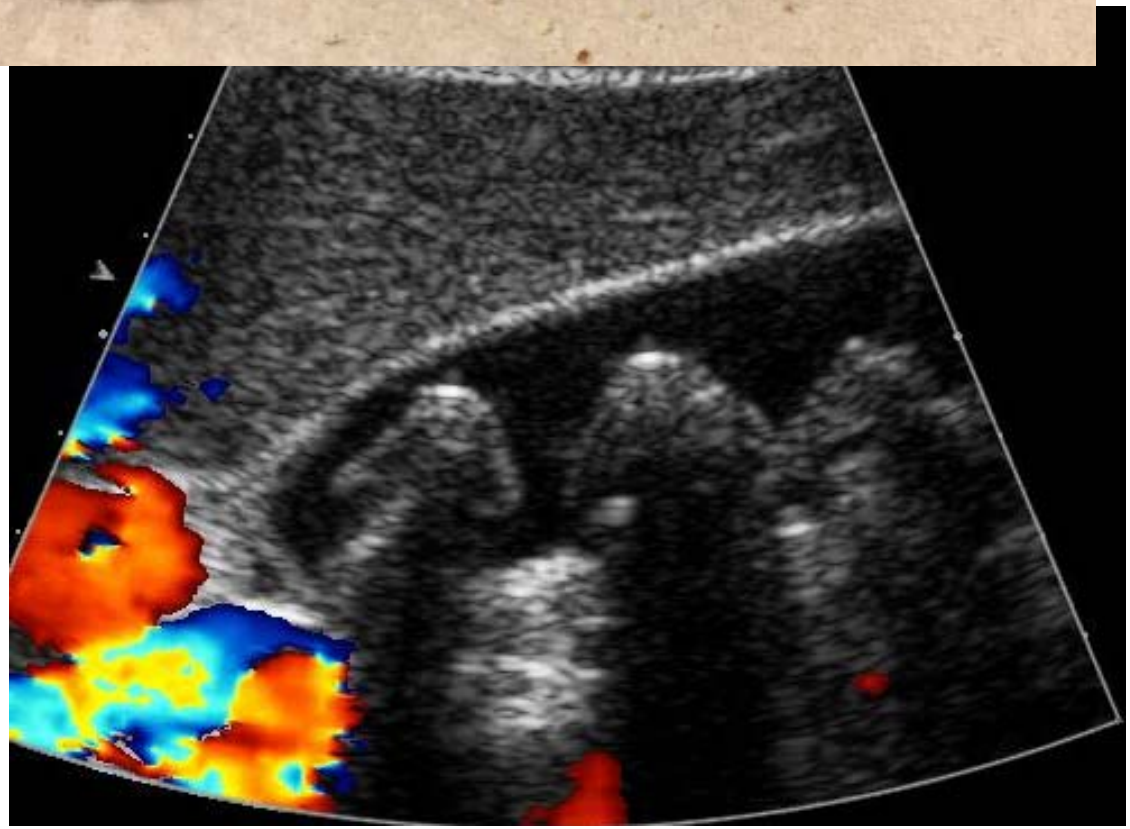
La lithiase cholestérolique



composition cholestérol ++

défaut solubilisation par les sels biliaires + atonie

jaunes/bruns , mous , friables , réguliers avec facettes , radiotransparents , souvent multiples



La lithiase pigmentaire

- noire

(riche en Ca , Cu , Fe , Mg , Mang)

défaut de glucuronoconjugaison : bilirubine non conjuguée +++

hémolyse , cirrhose

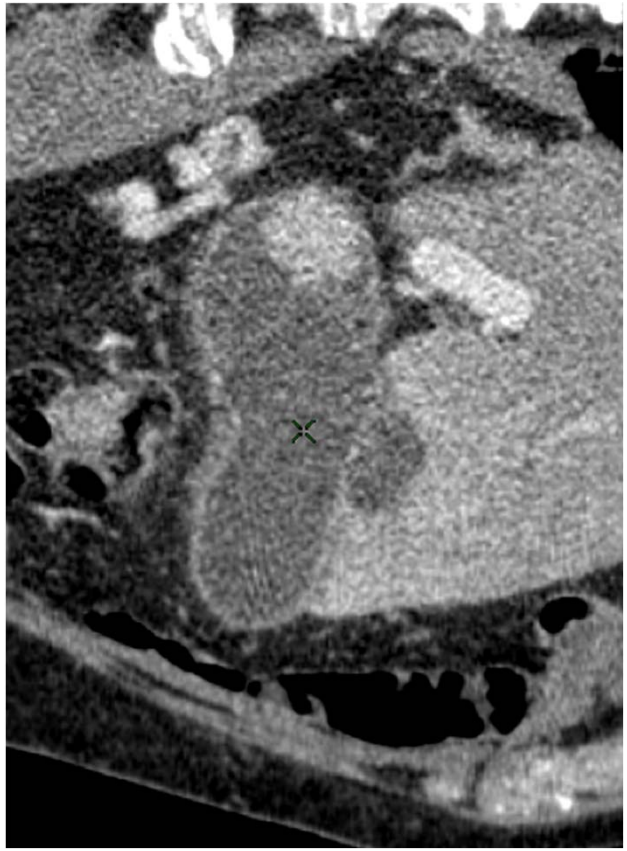
dure ,irrégulière radio opaque

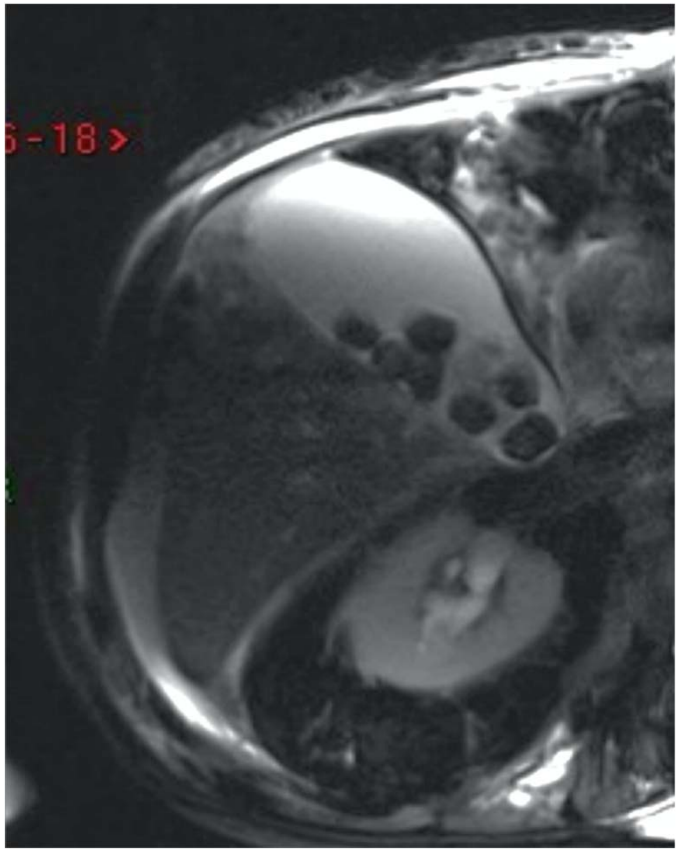
- brune rare

infection chronique des voies biliaires

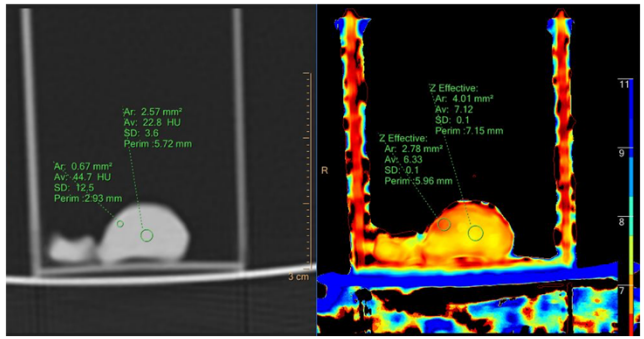
polymérisation de la bilirubine par B glycuronidase bactérienne

très friable , densité inter (parfois gaz / anaérobie)

