



Liver abscess caused by fish bone

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I. INTRODUCTION-

A liver abscess is defined as a pus-filled mass in the liver that can develop from injury to the liver or an intraabdominal infection disseminated from the portal circulation.[1] The majority of these abscesses are categorized into pyogenic or amoebic, although a minority is caused by parasites and fungi. Most amoebic infections are caused by Entamoeba histolytica. The pyogenic abscesses are usually polymicrobial, but some organisms are seen more commonly in them, such as E.coli, Klebsiella, Streptococcus, Staphylococcus, and anaerobes. While the incidence is low, it is essential to understand the severity of these abscesses because of the high mortality risk in untreated patients.[2] The usual pattern of abscess formation is that there is leakage from the bowel in the abdomen that travels to the liver through the portal vein. Many cases have an infected biliary tract that causes an abscess via direct contact. Liver abscesses can be classified in a variety of ways: One is by location in the liver. 50% of solitary liver abscesses occur in the right lobe of the liver (a more significant part with more blood supply), less commonly in the left liver lobe or caudate lobe. Another method is by considering the source: If the cause is infectious, the majority of liver abscesses can be classified into bacterial (including amoebic) and parasitic sources (including hydatiform cyst).

II. CASE REPORT -

A 53 year old male patient came to our Medicine OPD complaining of –

- Fever for 1month
- Loss of appetite 20 days.
- Pain abdomen for 10 days

HISTORY OF PRESENT ILLNESS–

- Patient was apparently alright 1 month back, to start with he developed intermittent high grade fever, highest recorded temperature is 104°F in evening after dinner. Fever is associated with chills and rigor, subsides only after taking antipyretic & cold sponging.
- He also complains of pain in the right upper abdomen for 10 days, which is dull aching in nature, radiates to right to shoulder ,associated with nausea, dry cough & most of time nocturnal, last for two to four hour, pain is more marked & gradually increasing in severity while lying in right lateral position .
- Also marked weight loss, loss of appetite and malaise.

PAST ILLNESS–No history of T2DM or Hypertension . No relevant surgical history.

PERSONAL HISTORY- Tobacco chewing for 20 years. Taking alcohol for 10years . He belongs to average socio-economic status . Married at age 27 yrs having 2 children. On Mixed Indian Diet.His bladder and bowel habits are normal

GENERAL EXAMINATION–

BP – 130/90 mmHg, Pulse 102/Min, Temp – 102 F, SpO2- 98% at Room Air, Respiration Rate – 20/Min, GCS- 15/15(E4 V5 M6).

Patient is Conscious but Lethargic and irritated.Patient has mild pallor with epigastric & right hypochondrium fullness, tender Hepatomegaly. There are no features of engorged veins, oedema, Lymphadenopathy, clubbing ,raised JVP and cyanosis .

COURSE IN HOSPITALIZATION–

Patient was transferred to ward and Treated with PRBC(packed red blood corpuscles) , Broad spectrum Anti-Biotics, anti-luminal, antipyretics and Other Supportive drugs.

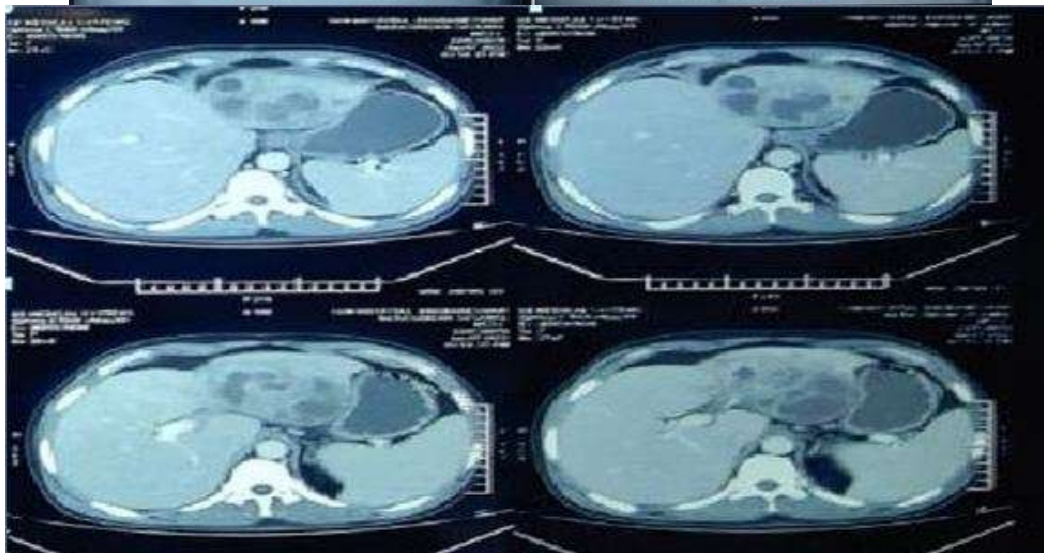
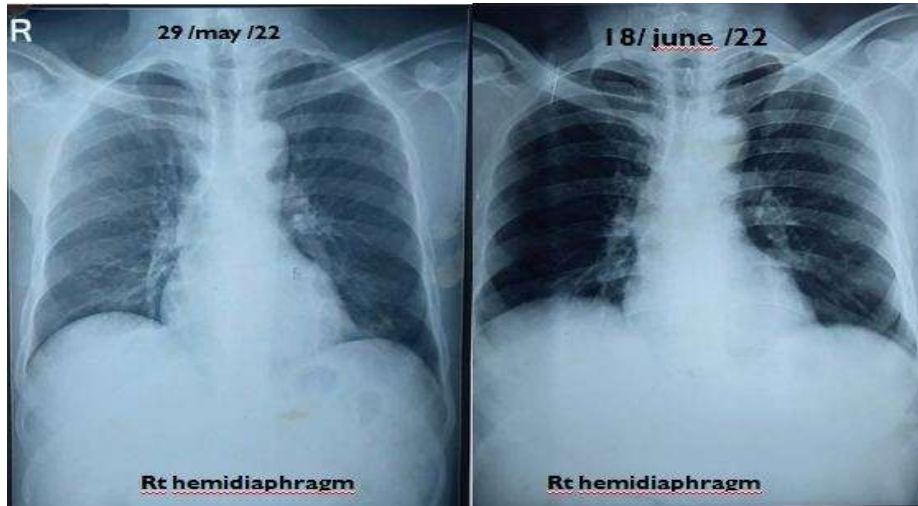
INVESTIGATIONS -

