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Acute portal venous thrombosis caused by severe sepsis and not associated with liver disease

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Abstract

Background: Portal venous thrombosis (PVT) can be generally classified into three categories- acute non cirrhotic PVT, chronic PVT (also called extrahepatic portal venous occlusion), and PVT in cirrhosis. PVT is usually seen in those with liver disease and it is rare in patients without. Management is usually with anticoagulation and finding and treating the etiology of the PVT. If untreated, patients can develop portal hypertension. This patient presented a unique diagnostic challenge as she had risk factors for numerous etiologies of PVT.

Case description: A 64 year old female with a history of hypertension, hyperlipidemia, and a lung nodule presented to the emergency department with fatigue, fever, nausea, and vomiting. She sought medical care upon returning home from a cruise in Mexico. She was febrile and tachycardic and, though she did not have any abdominal tenderness, stated that a few days prior she did have right upper quadrant abdominal pain. CT scan with IV contrast was ordered and showed a masslike area in the anterior left hepatic lobe, acute left PVT, and enlarged porta hepatis lymph nodes. Subsequently, when the patient was inpatient, an MRCP was ordered as there was concern for malignancy given the CT findings which redemonstrated the left portal venous thrombosis and the mass described on the CT was further determined to be hepatic periportal edema. Shortly after presentation she was started on broad spectrum antibiotics given concern for sepsis as well as anticoagulation for the PVT. Blood cultures grew streptococcus intermedius, strepconstellatus, and eikenella corrodens. Given a dental procedure two months prior, a TEE was performed which did not show vegetation and endocarditis was ruled out. Given a family history of colon cancer she had a colonoscopy which showed diverticulosis with friable mucosa. That was ultimately thought to the be etiology of her bacteremia.

Discussion: This case was interesting in that the patient had risk factors for multiple etiologies of her sepsis and PVT. She had recent dental work, recent travel on the cruise, a history of a lung nodule, family history of colon cancer, initial CT scan with concern for liver mass and possible metastatic disease. She had a thorough work up that ultimately led to the thought that her bacteremia was due to diverticular disease and her sepsis was likely the etiology of her PVT. She was placed on antibiotics for 4 weeks outpatient as well as started on oral anticoagulation with a plan for repeat imaging of the PVT at the three month mark to determine length of anticoagulation.

Background

PVT is generally classified into three categories- acute noncirrhotic, chronic, and PVT in cirrhosis. It is more common in patients with liver disease and can be detrimental if left untreated. It is considered rare in the general population, but has been noted to have a prevalence of 4.4-15% in those with cirrhosis. It is also associated with being the etiology for 5%-10% of portal hypertension cases. (1)

Treatment usually includes anticoaguation, but at times this is contraindicated due to bleeding risk. Some patients with this condition are candidates for thrombolysis, thrombectomy, TIPS, and liver transplant (2). Hypercoaguable states, such as malignancy, can increase risk for PVT. Complications of PVT include portal hypertension with gastrointestinal hemorrhage, small bowel ischemia, and acute ischemic hepatitis. Acute PVT is usually associated with abdominal pain and sometimes acute ascites. Chronic PVT has evidence of collateral circulation on imaging.

Interestingly, local inflammation (diverticulitis, appendicitis, cholecystitis) can also lead to PVT and increase the risk for pylephlebitis. This patient case is an example of this phenomenon (3).

Case Description

Chief Complaint: "Nausea, vomiting, fatigue, chills"

History of Present Illness: A 64 year old female presented to the emergency department with fatigue, fever, nausea, and vomiting. She flew back to the United States from a cruise to Mexico the day of presentation to the Emergency department. She stated that she was around people on the cruise who had similar symptoms of vomiting and chills. Her symptoms started two days prior with chills and diaphoresis and right upper quadrant abdominal pain which had resolved. Upon boarding the plane on day of presentation she experienced nausea, vomiting, and while she was vomiting urinary incontinence. She also admitted to a right frontal headache, but stated she regularly gets headaches and this was not abnormal in quality or severity.
She later also admitted to dental surgery 2 months prior to presentation.
She denied chest pain, shortness of breath, a current feeling of nausea, dysuria, diarrhea, constipation.

Past Medical History: Hypertension, hyperlipidemia, lung nodule

Past Surgical History: Cesarean section, total abdominal hysterectomy with bilateral salpingoopherectomy

Social History: Never smoker, occasional tobacco use

Vital Signs: Temperature 39.3 C Orally, Pulse 113, Resp. 20, BP 146/68, SpO2 97% RA

Physical Exam:
Constitutional: She appears well-developed and well-nourished. No distress. She is alert.
HENT: Head: Normocephalic and atraumatic. Nose: Nose normal. Mouth/Throat: Oropharynx is clear and moist. Eyes: EOM are normal. Pupils are equal. Neck: Normal range of motion. Neck supple.
Cardiovascular: Regular rhythm, S1 normal, S2 normal and intact distal pulses. Tachycardia present.
Pulmonary/Chest: Effort normal and breath sounds normal. No respiratory distress. She has no wheezes. She has no rales.
Abdominal: Soft. Normal appearance and bowel sounds are normal. There is no tenderness, rebound, or guarding.
Neurological: She is alert and oriented to person, place, and time.
Skin: Skin is warm and dry.