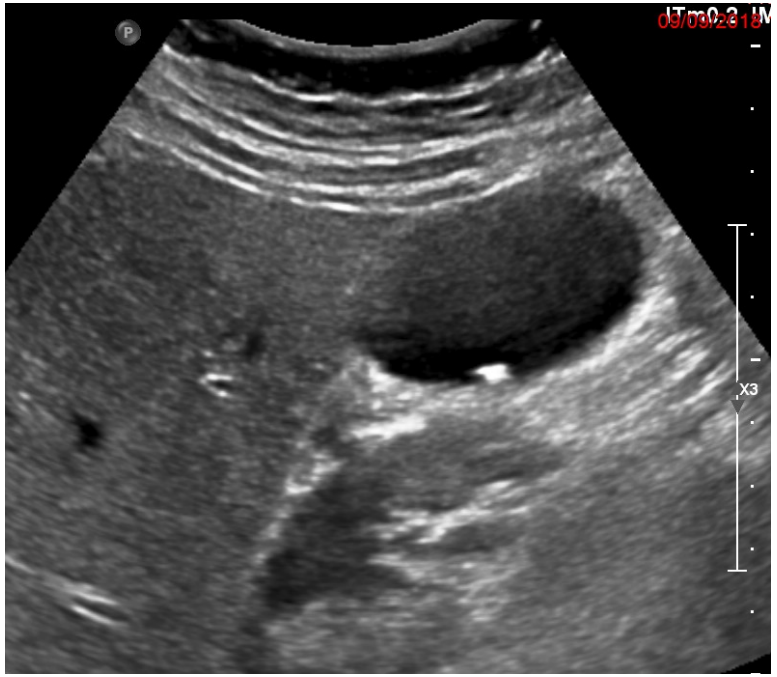


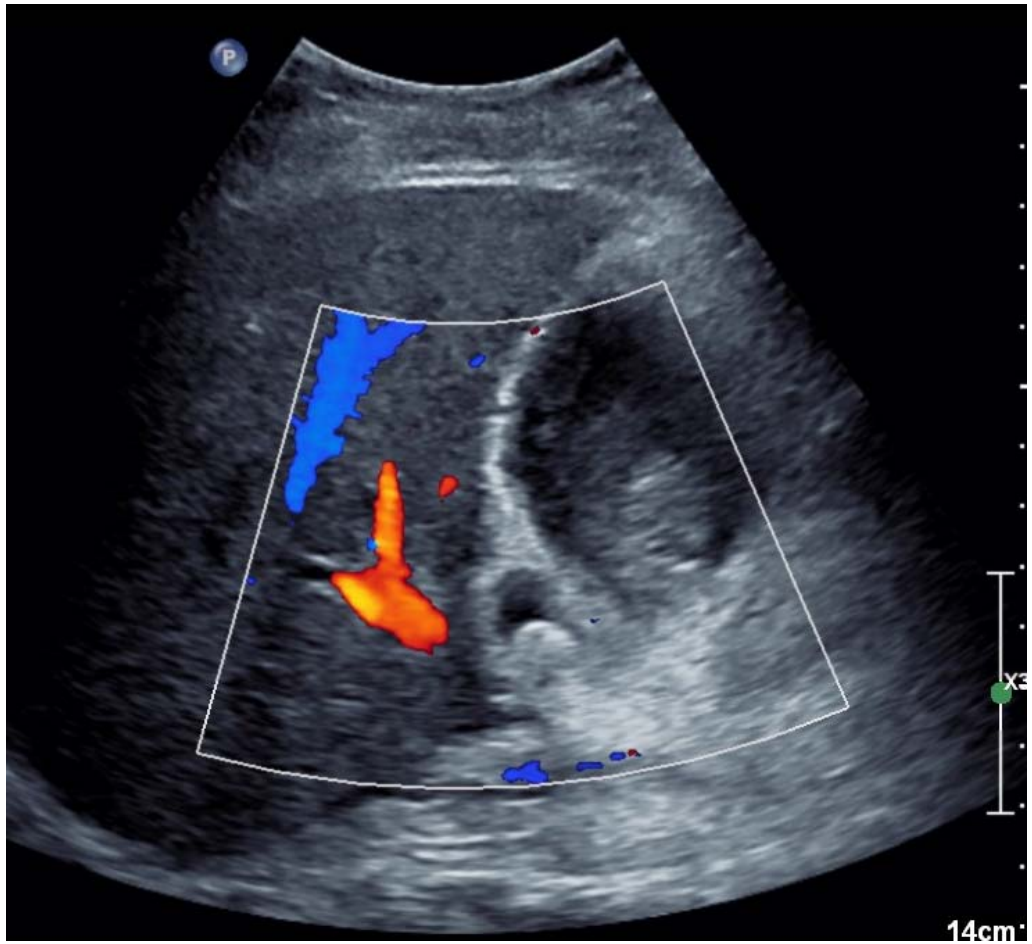

6-18>

Lithiase mixte



Microlithiase vs granulôme




P
14cm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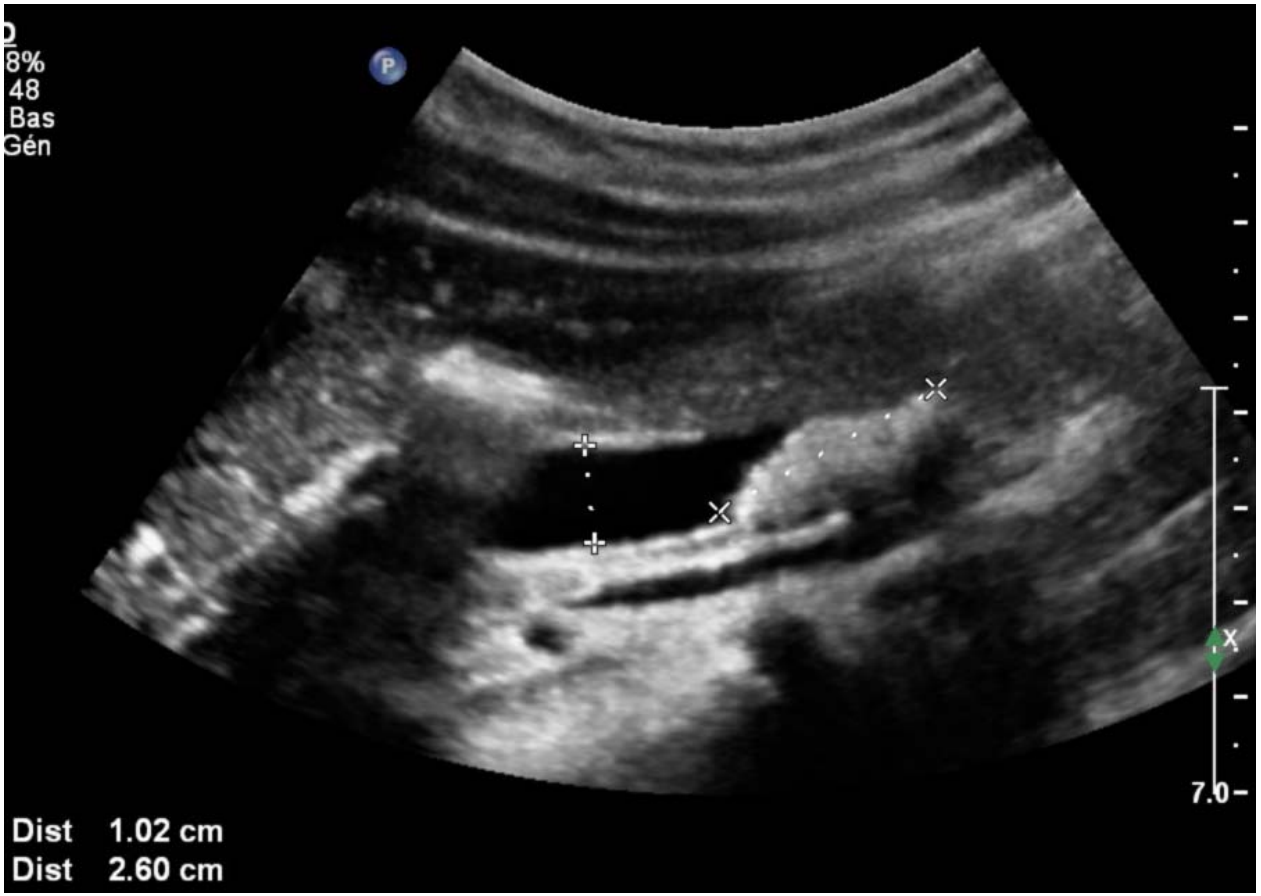
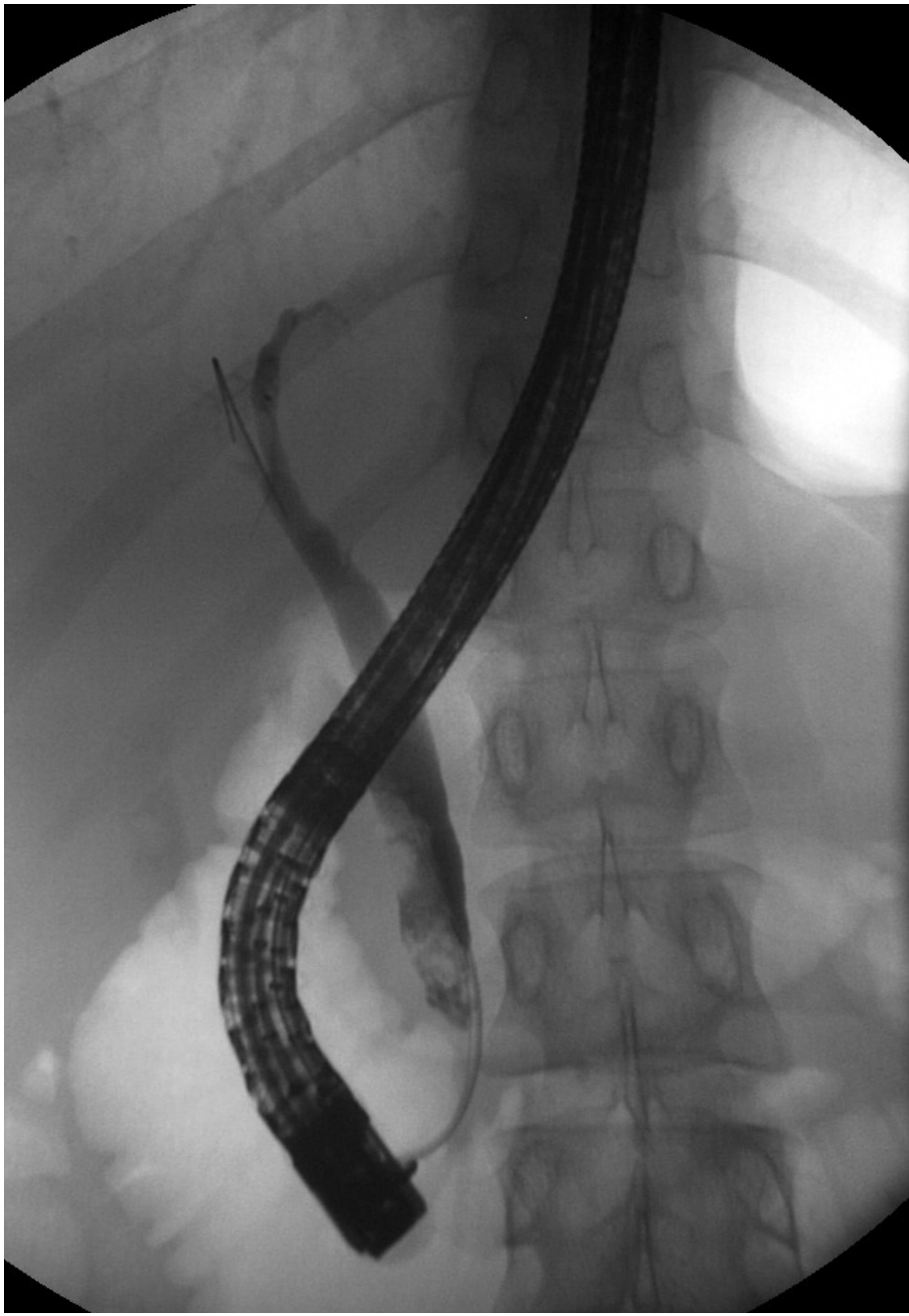
Lithiase des voies biliaires

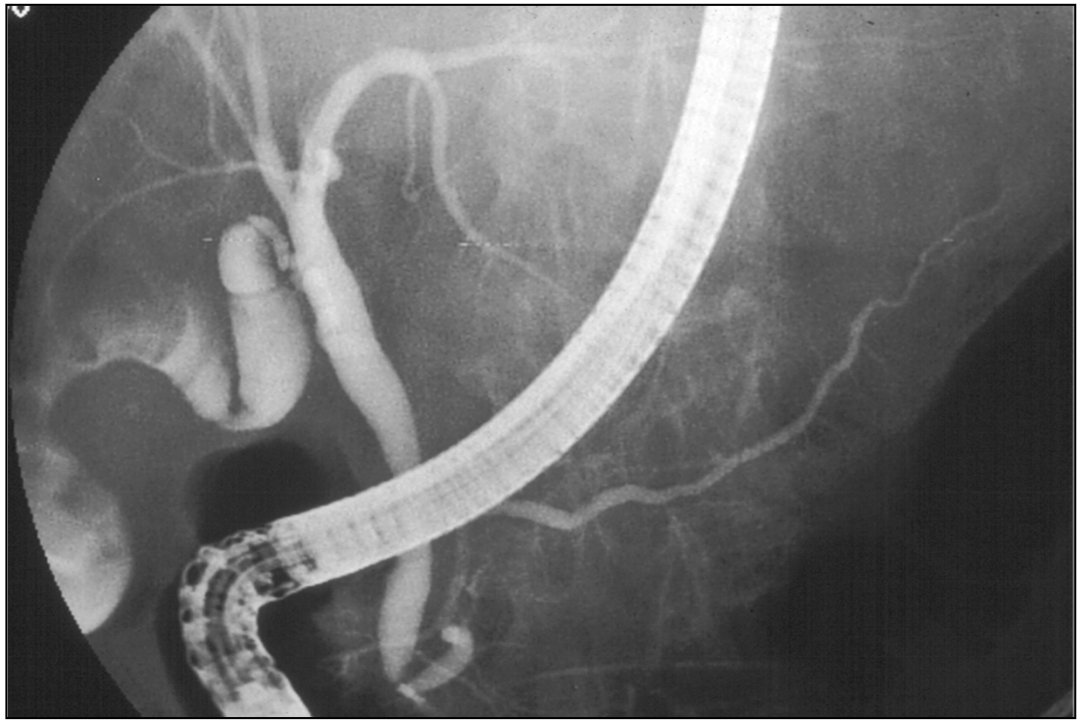
- Elle provient souvent de la migration d'un calcul vésiculaire
- Elle peut également se former dans les voies biliaires en étant favorisée par la stase et l'infection
- Elle peut provoquer un obstacle (ictère, angiocholite) ou une pancréatite aiguë

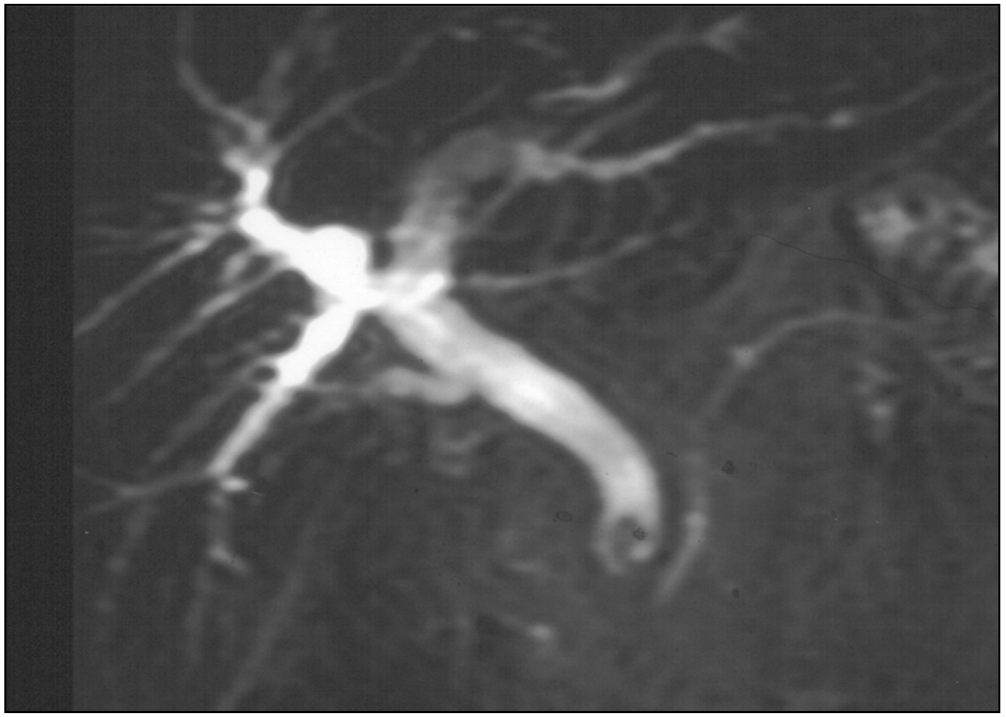

8%
48
Bas
Gén
7.0
Dist 1.02 cm
Dist 2.60 cm



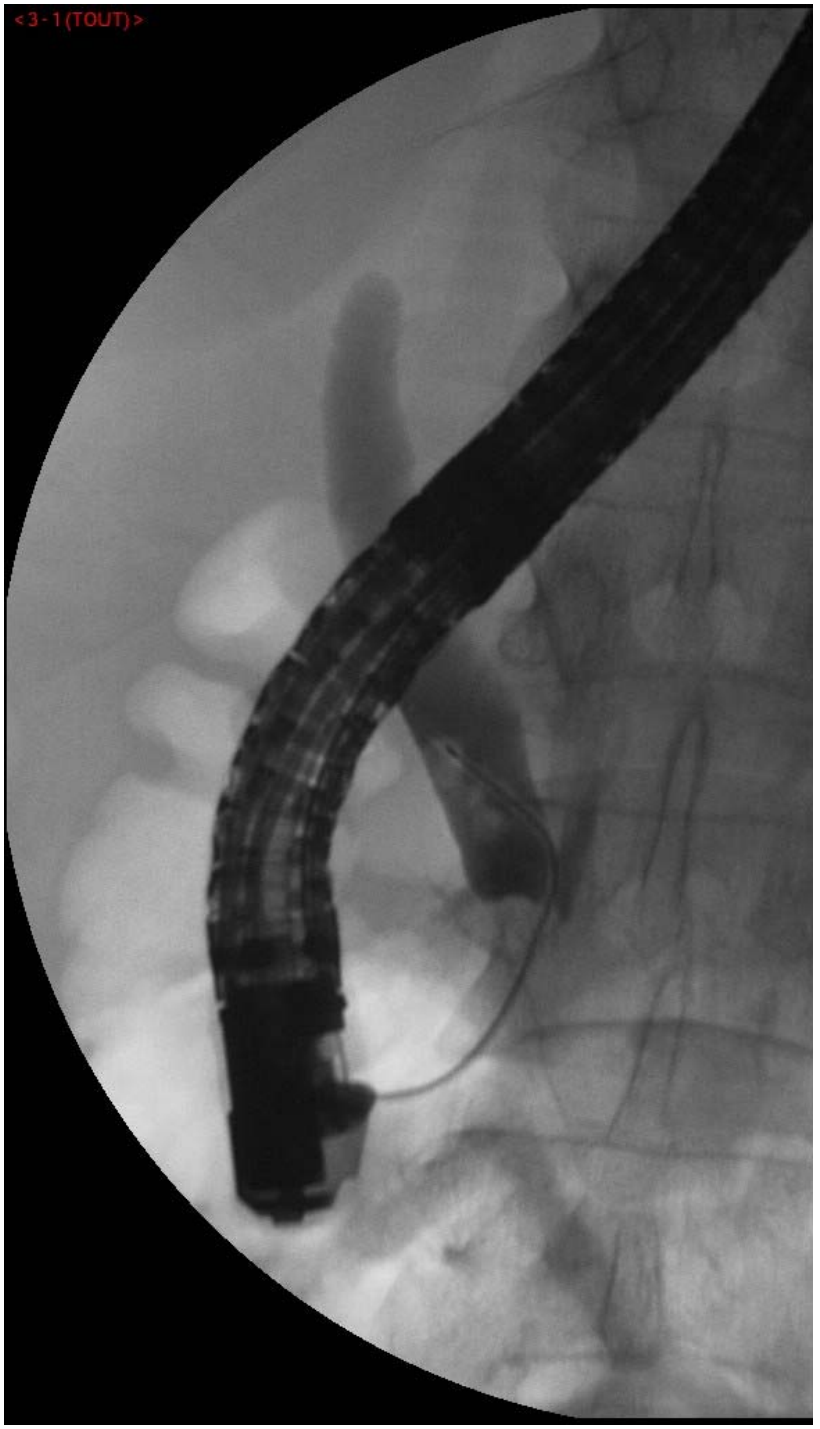
La lithiase des voies biliaires

- Echographie : sensibilité faible
- CholangioIRM : sensibilité élevée
- Echoendoscopie : lithiase du cholédoque