PARAMETERS	ADMISSION DAY	DAY 3 OF HOSPITALIZATION	POST SURGERY	ON DISCHARGE
TLC	99610/UL	89640/UL	41790/UL	31760/UL
RBC COUNT - m	2.37million/cumm	2.94million/cumm	2.89million/cumm	3.89million/cumm



HB%	6.7 gm%	10.2 gm%	9.1gm%	13.9gm%
PCV	25.9 vol%	24.7 vol%	23.7 vol%	24.7 vol%
MCV	115.6 fl	94.9fl	83.7.9fl	83.7.9fl
TPC	173000	189000	260000	260000
RDW-SD	56.7 fl	55.9fl	54.9fl	57.9fl
NEUTROPHIL	84.4%	78.4%	75.4%	71.4%
LYMPHOCYTE	30.7%	32.3%	12.3%	9.3%
MONOCYTES	4.0%	2.9%	3.9%	2.9%
EOSINOPHILS	0.5%	0.3%	0.1%	0.0%
BASOPHILS	0.4%	0.3%	0.3%	0.1%
CRP	50.5 mg/dl	50.5 mg/dl	30.9mg/dl	10.5 mg/dl
ESR	140			28
S. IRON	133 microgram/dl			
LDH	2412 IU/L			
UREA	19 mg/dl			
CREATININE	1.11 mg/dl			
SODIUM	135.7 mmol/l			
POTASSIUM	4.59 mmol/l			
URIC ACID	5.1 mg/dl			
CHLORIDE	105 mmol/L			
CALCIUM	7.0 mg/dl			
PHOSPHORUS	3.9 mg/dl			
TOTAL BILIRUBIN	0.58 mg/dl			0.38 mg/dl
DIRECT BILIRUBIN	0.86 mg/dl			0.51 mg/dl
ALT	43.7 U/L			40.7 U/L
AST	50.6 U/L			54.6 U/L
ALP	135 U/L			115 U/L
TOTAL PROTEIN	6.9 gm/dl			5.9 gm/dl
ALBUMIN	3.2 gm/dl			3.1 gm/dl
GLOBULIN	3.7 gm/dl			3.6 gm/dl
A/G RATIO	0.86			0.86
T3	1.39			
T4	9.7			
TSH	1.663			
PT	13.5 sec	13.6 Sec		13.86 Sec
INR	1.93	1.86		1.76
APTT	31.8	32.3		31.8
Hepatitis & HIV Panel – Negative.				
USG – liver abscess in left lobe , mild Hepatomegaly.				
CHEST X-RAY – RIGHT HEMI-DIAPHRAGM				
CECT ABDOMEN – multiple inter-communicating liver abscess in segment II & III, thin curvilinear foreign body in left sub-hepatic region abutting antro-pyloric region of				

stomach with other end in segment III.