Lab Results

Urinanalysis			
Blood	negative	**WBC**	2/HPF
LE	negative	**Protein**	100 mg/dL
Nitrite	negative	**Ketone**	5mg/dL
RBC	1/ HPF	**Bacteria**	none

BMP			
Na	131 mmol/L	**AG**	14
K	4.1 mmol/L	**BUN**	21 mg/dL
Cl	98 mmol/L	**Cr**	0.69 mg/dL
CO2	19	**Glucose**	115 mg/dL

LFTs, Lipase, Lactic Acid			
ALT	46 IU/L	**D bili**	14 mg/dL
AST	42 IU/L	**Alk Phos**	109 IU/L
Albumin	2.8 g/dL	**Lipase**	20 IU/L
T. bili	0.6 mg/dL	**LA**	1.3 mmol/L

CBC with Diff					
WBC	24.2 K/uL	**Neutrophil %**	88	**Abs. Neut**	21.1 K/uL
RBC	4.07 M/uL	**Lymphocyte %**	3	**Abs. Lymph**	0.8 K/uL
Hgb	11.2 g/dL	**Monocyte %**	9	**Abs. Mono**	2.3 K/uL
Hct	34.4 %	**Eosinophil %**	0	**Abs Eosinophil**	0.0 K/uL
Plt	295 K/uL	**Basophil %**	0	**Abs. Basophil**	0.0 K/uL

Imaging Results

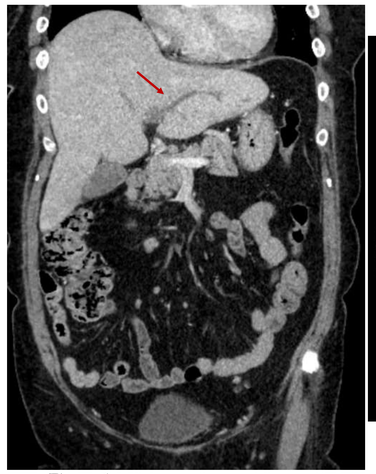

Figure 1

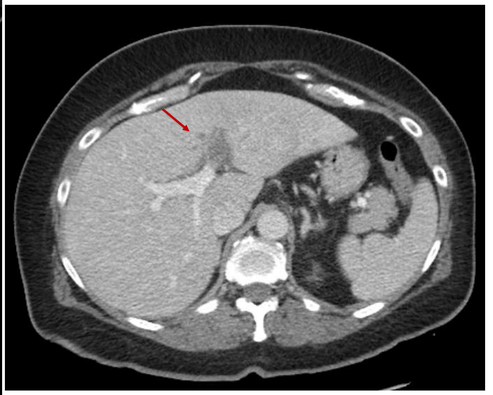

Figure 2

Figure 1 and 2. Radiology read: "Ill-defined masslike area within the anterior left hepatic lobe is seen measuring 5.0 x 2.3 cm in size. There is mild dilatation of the left intrahepatic biliary tree. Difficult to exclude possible mass at the proximal left main bile duct. The left portal vein is thrombosed with what appears to be acute thrombus present in the proximal left main portal vein. Findings are overall suspicious for underlying neoplastic process. A follow-up MRI/MRCP with contrast is recommended for further evaluation.
2. Mildly enlarged upper abdominal and porta hepatis lymph nodes are seen measuring up to 18 mm in short axis diameter. While findings may be reactive, possibility of metastatic disease not excluded."

Discussion

In the emergency department, the decision was made to proceed with a CT of the abdomen/pelvis given the patient's septic picture (WBC 24, HR 113, Temp 39.3 C) with no clear source and reported earlier right upper quadrant abdominal pain. This decision revealed her unique pathology of the portal venous thrombosis, raised concern for hepatic malignancy, and she was started on anticoagulation. The next question, why did she have PVT was further investigated by the inpatient team.

She had an MRCP which showed the area on concern on CT for ill defined mass was consistent with edema and no mass was visualized. Within 24 hours her blood cultures resulted as positive for multiple organisms (streptococcus intermedius, strepconstellatus, and eikenella corrodens) and the hunt continued for the source of her bactermia. Given her history of dental procedures she had a TEE that ruled out endocarditis, a colonoscopy which showed friable tissue and was thought to be the source of her bacteremia and PVT.

Upon discharge, she was placed on IV antibiotics for 4 weeks outpatient as well as started on oral anticoagulation with a plan for repeat imaging of the PVT at the three month mark to determine length of anticoagulation.

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