4C1-S #25
H4.5MHz 80mm
Abdomen
NTHI General
72dB S1/+1/3/ 3
Gain= 5dB Δ=2
Store in progress
Dist = 0.83cm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REVIEW

Low-Phospholipid Associated Cholelithiasis (LPAC) syndrome: A synthetic review

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Available online 26 March 2019

KEYWORDS
LPAC syndrome;
MDR3;
ABCB4;
Intrahepatic lithiasis;
Surgery;
Cholangiocarcinoma;
Liver transplant

Summary Low-Phospholipid Associated Cholelithiasis (LPAC) is a genetic disease responsible for the development of intrahepatic lithiasis. It is associated with a mutation of the ABCB4 gene which codes for protein MDR3, a biliary carrier. As a nosological entity, it is defined by presence of two of the three following criteria: age less than 40 years at onset of biliary symptoms, recurrence of biliary symptoms after cholecystectomy, and intrahepatic hyperechogenic foci detected by ultrasound. While the majority of clinical forms are simple, there also exist complicated forms, involving extended intrahepatic lithiasis and its consequences: lithiasis migration, acute cholangitis, intrahepatic abscess. Chronic evolution can lead to secondary sclerosing cholangitis or secondary biliary cirrhosis. In unusual cases, degeneration into cholangiocarcinoma may occur. Treatment is built around ursodeoxycholic acid, which yields dissolution of biliary calculi. Complicated forms may call for interventional, radiological, endoscopic or surgical treatment. This synthetic review illustrates and summarizes the different aspects of this entity, from simple gallbladder lithiasis to cholangiocarcinoma, as well as secondary biliary cirrhosis requiring liver transplant, on the basis of clinical cases and the iconography of patients treated in our ward.

Introduction

While the incidence of vesicular cholesterol cholelithiasis in the general population is elevated, only 10 to 25% of patients are symptomatic. In cases of familial, early or diffuse intrahepatic cholelithiasis (gallstones), genetic origin should be suspected. In 2001, Rosmorduc et al. described

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1878-7886/

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Low phospholipid-associated cholestasis and cholelithiasis

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Summary
Low phospholipid-associated cholestasis and cholelithiasis (LPAC) is a genetic disorder characterized by cholesterol gallbladder and intrahepatic stones. It is caused by a mutation of the gene *ABCB4*, which encodes the canalicular protein ABCB4/MDR3, a flippase that plays an essential role in the secretion of phosphatidylcholine into bile. Failure of this protein leads to secretion of bile that is poor in phospholipids and, hence, highly lithogenic, with potent detergent properties. This, in turn, leads to cholangiocyte luminal membrane injury and biliary lesions causing cholestasis. The diagnosis should be suspected when at least two of the following criteria are present: onset of symptoms before the age of 40 years; recurrence of biliary symptoms (biliary colic, jaundice, cholangitis, acute pancreatitis) after cholecystectomy; presence of echogenic foci within the liver indicative of intrahepatic stones or biliary sludge; previous episode(s) of intrahepatic cholestasis of pregnancy; and family history of gallstones in first-degree relatives. Intrahepatic stones can be demonstrated by ultrasonography with color Doppler examination, computed tomography and magnetic resonance imaging with magnetic resonance cholangiography, and the diagnosis confirmed by *ABCB4* genotyping. Therapy with ursodeoxycholic acid offers prompt relief of symptoms and usually prevents complications. In some cases, however, surgery may be necessary.

Abbreviations:
LPAC: Low phospholipid-associated cholestasis and cholelithiasis
UDCA: ursodeoxycholic acid

Introduction

Low phospholipid-associated cholestasis and cholelithiasis (LPAC; OMIM *171060) is a syndrome first described in 2001 by Rosmorduc, Hermelin and Poupon at the Saint-Antoine Hospital in Paris [1]. The disorder is caused by a mutation in the class III multidrug resistance/ATP-binding cassette, subfamily B, member 4 (*MDR3*/*ABCB4*) gene, which encodes the bile canalicular protein MDR3. MDR3 (now known as ABCB4) is a member of the superfamily of ABC proteins. It acts as a flippase, moving the phospholipid phosphatidylcholine from the inner leaflet of the canalicular membrane to the outer leaflet. From there, phosphatidylcholine is washed out into bile by bile acids. Thus, ABCB4 plays a crucial role in the transport of phosphatidylcholine into bile. Mutations of the gene lead to defective protein that is totally or partially unable to transport this major phospholipid into bile and, therefore, to the secretion of bile

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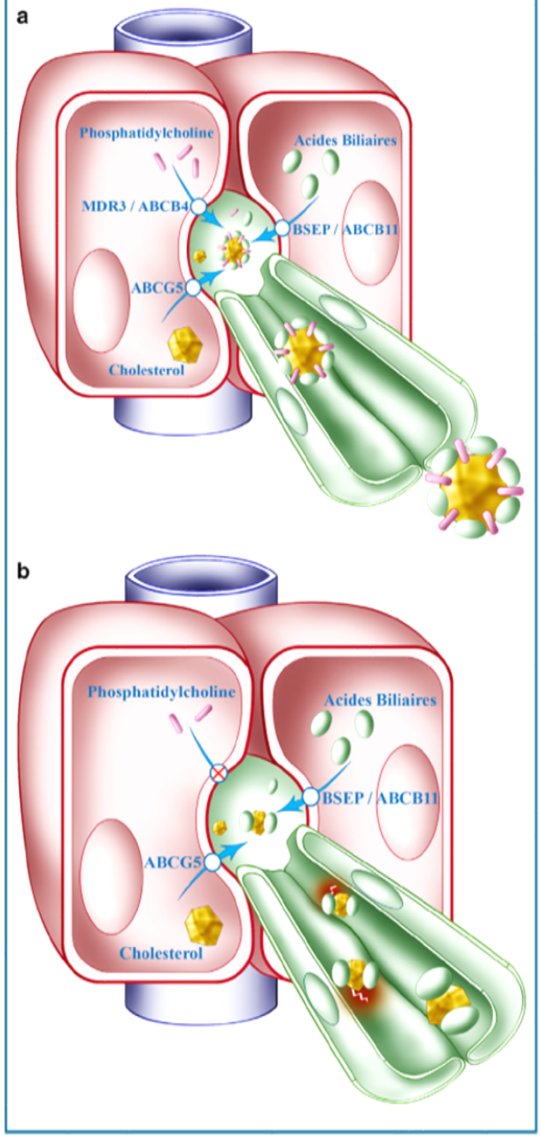


Figure 1. Pathophysiology of the LPAC syndrome; a: normal physiology: phosphatidylcholine is excreted by the MDR3 protein in the bile canaliculi at the level of the biliary hepatocyte pole. The micelles having been formed are mixed and stable, and cholesterol is solubilized in the bile; b: LPAC syndrome: The MDR3 protein (MultiDrug Resistance 3) is absent or deficient. In the absence of phosphatidylcholine, the micelles become simple, unstable and less able to solubilize cholesterol. The cholesterol precipitates and forms calculi. Along with lack of the protective effect of phosphatidylcholine, the presence of micro-crystals leads to chronic attacks on the cholangiocytes. The hepatocytes are designated in red, and the cholangiocytes in green. In the foreground in blue, a central lobular venule appears.

