As, found in 13% of liver cirrhosis with clinically significant portal hypertension. About 5% of liver cirrhosis with ascites is associated with hepatic hydrothorax. Hydromediastinum is a rare condition, and all reported cases are idiopathic (i.e., in origin), and associated with insertion of central venous catheter. No spontaneous hydromediastinum had been reported before. We reported a case of spontaneous hydromediastinum, associated with decompenated liver cirrhosis and hepatic hydrothorax.

Case Report: A 66 years-old male with HBV-related liver cirrhosis was referred to Far Eastern Memorial Hospital for massive ascites. He was positive for HBsAg, anti-HBe, negative for HBeAg, anti-HDV or anti-HCV. His serum albumin at that time was 1.7 g/dl, total bilirubin 1.6 mg/dl, direct bilirubin 0.9 mg/dl and PT 17.3 sec (control 11.3 sec). He was treated with large volume paracentesis, furosemide and spironolactone but effective diuretic dose could not be used because of frequent development of hepatic encephalopathy. The patient developed right-sided pleural effusion 7 months later. His serum albumin was 2.1 g/dl, total bilirubin 2.3 mg/dl, direct bilirubin 1.3 mg/dl and PT 17.9 sec (control 11.4 sec). Nuclear medicine study with intra-peritoneal injection of Technetium-99m-DTPA showed uptake of tracer in right pleural cavity and mediastinum after 2 hours. CXR and chest CT showed massive right-sided pleural effusion with minimal amount of fluid in mediastinum. The patient died from SBP with septic shock 3 months later.

Conclusion: Spontaneous hydromediastinum may be extension of hepatic hydrothorax through defect in parietal pleura. But its clinical significance in liver cirrhosis is not clear because the amount is minimal. Risk of mediastinitis may be possible if the patient developed SBP.

470 PRIMARY ISOLATED GASTRIC VARICES IN A PATIENT WITH NON HODGKINS LYMPHOMA

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A fifty-one-year-old man admitted with dizziness after 2 weeks of bright red blood per rectum. He was undergoing CHOP chemotherapy for biopsy proven NHL. Review of systems: persistent left sided abdominal pain radiating to left flank and post-prandial fullness. EXAM remarkable for hepatosplenomegaly and heme-stools. Hct 30 MCV 77 Alb 3.5 AST 92 ALT 60 PT 14 PTT 30 CT; ill defined, 20cm splenic mass extending to the upper pole of left kidney, 19 cm liver, no focal lesions. EGD: esophagitis, grade II fundic varices Colonoscopy: hemorrhoids Doppler U/S: hepatic portal flow in the portal vein, patent splenic vein, persistent splenic mass. + HCV antibody. See figures: EGD, U/S, CT. IGV (isolated gastric varices) is a rare clinical entity typically seen after treatment of esophageal varices with sclerotherapy (secondary IGV). IGV is usually due to segmental portal hypertension. Medline lists 228 cases of segmental portal hypertension secondary to splenic vein thrombosis mostly associated with chronic pancreatitis, pancreatic cancer, and myeloproliferative disorders. Only 1 case of IGV in the setting of Hodgkin’s lymphoma has been reported. IGV was found in only 4.7% cases in a review of 1,128 patients with portal hypertension. Of all IGV cases, only 15% had primary IGV as in our patient. Interestingly, our patient had no evidence of splenic vein obstruction as demonstrated by doppler U/S. IGV without venous thrombosis has been reported twice and the exact cause was not determined. We postulate that the presence of varices in this case, is due to extrinsic compression by the splenic mass. However, the rare chance of thrombus in proximal splenic vein cannot be excluded given the limitations of doppler U/S. There is a remote possibility of chronic HCV related liver disease contributing to fundic varices, however, occurrence of IGV in the setting of cirrhosis is less than 2% and there are no cases of IGV reported in the absence of cirrhosis. Though he is HCV antibody +, there is no indication of cirrhosis by the imaging studies noted above. In the absence of cirrhosis, HCV is unlikely to be the cause of his IGV. He remains asymptomatic for IGV. Liver bx is planned upon completion of CHOP. This is the first case of primary IGV in the setting of NHL. Absence of splenic vein thrombus as demonstrated by doppler U/S makes this case unique. Though rare, non thrombotic
etologies of portal hypertension should be considered in the differential of primary IGV.

