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Findings that shed new light on the possible pathogenesis of a disease or an adverse effect

Fusobacterium necrophorum – beyond Lemierres syndrome

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Summary

Fusobacterium necrophorum is a non-sporulating anaerobic gram negative bacillus and has traditionally been associated with Lemierre's syndrome. The authors report a 34-year-old male who presented to the emergency department with a week's history of dull epigastirc pain. Significant medical history included chronic pancreatitis secondary to alcohol use. The patient had radiological evidence of acute on chronic pancreatitis with thrombosis of the portal vein and multiple intrahepatic abscesses. CT-guided drainage of left upper quadrant revealed fluid collection in the pancreatic bed. The fluid culture grew *F necrophorum* and the patient was treated with tigecycline for 4 weeks. The patient improved symptomatically and his follow-up computerised axial tomography scan 2 months later showed resolving liver abscess, cavernous transformation of the portal vein and stable findings of chronic pancreatitis. This could represent an infection of the peripancreatic tissue with *F necrophorum* further leading to pylephlebitis.

BACKGROUND

Fusobacterium necrophorum is an anaerobic gram negative bacillus forming a part of the family Bacteroidaceae. These bacteria are a predominant part of the oral flora. It has traditionally been associated with Lemierre's syndrome. Lemierre syndrome, also known as postanginal septicemia or necrobacillosis, is an uncommon but potentially life-threatening complication of acute pharyngo-tonsillitis. It is characterised by acute primary infection of the oropharynx causing secondary thrombophlebitis of the internal jugular vein and associated with subsequent septicemia and septic embolisation.[1] When pharyngitis due to *Fusobacterium* species occurs, the physical proximity of the vessels in the lateral pharyngeal space permits extension from the peritonsillar space to the internal jugular vein. This usually occurs in less than a week from the development of pharyngitis. Once invasion of the internal jugular vein is achieved, the resultant bacteremia triggers platelet aggregation and thrombus formation.[2] Thrombus formation and rapid bacterial growth result in a nidus for metastatic septic embolisation, commonly to the lungs and less commonly to the large joints.[3]

We report a patient with radiological evidence of acute on chronic pancreatitis with thrombosis of the portal vein and multiple intrahepatic abscesses. *F necrophorum* was isolated from the peripancreatic and perihepatic fluid. This could represent an infection of the pancreatic and the peripancreatic tissue with *F necrophorum* further leading to thrombophlebitis of the portal vein and subsequent septic embolisation. This could reflect pathology similar to the clinical features seen in Lemierre's syndrome.

CASE PRESENTATION

A 34-year-old Caucasian male presented to the emergency department with a week's history of dull epigastric pain. The pain had been increasing in intensity and was discomforting to the patient. No aggravating or relieving factors were noted and there was no radiation of the pain. The patient had history of excessive alcohol use until 1 year prior to current presentation. He was diagnosed with chronic pancreatitis one year back and has had abdominal pain on and off since then. He also had steatorrhea suggestive of pancreatic exocrine dysfunction; however previous investigation had not shown any clinical significant vitamin deficiency. The current presentation was described as an exacerbation of his previous symptoms. There were no other associated symptoms and the patient was able to tolerate a liquid diet.

The patient was afebrile and haemodynamically stable. He was cachectic and pallor was noted. Respiratory and cardiovascular examinations were unremarkable. Epigastirc tenderness was elicited and moderate abdominal distention without any fluid thrill was noted. Neurological examination including cranial nerve examination was unremarkable.

INVESTIGATIONS

Routine laboratory investigations showed low haemoglobin of 12.8 g/dl, elevated white blood cell count of 26 100 cells/mm^3 with absolute neutrophil count of 25 300 cells/mm^3. Platelet count was in the normal range as the electrolytes, renal function and liver function tests. Computerised axial tomography (CAT) scan of abdomen and pelvis showed acute pancreatitis superimposed upon underlying chronic calcified pancreatitis with fluid collection in the lesser sac, porta hepatis, perisplenic region and left subphrenic region consistent with pseudocysts. There were low density lesions in both lobes of the liver consistent with intrahepatic abscesses (figure 1). There was acute thrombosis of left and right portal vein branches. The spleen was enlarged with lesions consistent with splenic abscesses and there was occlusion of the splenic vein and