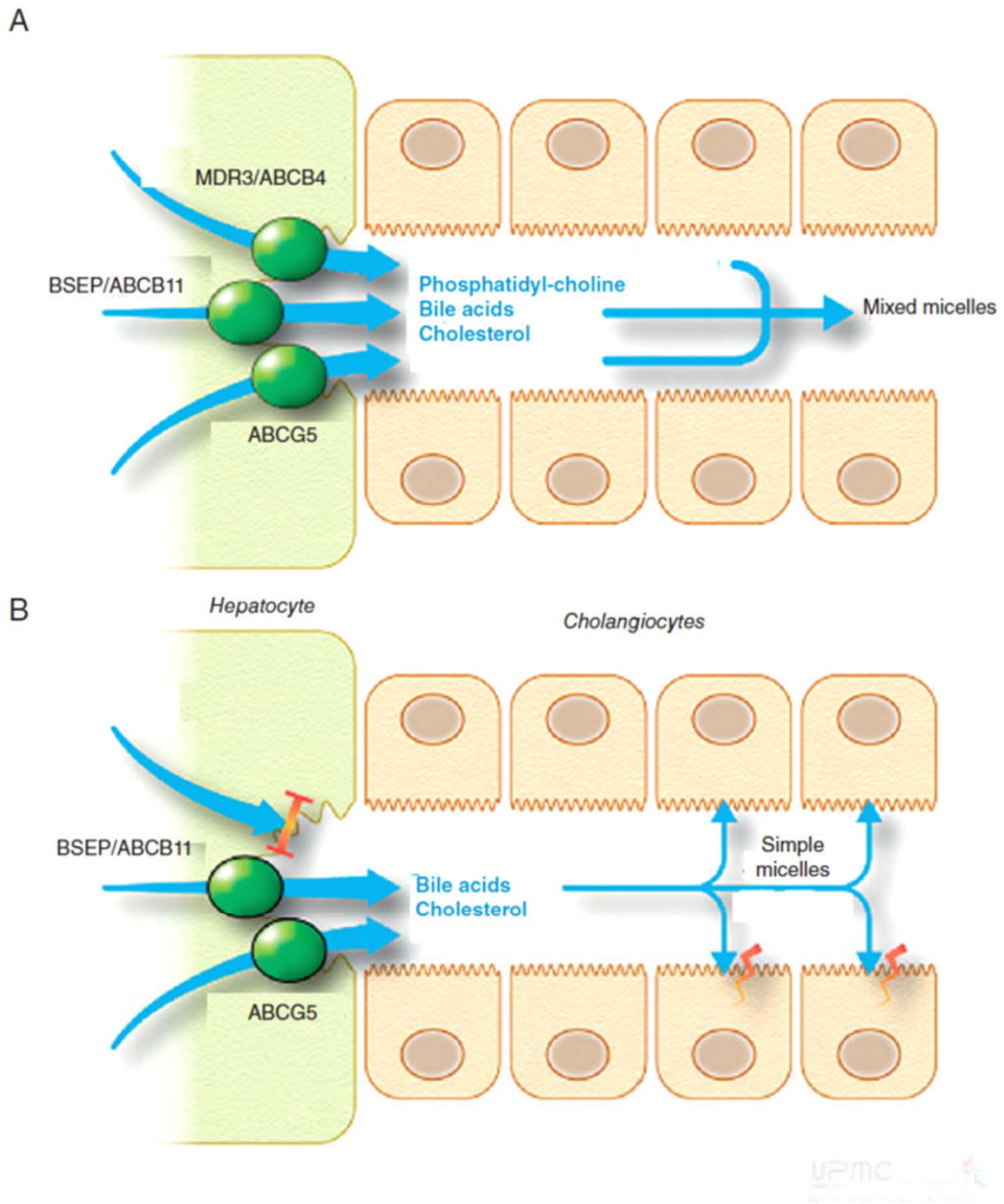
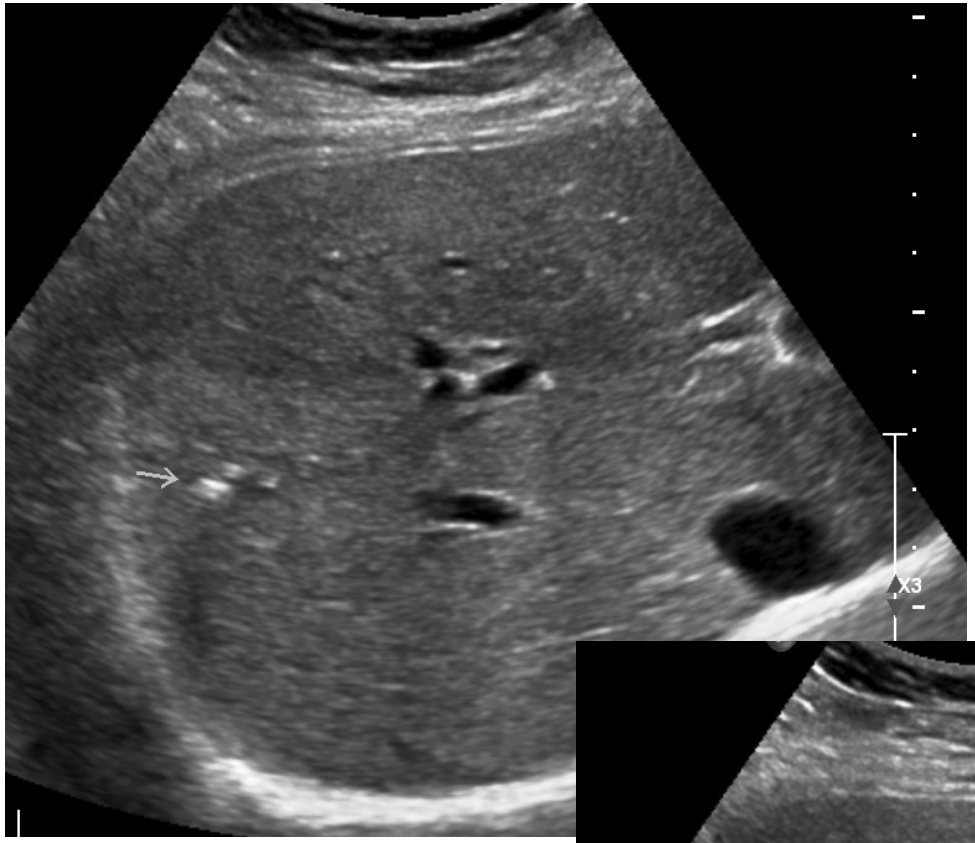
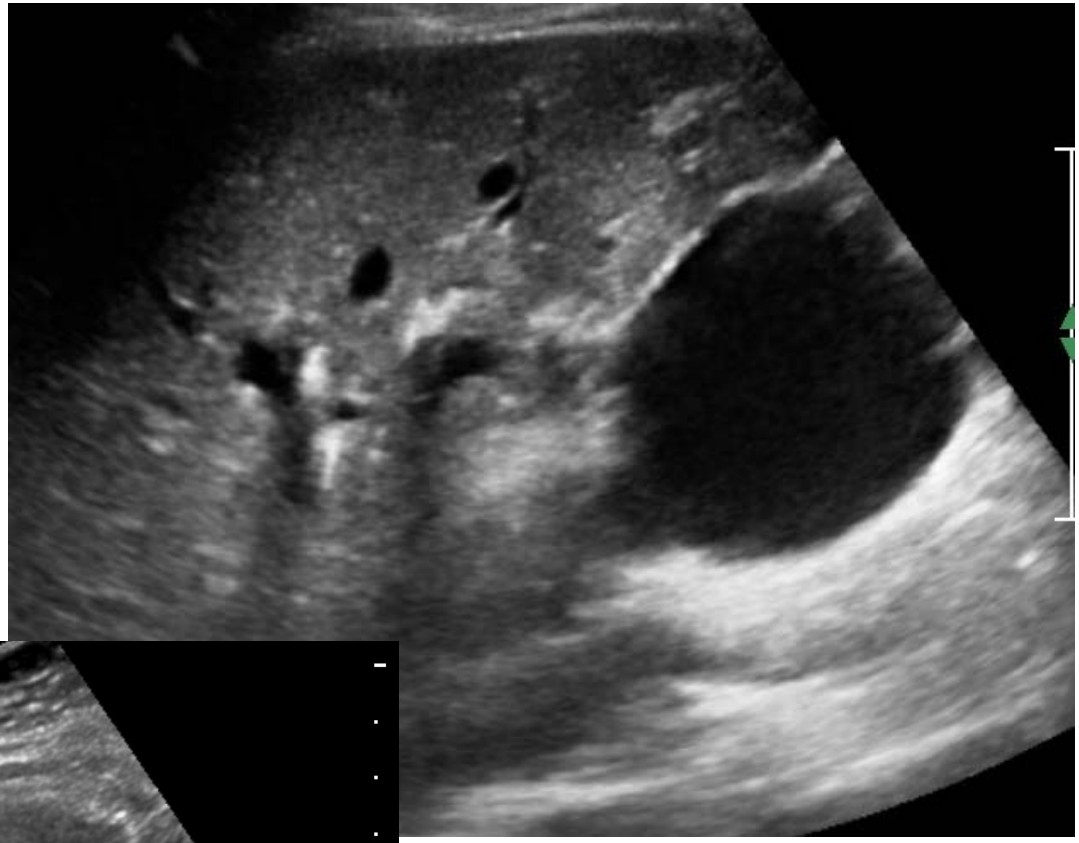


Figure 4 (A) Bile acid, cholesterol and phosphatidylcholine transport through the canalicular membrane in normal hepatocytes. (B) When MDR3/ABCB4 is defective, bile acids are transported without phospholipids. The bile acids then form simple micelles with potent detergent activity that can damage the neighboring cholangiocytes.


P
X3
12cm


P
X3
10cm


X3




X3

	Lithiase de la vésicule biliaire classique	**Lithiase dans le contexte du syndrome LPAC**
Âge au début des premiers symptômes	Après 50 ans	Avant 30 ans
Morphotype	Association avec un excès de poids, l'obésité	Poids normal
Sexe	Sex-ratio 1,5 femme / 1 homme	Sex-ratio 3 femmes / 1 homme
Imagerie	Lithiase isolée de la vésicule biliaire	Lithiase intrahépatique associée
Histoire de famille	-	Lithiase familiale au 1er degré, symptomatique avant 40 ans
Histoire personnelle	-	Cholestase gestationnelle
Cholécystite	Fréquent	Rare
Complications de la lithiase	Rare	Fréquent (migration, cholangite aiguë, pancréatite aiguë, etc.)
Récurrence des symptômes après cholécystectomie	Rare	Très fréquent (par définition)

Vésicule biliaire : tumeurs & pseudotumeurs

- Granulome à cholestérine (< 6 mm)
  - Unique
  - Multiples (cholestérolose, vésicule fraise)
- Adénomyose
  - Hyperplasie de la muqueuse et de la musculeuse
    - Epaississement pariétal (localisé, diffus)
    - Artéfacts en queue de comète
- Tumeur
  - Polype (> 6 mm)
  - Adénocarcinome
    - Polype > 10 mm ou épaississement pariétal focal
    - Si découverte tardive : masse envahissant foie et voies biliaires

Autres anomalies pariétales

granulome < 6 mm
6 / 10 mm pseudo polype
vrai polype
> 10 mm adénocarcinome ?

Granulomes et polypes

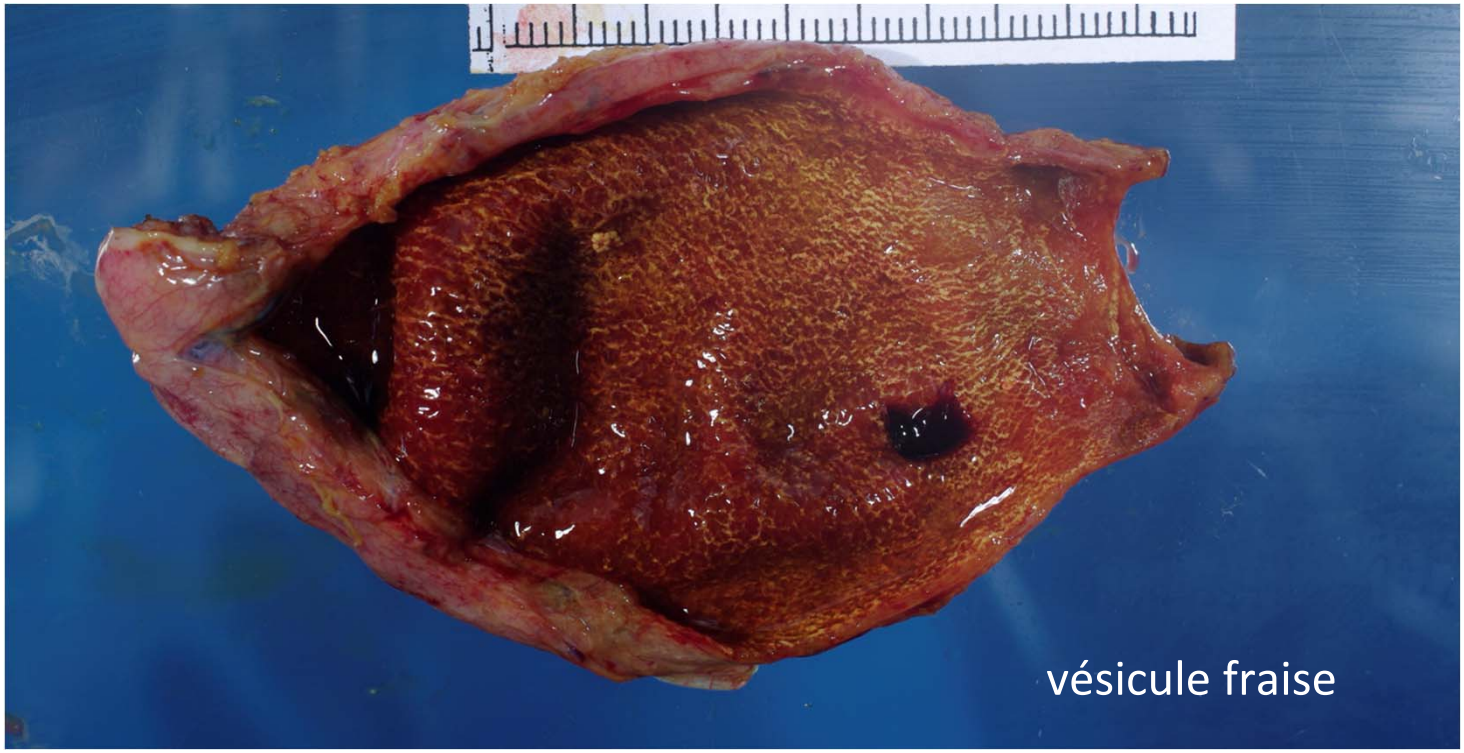
cholestérolose

- forme diffuse : ensemble de la paroi

=> granulations hyperéchogènes multiples non mobiles sans cône d'ombre

- forme focale : « polype » cholestérolique

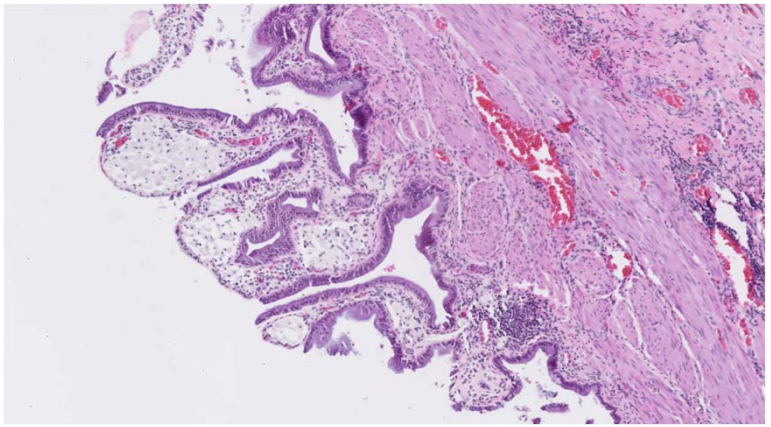
cholestérolose

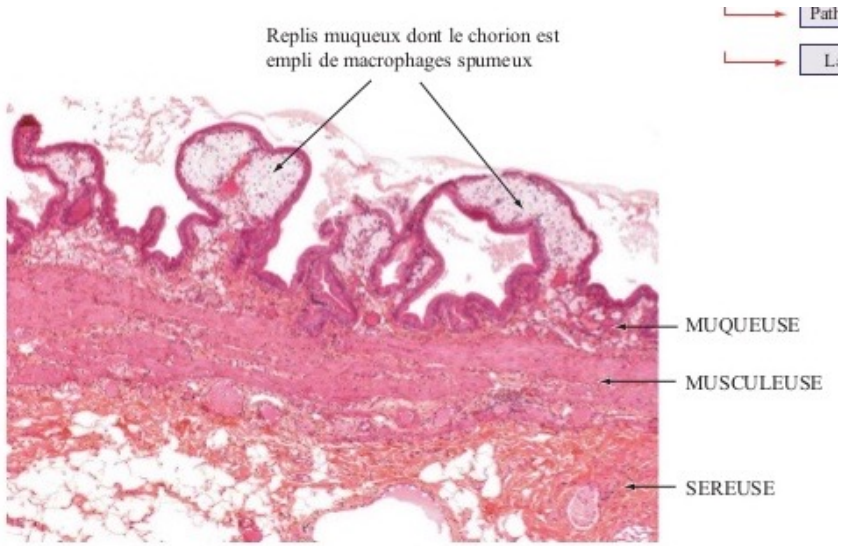


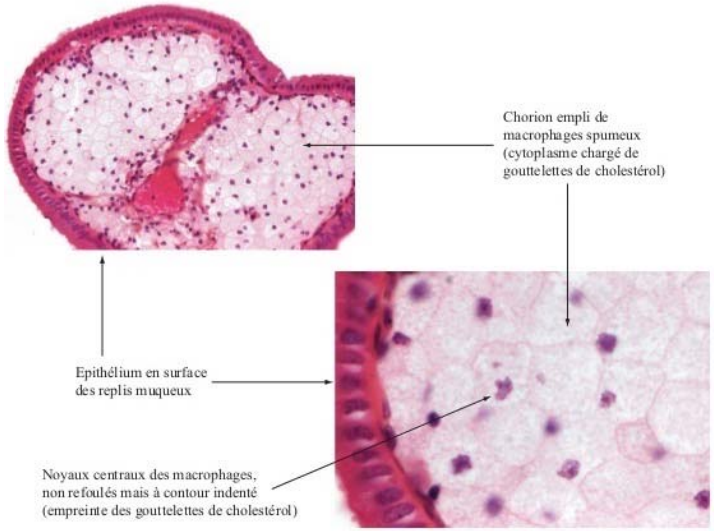
cholestérolose

hyperplasie de la muqueuse avec accumulation de dépôts cholestéroliques dans les macrophages du chorion