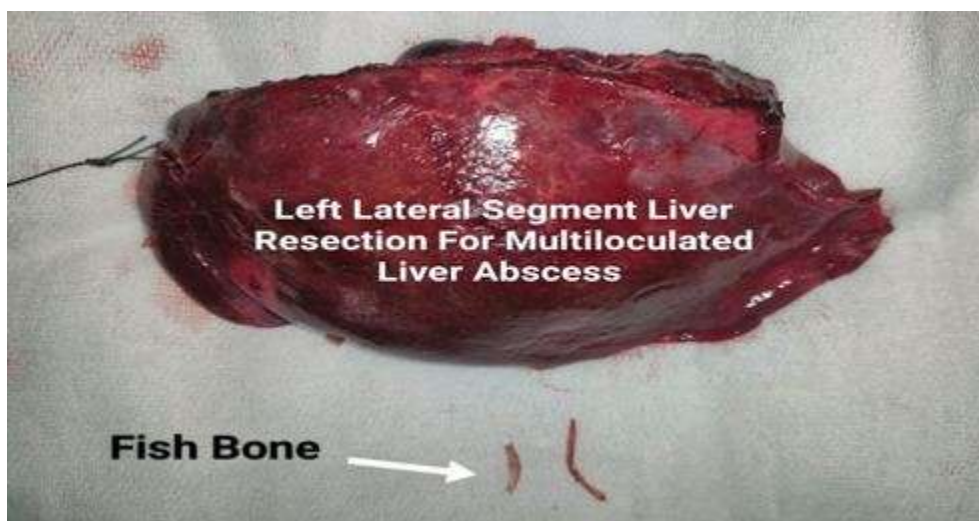
CECT Abdomen (02.06.2022): Multiple inter communicating liver abscess in Seg II and III with small left Subhepatic extension. Thin curvilinear foreign body in left sub hepatic region abutting antro pyloric region of stomach with one end entering into Seg III abscess. Right distal ureteric calculus.

CECT Abdomen (07.06.2022): No extravasation of contrast on positive oral contrast.

TREATMENT / MANAGEMENT-

Managed with Empirical antibiotic for coverage of Enterobacteriaceae, anaerobes, streptococci, enterococci, and Entamoeba histolytica. Such antibiotic regimens include Beta-lactam Beta-Lactamase inhibitor plus metronidazole.

Exploratory Laparotomy-hepatic resection of lobe (seg II & III) with liver abscess drainage & foreign body (two fish bone length 3.2 & 1.8 cm) extracted and repair of gastric perforation at antro-pyloric region of stomach.



III. DISCUSSION:-

Bacterial liver abscesses are common clinical infectious disease of the liver and mainly present as fever, chill, and stomachache.

Bacteria can invade the liver through three routes (biliary tracts, hepatic arteries, and portal veins) to cause the abscess. In comparison, the liver abscess that resulted from the penetration of foreign bodies through the stomach wall after entering the digestive tracts is rarely seen in the clinical practice.

Foreign bodies usually get impacted at the oesophageal sphincters, pyloric canal, duodenum, ileocaecal valve, and anus where there is expected normal extrinsic impression or anatomical narrowing. Perforations are more common in the oesophagus causing pneumo-mediastinum, retropharyngeal abscess, mediastinal abscess, and bleeding. Perforations and complications beyond the esophagus are rare.³ Normally, 80% to 90% of the foreign bodies in the digestive tract can be eliminated by gastric acid, anti-bacterial gastric juice & inflammatory action.

However 1% of the fish bone is sufficiently large or hard that cannot be digested by gastric juice or pass through, due to pyloric stenosis, the sharp fish bone is very likely to penetrate the stomach wall due to the rapid contraction of the stomach during peristalsis.

Perforation usually in stomach lesser curvature because of the anatomical acute angularity with abscess most commonly developing in left hepatic lobe, because of its proximity .

Foreign body width larger than 2 to 2.5cm, can't get through the pylori and longer than 5 to 6cm, cannot pass through the duodenum preventing from being discharged out of digestive tract.

The classic clinical features of hepatic abscess like fever, abdominal pain, and jaundice is usually not seen, most patients have vague symptoms such as anorexia, and vomiting with leukocytosis, deranged liver function test, making the diagnosis challenging without definitive history of fish bone ingestion.

Microorganisms found in the abscess cultures are the common bacterial flora seen in the oropharynx.^[5]

Small abscess less than 5 cm treated with antibiotics. Larger abscess with multi-loculations treated with surgery, draining of the abscess, and removal of the foreign body.

IV. CONCLUSION:-

In conclusion, Secondary liver abscess, unlike other liver abscess ,due to foreign body require urgent reasonable and therapeutic regimen according to the medical history ,laboratory examination and imaging manifestation.

The patients with accidental fish bone ingestion complications present with nonspecific features of acute abdomen. The clinical diagnosis is challenging without a definitive history of fish bone ingestion. A CT scan with its multiplanar capability is highly valuable to diagnose and accurately localize the ingested fish bone, which can direct accurate timely treatment. In addition, the CT can also provide a comprehensive evaluation of the complications of fish bone ingestion including hepatic abscess. [3,5]

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