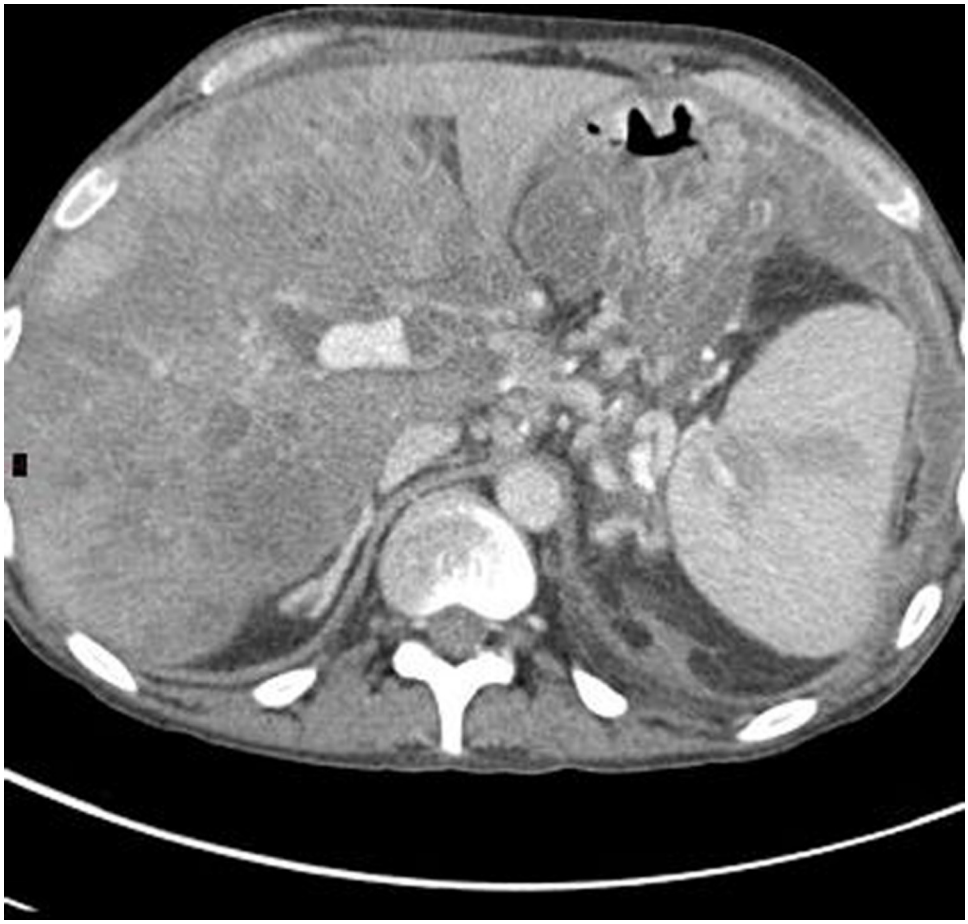

Figure 1 Computerised axial tomography scan of abdomen showing thrombosis of the portal vein along with hepatic and splenic abscesses.

superior mesenteric vein thrombosis with numerous collaterals throughout the upper abdomen. There was however no evidence of hepatic parenchymal changes suggestive of hepatic cirrhosis.

TREATMENT

The patient was initially started on intravenous vancomycin and meropenem. The infectious disease service was consulted and antibiotics were changed to intravenous tigecycline and meropenem. The blood cultures remained negative after a week of incubation. CT-guided drainage of left upper quadrant revealed fluid collection in the pancreatic bed. This was initially haemorrhagic and later purulent. The fluid culture grew *F necrophorum* on the third day. Considering contiguous infection from the pancreatic and peripancreatic tissue causing septic thrombophlebitis of the portal vein, other causes such as hypercoagulable state were not considered.

CAT scan of the neck and chest was specifically ordered to evaluate for the classical features of Lemierre's syndrome associated with *F necrophorum*. There was no evidence of the internal jugular vein. The patient further failed to give any preceding history suggestive of any oropharyngeal or respiratory infection. Lungs are the most common site of metastasis[3]; however the patient did not have any radiological evidence of any pulmonary involvement. Features of lateral pharyngeal space involvement leading to Horner's syndrome or trapezius muscle involvement were not present.

OUTCOME AND FOLLOW-UP

Meropenem was discontinued and the patient was continued on tigecycline for 4 weeks. The patient was followed up in the clinic. He improved symptomatically and his follow-up CAT scan 2 months later showed resolving liver abscess, cavernous transformation of the portal vein and stable findings of chronic pancreatitis. Cavernous transformation of the portal vein is the development of periportal collaterals around the recanalising or occluded main portal vein which occurs secondary to chronic portal vein thrombosis. Numerous collateral vessels that vary in character from small to markedly enlarged and serpiginous end up functionally 'replacing' the portal vein.

DISCUSSION

Septic thrombophlebitis of the portal vein also known as pylephlebitis is usually a complication of any intra-abdominal or pelvic infection that occurs in the region drained by the portal venous system, especially diverticulitis or appendicitis. Contiguous spread from nearby intra-abdominal infection has also been described. The diagnosis of septic thromobophlebitis is made on culture data in light of radiological diagnosis of acute thrombosis of any vessel.[4] Enteric gram negative bacilli and anaerobes, especially *Bacteroides*

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fragillis are the most common organisms associated with pylephlebitis.[4] There have been some isolated cases of pylephlebitis associated with Fusobacterium species.[5–9] Most of these patients had an identified gastrointestinal source of their portal vein infection. Liver abscesses and bowel ischemia are acute complications of pylephlebitis. Portal hypertension could be a long-term complication.

We report a patient with radiological evidence of acute on chronic pancreatitis with thrombosis of the portal vein and multiple intrahepatic abscesses. *F necrophorum* was the only organism isolated from the peripancreatic and perihepatic fluid. This could represent an infection of the pancreatic and the peripancreatic tissue with *F necrophorum* and contagious spread leading to pylephlebitis. Hepatic and splenic abscess could represent metastatic septic emboli from the infection.

Learning points

- *F necrophorum* is an anaerobic gram negative bacillus forming a part of the family Bacteroidaceae. These bacteria are a predominant part of the oral flora.
- Lemierre syndrome is characterised by acute primary infection of the oropharynx causing secondary throbophlebitis of the internal jugular vein and associated with subsequent septicemia and septic embolisation.
- When pharyngitis due to *Fusobacterium* species occurs, the physical proximity of the vessels in the lateral pharyngeal space permits extension from the peritonsillar space to the internal jugular vein; and the resultant bacteremia triggers platelet aggregation and thrombus formation.
- Septic thrombophlebitis of the portal vein also known as pylephlebitis is usually a complication of any intra-abdominal or pelvic infection that occurs in the region drained by the portal venous system, however contiguous spread from nearby intra-abdominal infection has also been described.
- Liver abscesses and bowel ischemia are acute complications of pylephlebitis. Portal hypertension could be a long-term complication.

Competing interests None.

Patient consent Obtained.

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