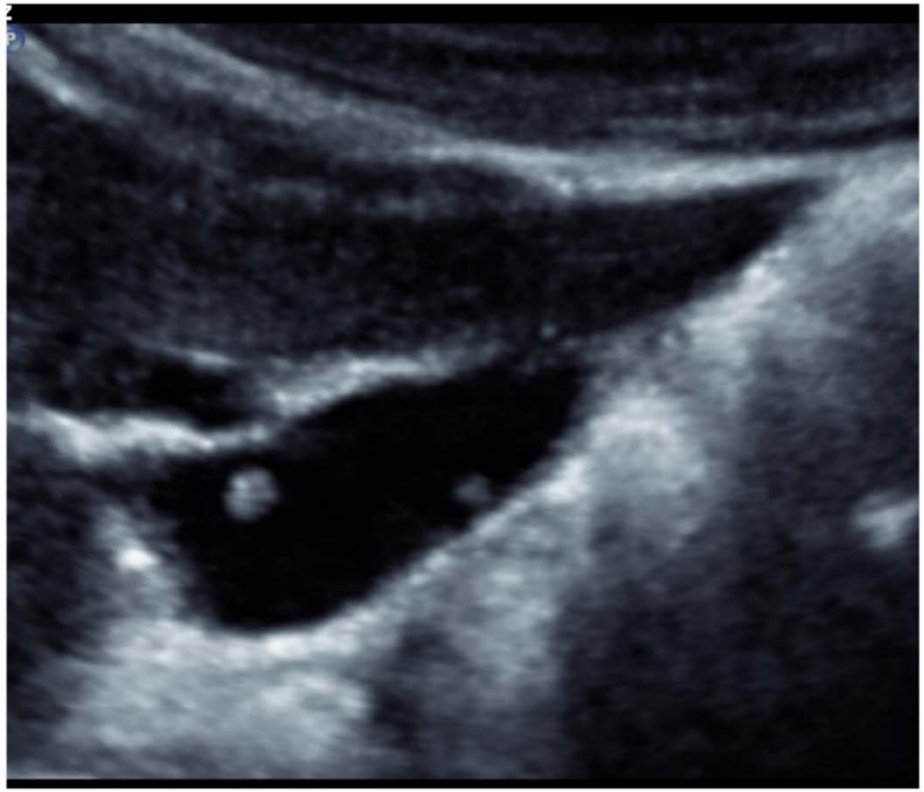
asymptomatique





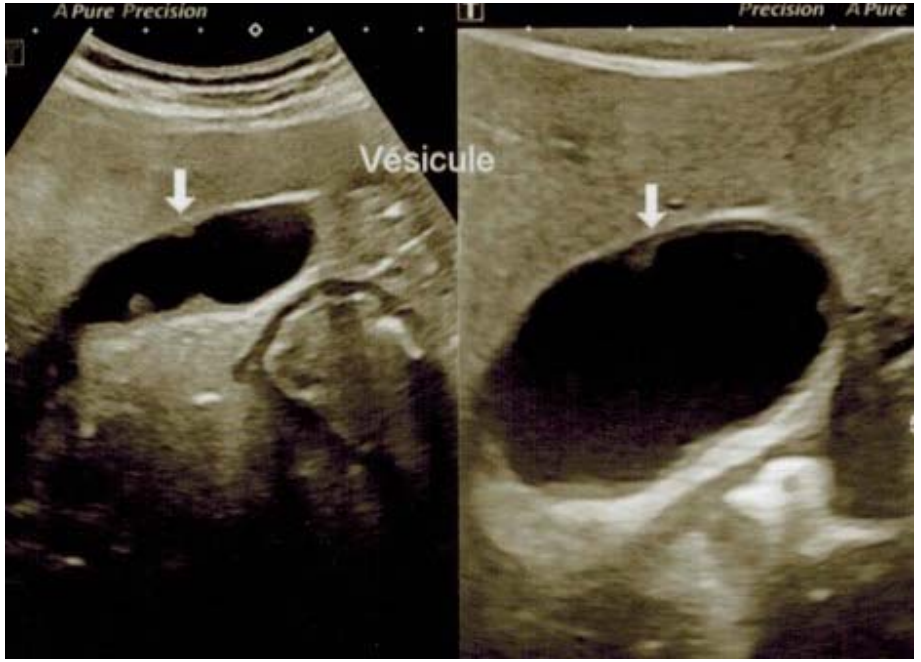


cholestérolose



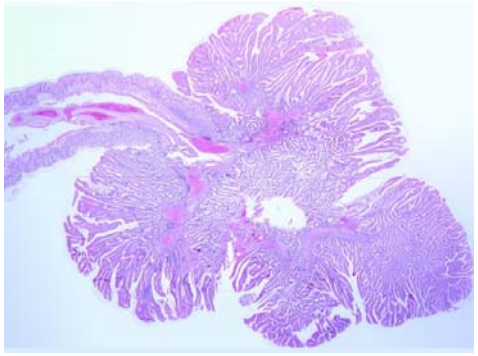
Multiples formations nodulaires appendues aux parois de la vésicule : granulomes

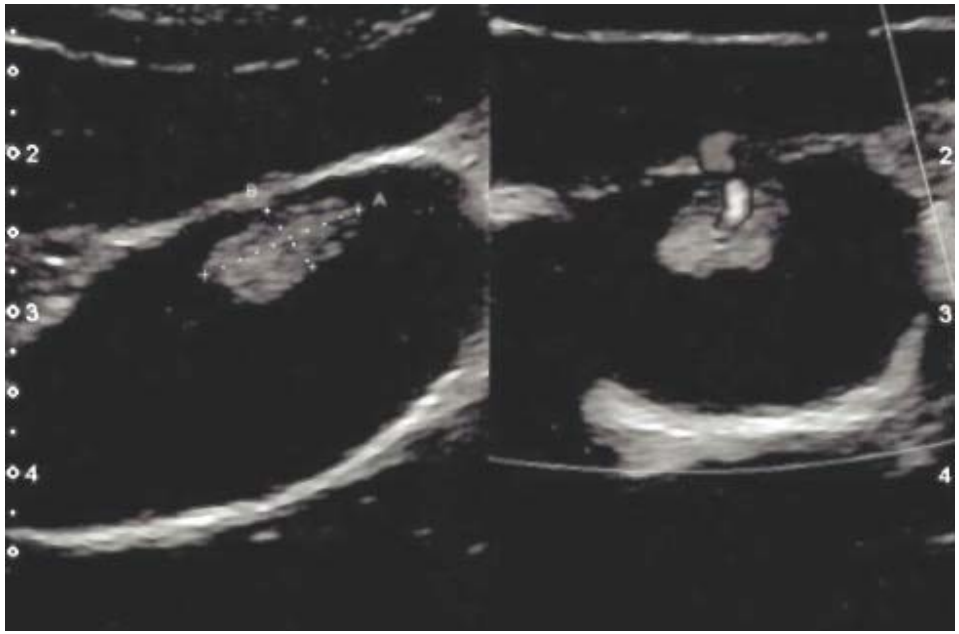
cholestérolose



- « polype »cholestérolique = granulome
formation échogène arrondie petite taille < 6 mm
pas de cône d'ombre rattachée à la paroi
D D parfois difficile avec le polype adénomateux

polypes /adénome

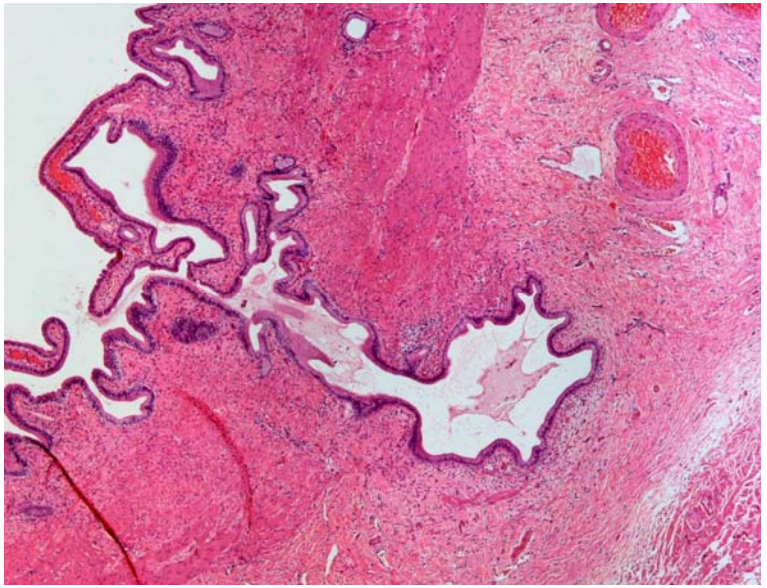




tumeur épithéliale bénigne

moins hyperéchogène voir isoéchogène au foie
souvent unique , plus grande taille >= 6 à 20 mm
signal Doppler
potentiel malin

adénomyomatose



- processus inflam chronique
- prolifération de l'épithélium avec invagination de la muqueuse jusque ds la musculeuse

(sinus de Rokitansky Aschoff)

pas de dégénérescence , asymptomatique

Adenomyomatosis

Prevalence: 3-5% (W/M: 3/1)
Mecanism: wall hyperplasia
Wall: > 10mm (diffuse ou focal)
Etiology: ?
Asymptomatic.
No R/

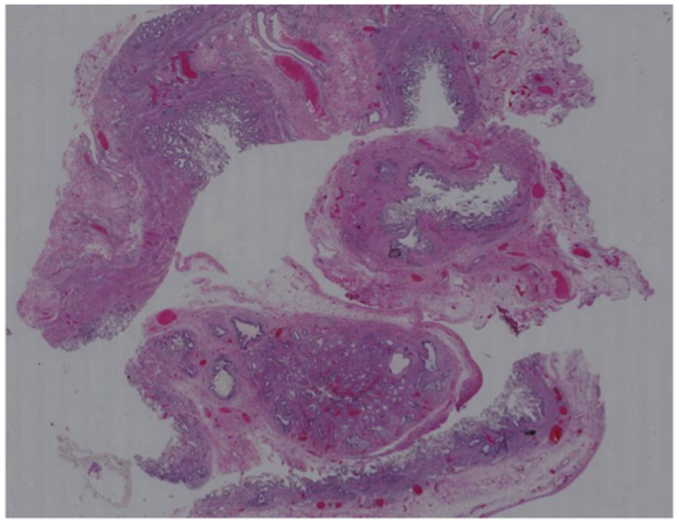
Lin *et al. BMC Gastroenterology* 2011, **11**:106
http://www.biomedcentral.com/1471-230X/11/106

BMC Gastroenterology

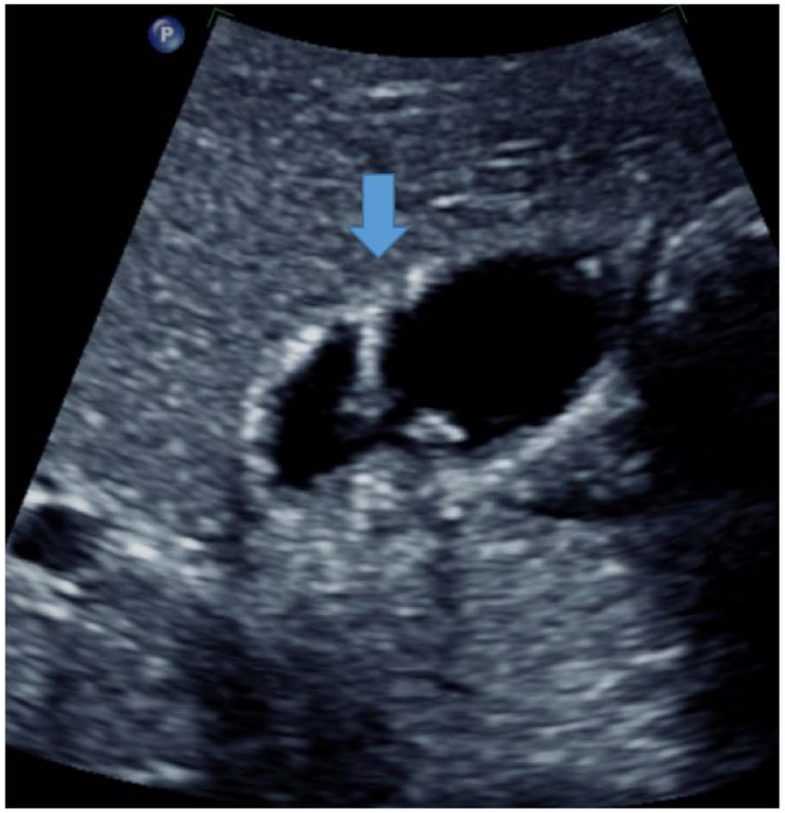
CASE REPORT Open Access

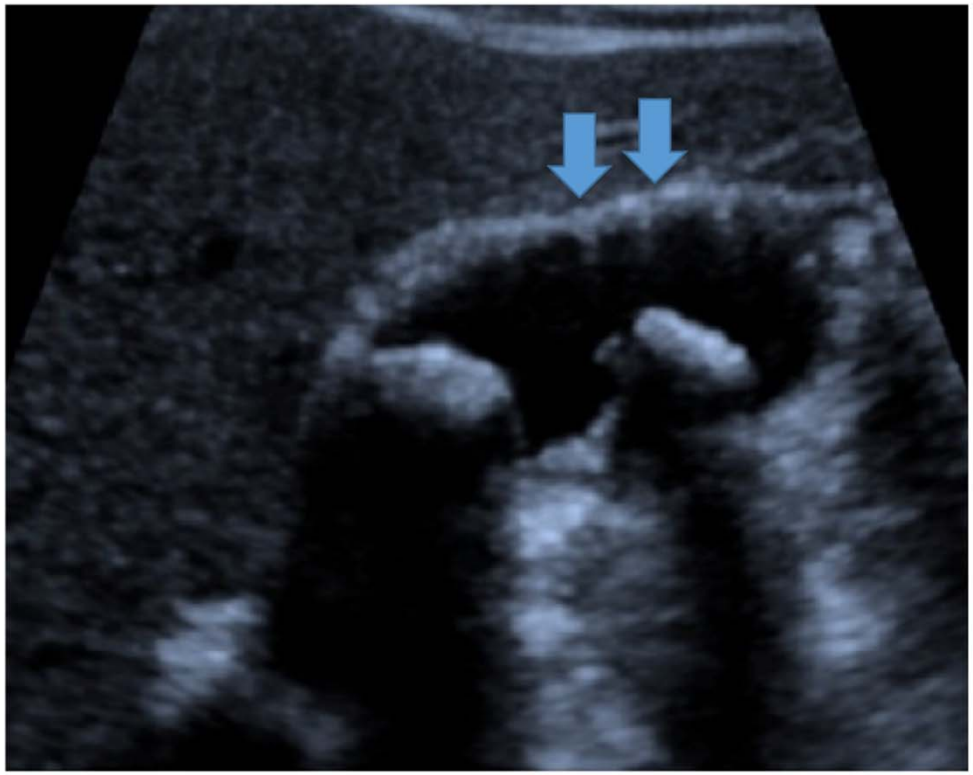
Rare gallbladder adenomyomatosis presenting as atypical cholecystitis: case report

Sheng-Hong Lin[1†], Feng-Yee Chang[2†], Ya-Sung Yang[2†], Jong-Shiaw Jin[3†] and Teng-Wei Chen[4*]



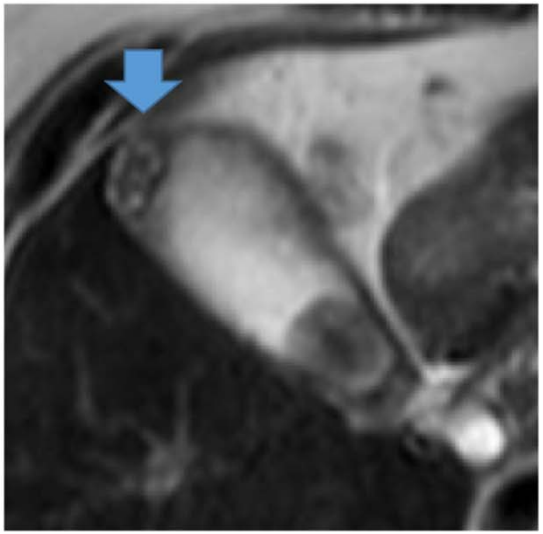
adénomyomatose

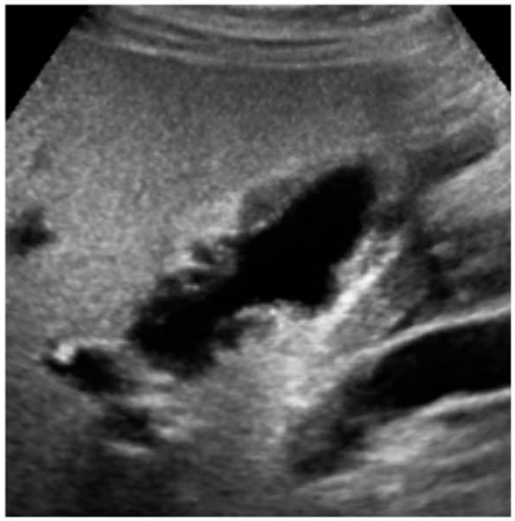


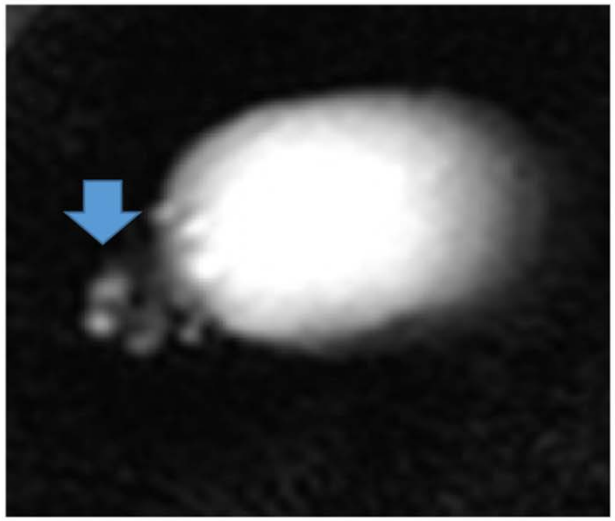


Adénomyose : artéfacts en queue de comète (flèche) associés ou non avec des calculs et/ou du sludge

adénomyose







Adénomyose de la paroi vésiculaire sous la forme d'un épaississement segmentaire en échographie et combiné à des petites expansions liquidiennes dans la paroi vésiculaire, mieux vues en IRM (flèches)

Accéder au document Medica
Réf. image Medica : med100000x1930xartorigx0163

Fig. 1. Vésicule biliaire ouverte longitudinalement et montrant de nombreux calculs intra-muraux sous l'aspect de taches verdâtres / Fig. 2. Coupe transversale de la vésicule précédente avec deux calculs intra-muraux / Fig. 3. Un calcul intra-mural, riche en pigments, desquammation partielle de l'épithélium diverticulaire / Fig. 4. Coupe de vésicule biliaire montrant deux canaux de Luschka sensiblement normaux. Amas lymphoïde inflammatoire près du diverticule gauche / Fig. 5. Calcul intra-mural visiblement développé dans un diverticule de Luschka. Sclérose et réaction inflammatoire péridiverticulaire - La Presse médicale - [Articles originaux]

20e siècle	
Revue :	La Presse médicale - [Articles originaux], 1930, Articles originaux
Edition :	Masson et Cie, 1930
Cote :	100000x1930xartorig

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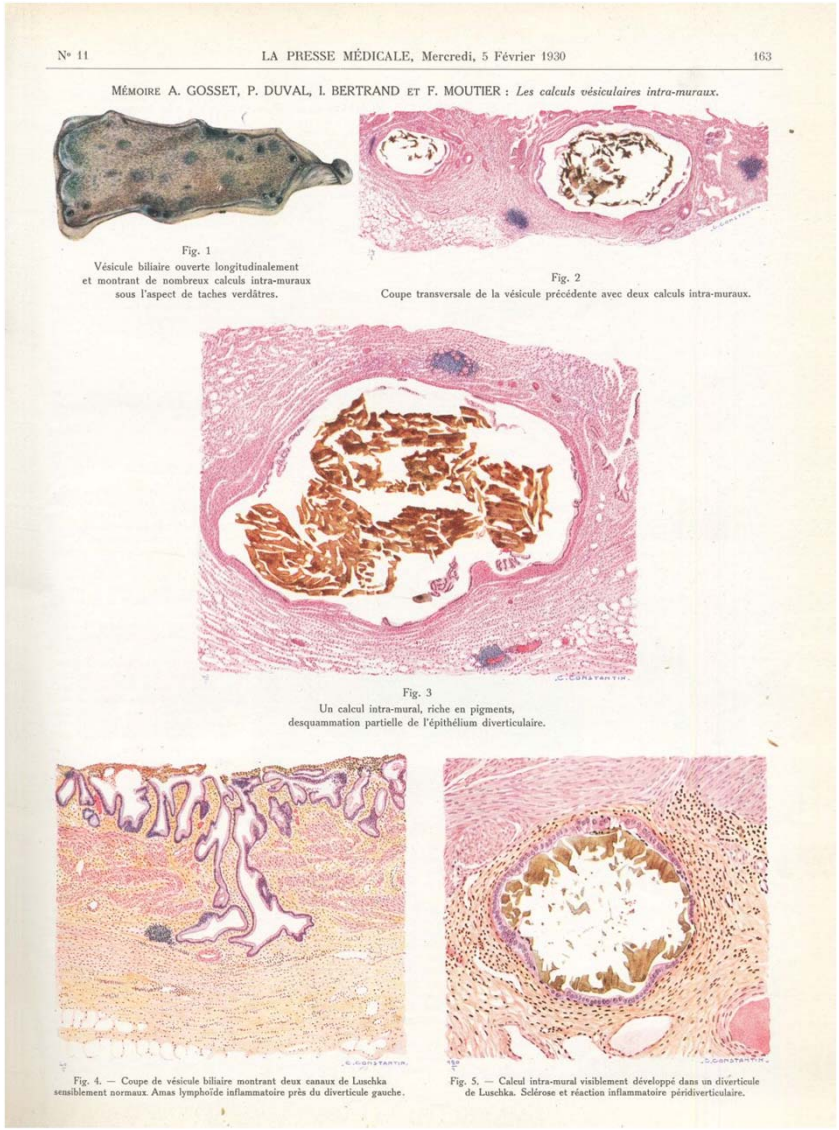


MÉMOIRE A. GOSSET, P. DUVAL, I. BERTRAND ET F. MOUTIER : *Les calculs vésiculaires intra-muraux.*

Fig. 1
Vésicule biliaire ouverte longitudinalement et montrant de nombreux calculs intra-muraux sous l'aspect de taches verdâtres.

Fig. 2
Coupe transversale de la vésicule précédente avec deux calculs intra-muraux.

Fig. 3
Un calcul intra-mural, riche en pigments, desquammation partielle de l'épithélium diverticulaire.

Fig. 4. — Coupe de vésicule biliaire montrant deux canaux de Luschka sensiblement normaux. Amas lymphoïde inflammatoire près du diverticule gauche.

Fig. 5. — Calcul intra-mural visiblement développé dans un diverticule de Luschka. Sclérose et réaction inflammatoire péridiverticulaire.

Vésicule biliaire : cholécystite

- US
- CT
- IRM

CHOLECYSTITE

Pathological Classification
Y Kimura, J Hepatobiliary Pancr Sc 2013

Acute non complicated ch
- Edematous form

Complicated form ch
- Necrosing
- Suppurative
- Emphysematous

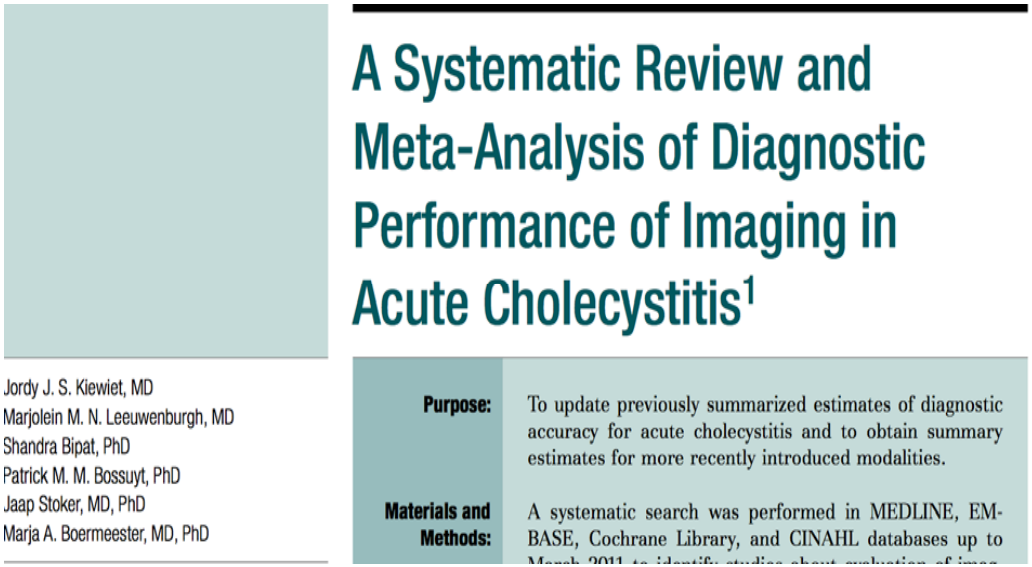
Chronic
- Chronic
- Xanthogranulomatous f

Clinico-Radiological Classification
E Smith1 AJR 2009

- Acute non complicated ch
- Acute complicated ch
  - Gangrenous => Perforation
  - emphysematous
  - Suppurative cholecystitis
  - Hemorrhagic
- Acalculous
- Chronic

Scope of the Disease

Prevalence = 5 %



A Systematic Review and Meta-Analysis of Diagnostic Performance of Imaging in Acute Cholecystitis[1]

Jordy J. S. Kiewiet, MD
Marjolein M. N. Leeuwenburgh, MD
Shandra Bipat, PhD
Patrick M. M. Bossuyt, PhD
Jaap Stoker, MD, PhD
Marja A. Boermeester, MD, PhD

Purpose: To update previously summarized estimates of diagnostic accuracy for acute cholecystitis and to obtain summary estimates for more recently introduced modalities.

Materials and Methods: A systematic search was performed in MEDLINE, EMBASE, Cochrane Library, and CINAHL databases up to

radiology.rsna.org ▪ ***Radiology:*** Volume 264: Number 3—September 2012

145 to 153 in 2013
at St-Luc University Hospital

900 beds
> 50.000 emergency visits/ year

US :

- **Signe de Murphy**

- **Majoration du diamètre transverse > 4 cm**

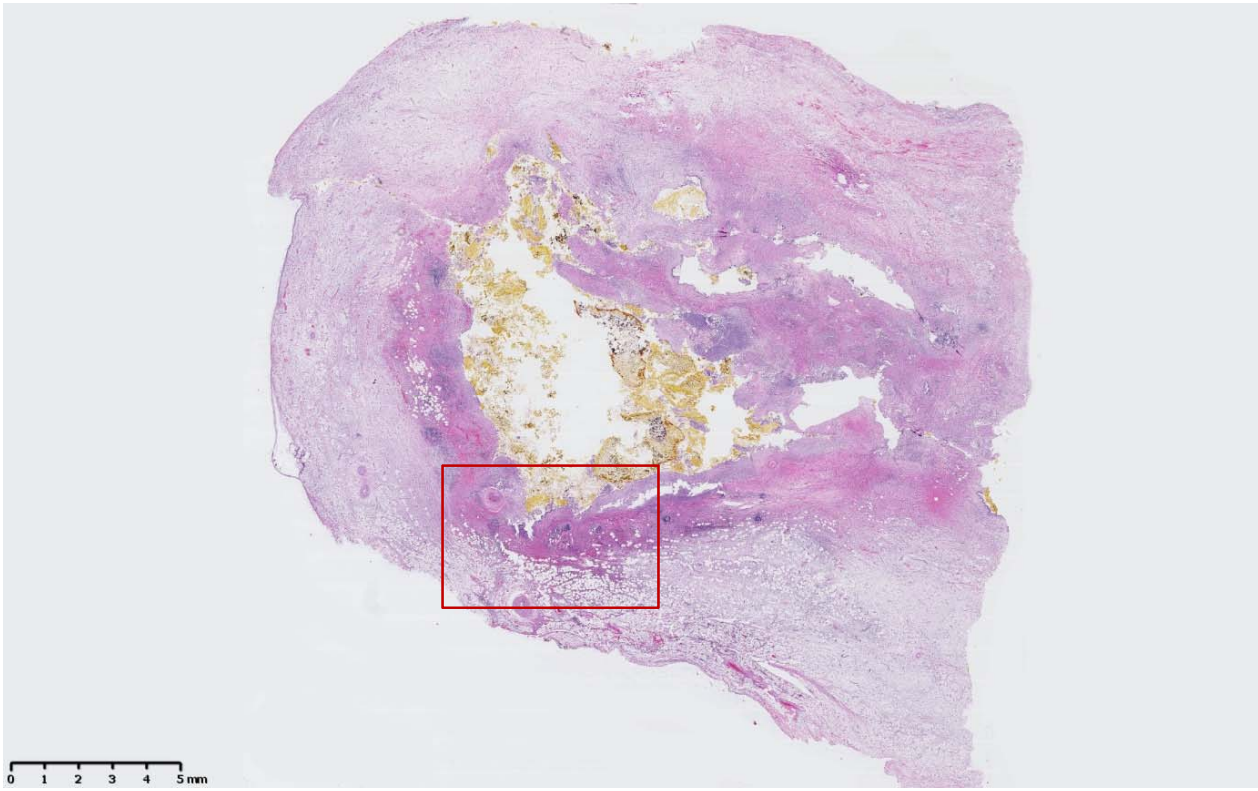
- **Calcul ou de sludge**

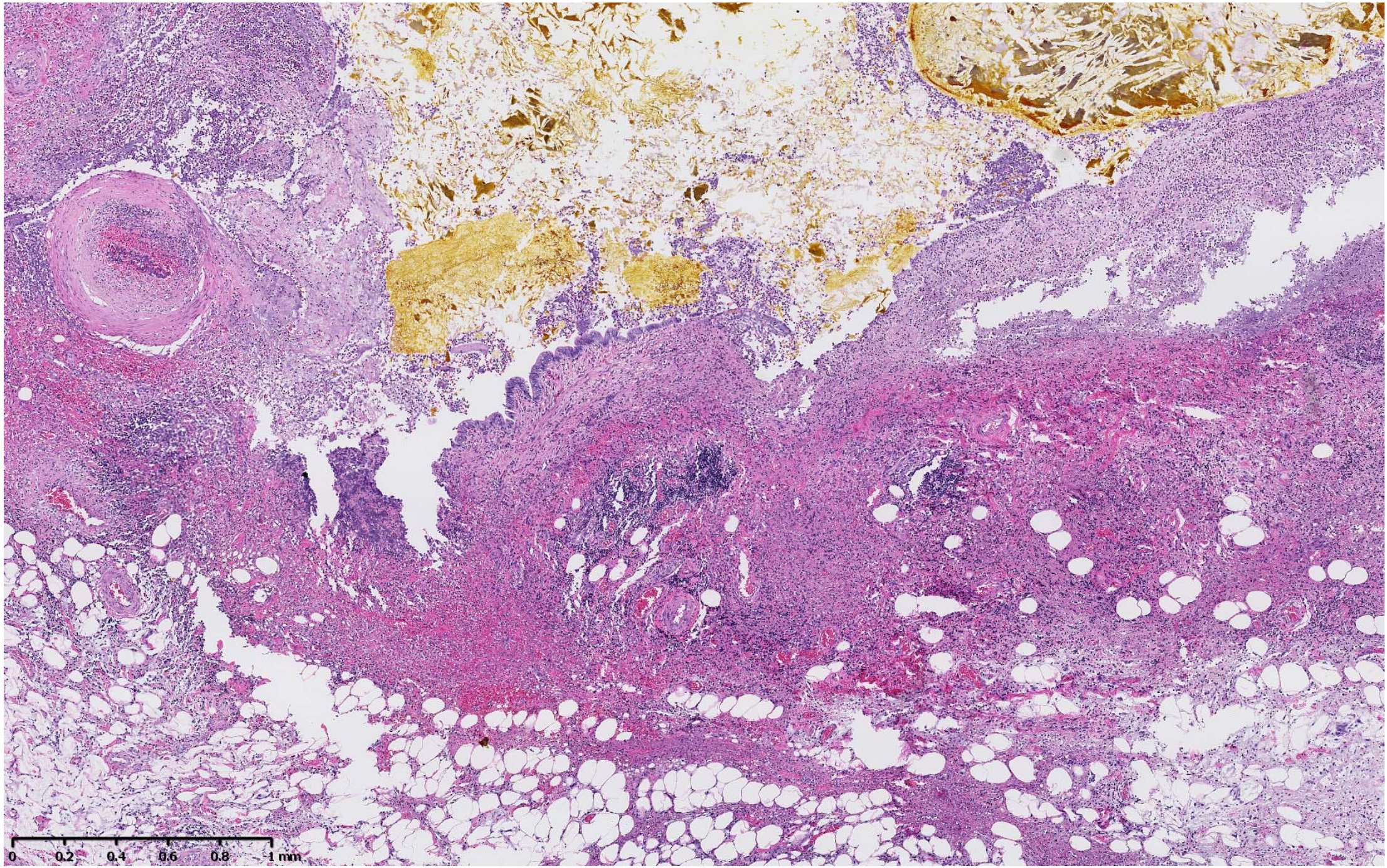
- **Paroi de la vésicule > 5 mm**

- **Uquide périvésiculaire**

Cholécystite Aiguë

Cholécystite Aiguë




0
0.2
0.4
0.6
0.8
1 mm

Cholécystite Gangréneuse

- Forme Sévère de cholécystite = > Altérations Ischémiques / nécrotiques
- Incidence : 2 à 30 %
- 10 à 40 % de cholécystites classiques se compliquent
- âgés, hommes, Diabète

Nikfarjam et al, HPB 2011, 13, 551–558

Cholécystite Gangréneuse

- hémorragie :

= > intraluminale

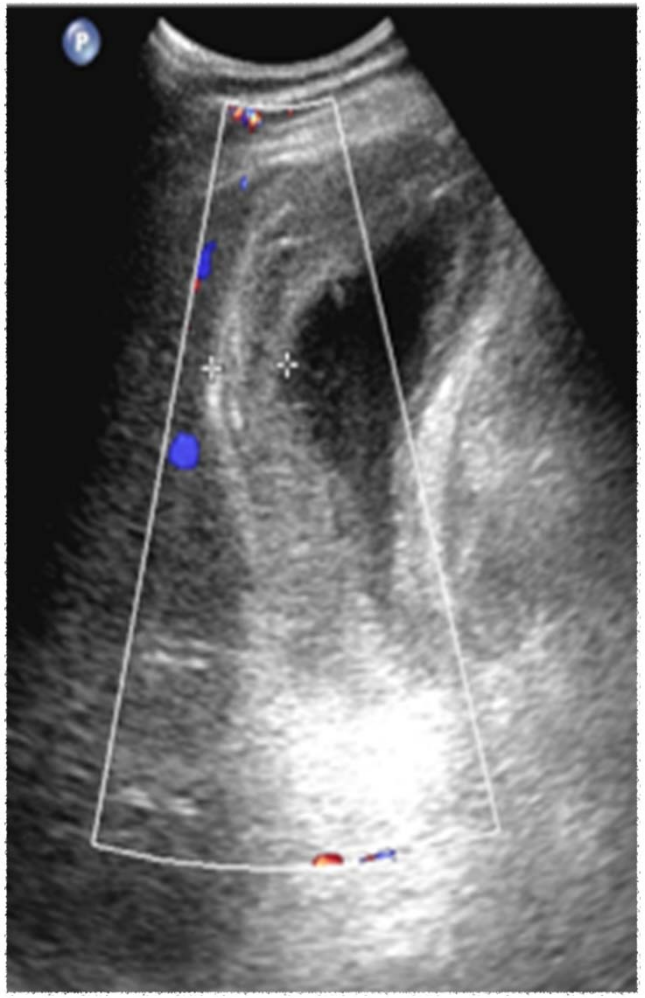
=> péritonéale

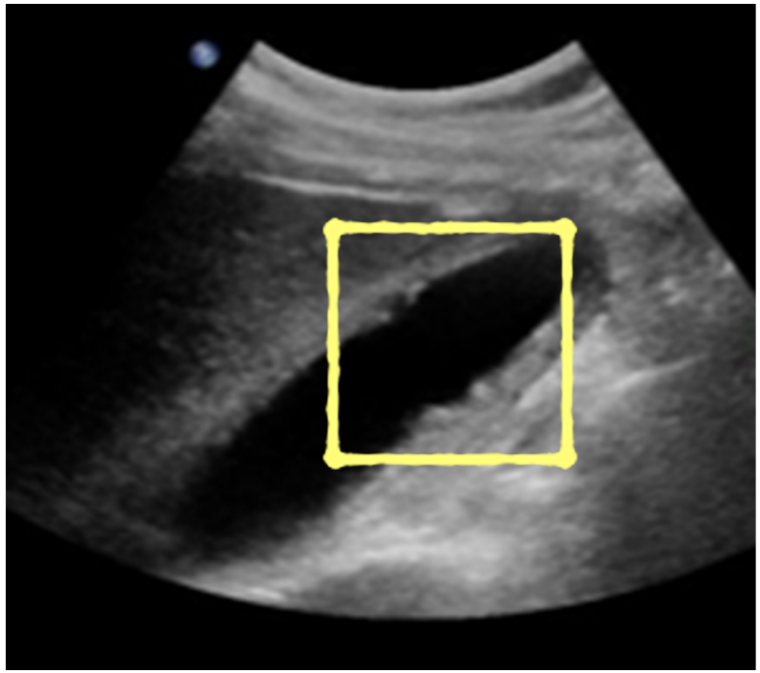
- perforation:

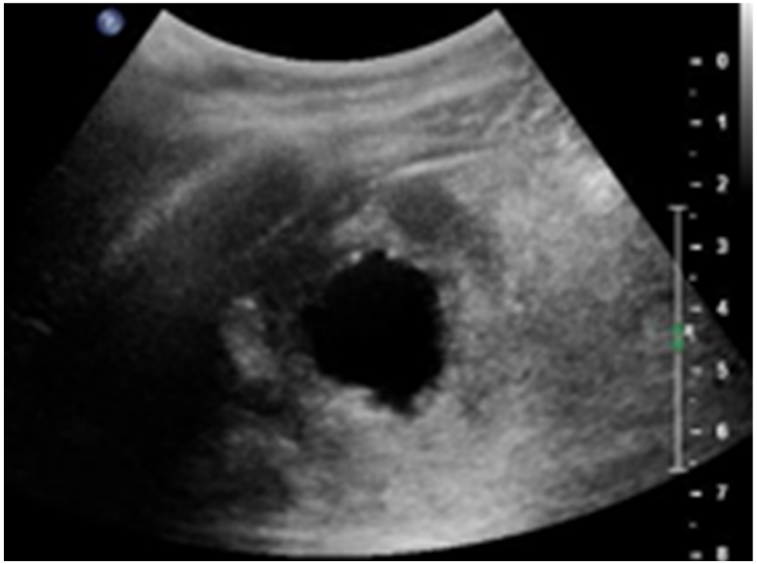
=> Dans le péritoine : péritonite biliaire aigue(10%)

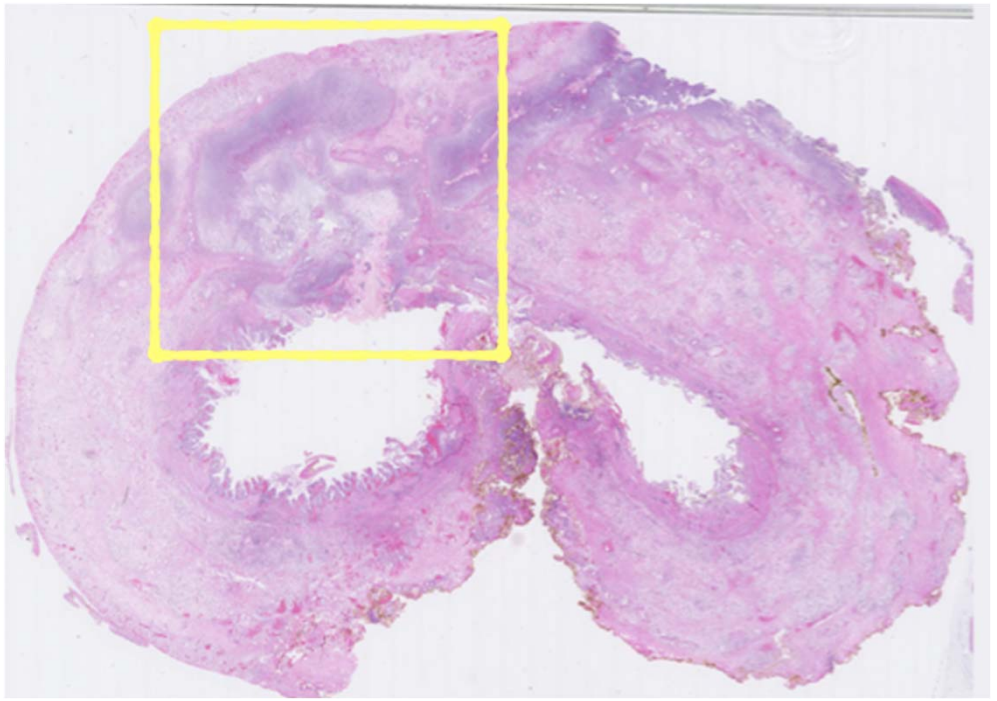
= > forme focale « couverte» : (subaiguë) abcès périvésiculaire (60%)

= > fistule bilio-enterique : iléus biliaire (30%)







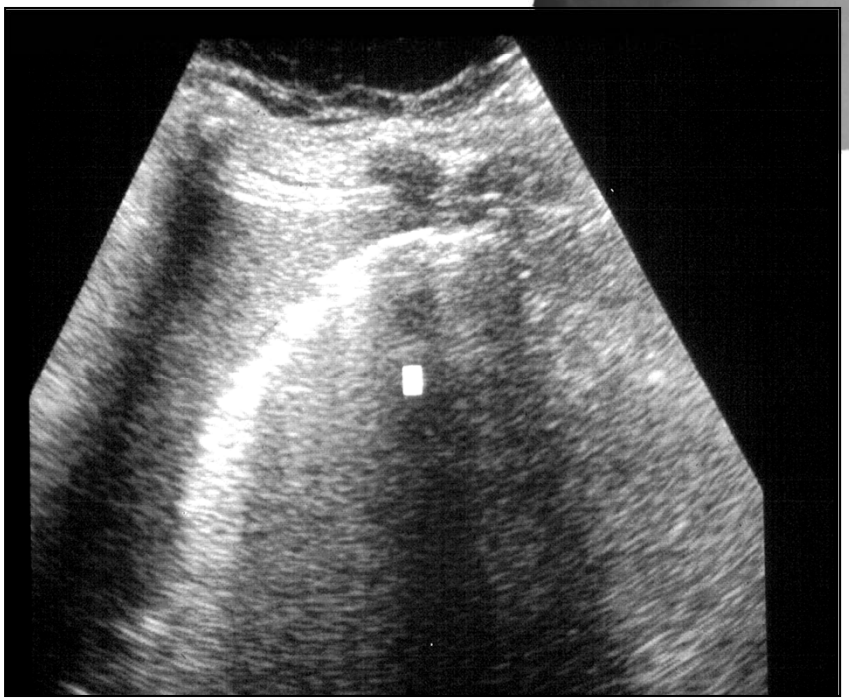


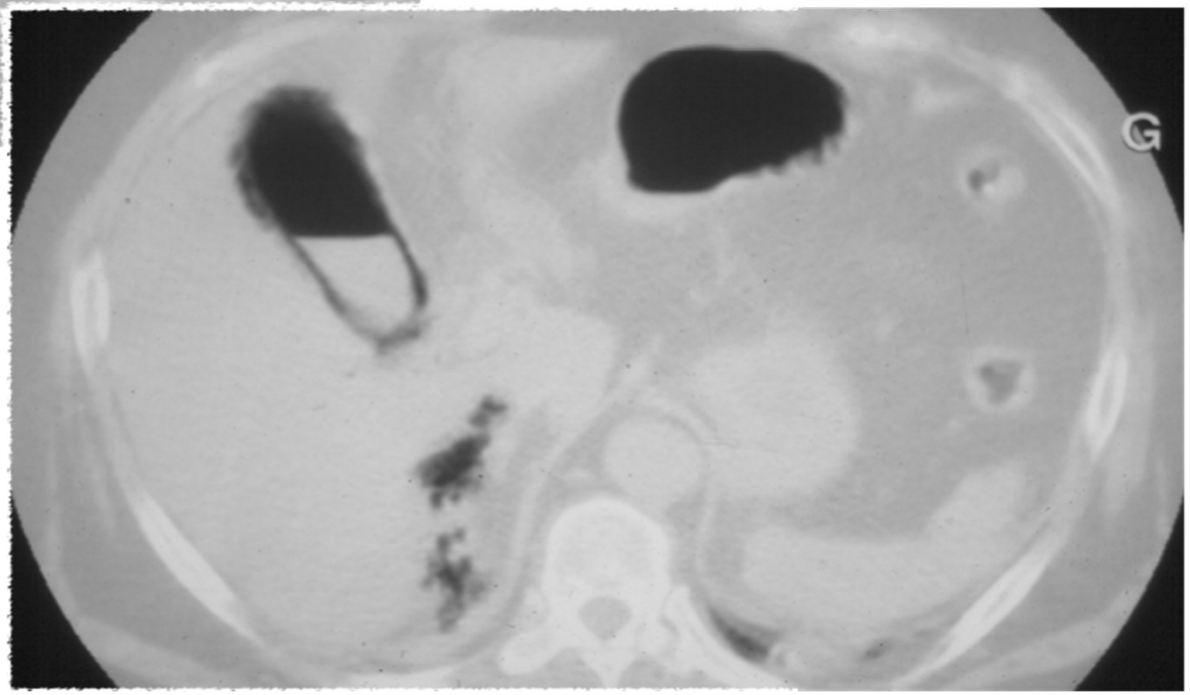
Gangrenous Cholecystitis & CT

ELEMENTS CLES
= > paroi irrégulière
= > défaut de prise de contraste
= > épaisseur de la paroi
= > petit axe de la vésicule

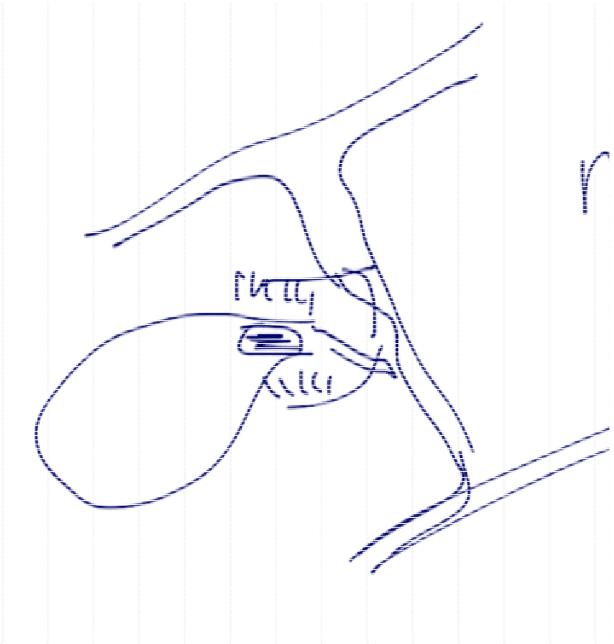
G.L. Bennet, CT Findings in Acute Gangrenous Cholecystitis, AJR February 2002 vol.178 no.2 275-281

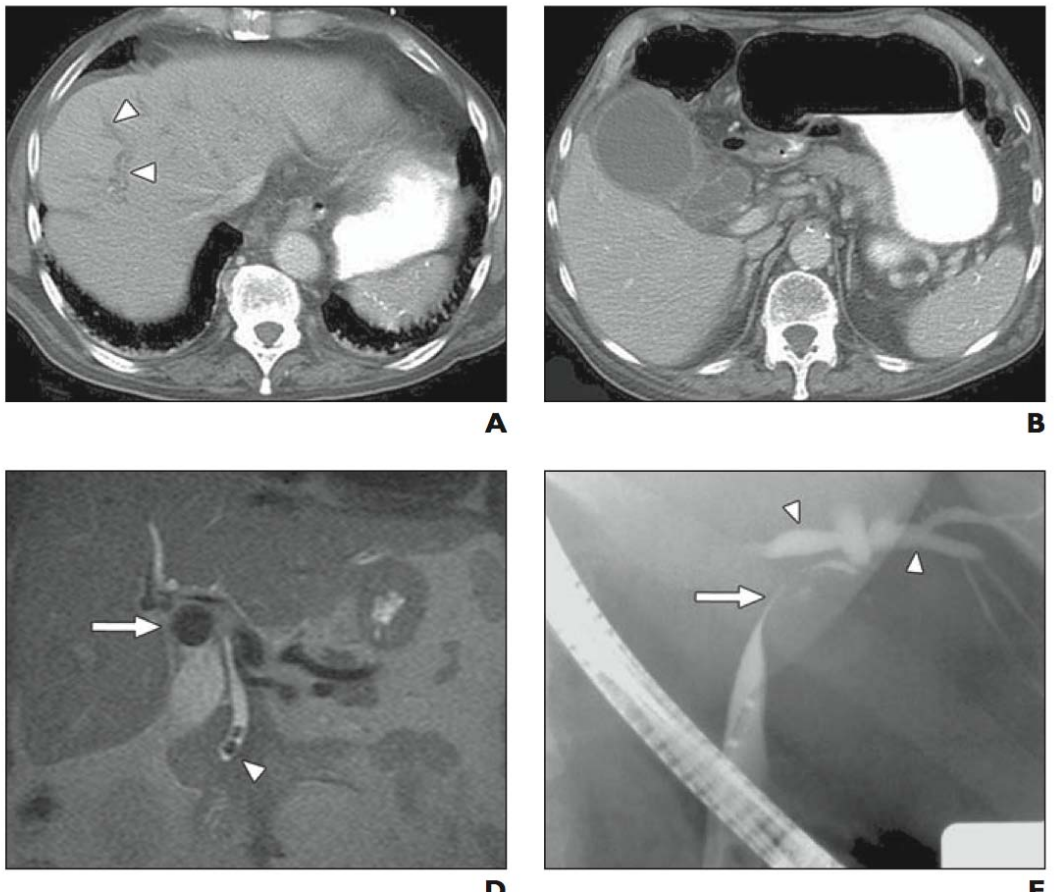
DD Cholécystite Emphysémateuse





Mirizzi Syndrom

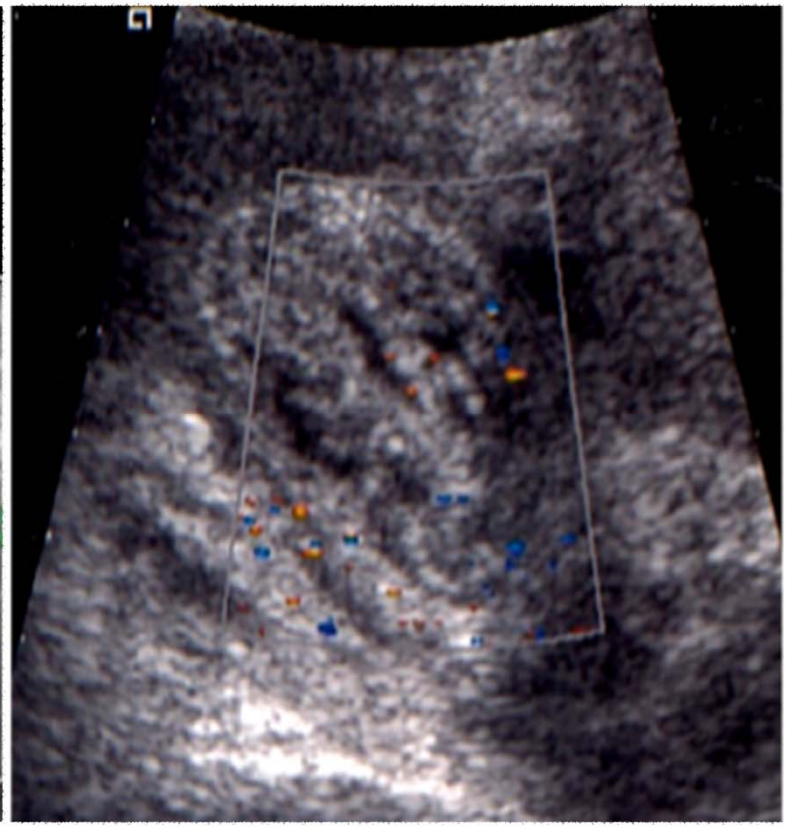




Ethan A. Smith[1]
Jonathan R. Dillman[1]
Khaled M. Elsayes[1]
Christine O. Menias[2]
Ronald O. Bude[1]

Cross-Sectional Imaging of Acute and Chronic Gallbladder Inflammatory Disease

AJR 2009; 192:188–196



Cirrhose **Hépatite** **Insuffisance Cardiaque**

Conclusion

- Lithiase vésiculaire
  - Diagnostic différentiel
- Lithiase des voies biliaires
  - Dans la voie biliaire principale
  - En intra hépatique
- Cholécystite ou DD