471

OVARIAN CANCER METASTATIC TO THE STOMACH DIAGNOSED BY EUS-GUIDED FINE NEEDLE ASPIRATION

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Ovarian cancer is the seventh most common cancer in women and the leading cause of death among those with gynecological malignancies. Epithelial ovarian cancers commonly spread along intraperitoneal and lymphatic channels. Gastric metastasis of ovarian cancer is extremely rare. In this report we describe the endosonographic appearance of ovarian cancer metastatic to the stomach and demonstrate the utility of EUS-FNA in providing a diagnosis. A 62 year-old woman with a history of ovarian cancer presented with gastrointestinal complaints of belching, reflux and epigastric discomfort. Seven years prior to presentation, she was diagnosed with ovarian cancer. She initially underwent surgical debulking followed by 6 cycles of paclitaxel and cisplatin. After this she was monitored closely, and was thought to be disease-free until one year prior to presentation when her CA-125 started to rise. Chest, abdomen and pelvic CT revealed no recurrence. EGD revealed a submucosal mass with central ulceration in the proximal body of the stomach. Multiple biopsies were obtained showing only mild chronic gastritis. The patient was referred for endosonography of the mass. EUS demonstrated a heterogenous 4-cm mass with irregular borders arising from the muscularis propria. FNA was performed using a 22g needle. The preliminary endosonographic and cytologic diagnoses were gastrointestinal stromal tumor (GIST). However, final pathologic findings were consistent with a poorly differentiated carcinoma. Immunoperoxidase staining was positive for CA-125, and cytokeratins 7 and AE1:3. These findings were consistent with metastatic ovarian cancer and the patient subsequently began another chemotherapeutic regimen. Two previously reported cases of ovarian cancer and intramural gastric metastases presented with gastrointestinal bleeding. The majority of submucosal tumors (SMT), however, do not cause symptoms. The role of EUS in diagnosing SMTs is now well established. By EUS, metastatic deposits in the stomach appear as hypoechoic heterogenous masses which may involve any of the sonographic layers. For gastric SMTs, EUS-FNA is only 60% sensitive in providing diagnostic pathologic material. The patient in our case-report had an unrevealing CT and the only indication of recurrent disease was an increase in serum CA-125. Hence EUS-FNA was key in diagnosing metastatic disease in this patient.

472

SWEET’S SYNDROME: A RARE EXTRAITESTINAL MANIFESTATION OF INFLAMMATORY BOWEL DISEASE

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Sweet’s syndrome (SS) also known as acute febrile neutrophilic dermatosis is characterized by the acute onset of painful and erythematous cutaneous plaques. These skin lesions are usually accompanied by fever, arthralgias and leukocytosis. The histopathological feature of this condition is the dense dermal infiltrate of mature polymorphonuclear leukocytes. SS has been reported in association with a variety of diseases. We present two patients with Ulcerative Colitis (UC) presenting with SS and exacerbation of their inflammatory bowel disease (IBD). A 55 year-old woman with UC for two years presented with acute onset of multiple erythematous cutaneous plaques scattered over her extremities, head, face and back. The patient also had fever, arthralgias, general malaise and symptoms suggestive of UC exacerbation (bloody diarrhea, LLQ pain and tenesmus). Her symptoms did not improve with oral steroids after one week and her skin lesions worsened. Skin biopsy showed neutrophilic dermatitis, and lesions were characteristic of SS. Oral steroid therapy was increased to prednisone 60mg PO QD. Within one week she had improvement with complete resolution of the cutaneous lesions and UC symptoms. Prednisone was weaned with no recurrence of skin lesions. A 40 year-old man with UC for 19 years started with back pain for which he used NSAID’s. The patient then developed increase in bowel movements, loose bloody stools, lower abdominal pain and tenesmus. He was started on therapy with azathioprine in addition to mesalamine 4.8 g/day. One week prior to admission he also developed painful erythematous plaques on his extremities associated to fever, arthralgias and general malaise. He had WBC 14,400 mm3, Hgb 10.0 g/dl and ESR 60. Skin biopsy was compatible with SS and sigmoidoscopy showed active UC. The patient was admitted and treated with methylprednisolone 48 mg IV QD. Three days after admission he developed a bullous skin lesion of 4 cm in diameter on his left anterior leg with a central dark pigmented area consistent with pyoderma gangrenosum. After several days the skin lesions and UC symptoms started to improve. He was discharged home with oral steroids, azathioprine and mesalamine with resolution of all symptoms. A spectrum of extraintestinal manifestations may occur in patients with UC. SS is a rare skin disorder, which must be considered in the differential diagnosis of patients with IBD and skin lesions.

473

AMYLOIDOSIS CAUSING GASTRIC OUTLET OBSTRUCTION IN A PATIENT WITH NON-HODGKIN’S LYMPHOMA

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A 66 year-old white woman with a history of rheumatoid arthritis was hospitalized for the work-up of diffuse abdominal pain and a palpable abdominal mass. The patient reported worsening diffuse abdominal pain associated with nausea, recurrent vomiting, and early satiety. She denied any history of hematemesis, melena, pancreatic, or hepatobiliary disease. On physical examination, the patient was afibrile. The abdomen was distended, diffusely tender, and left upper quadrant fullness was noted. Significant laboratory data included a white blood cell count of 16000/ cubic millimeter ( normal: 4500–11500), a hemoglobin of 9.7 g/dl (12–15), albumin 2.3 g/dl (3.5–5), iron 6 mg/dl (50–160) and the iron saturation was 3% (20–55). Platelets, bilirubin, transaminases and pancreatic enzymes were within normal limits. Computed tomography (CT) of the abdomen and pelvis revealed diffuse soft tissue masses in addition to ascites. A large (8 cm) mass was noted to be compressing the distal stomach and causing marked gastric distention. A CT guided biopsy of one of the masses revealed amyloid deposition in addition to infiltration with plasma cells. An upper endoscopy showed a large, ulcerated antral mass causing complete gastric outlet obstruction. Hematoxylin/Eosin (H&E) and Congo Red staining of the endoscopic biopsies confirmed the presence of amyloid deposits. In view of the presence of plasma cells on biopsy, a bone marrow biopsy was performed and a diagnosis of non-Hodgkin’s B-cell lymphoma was made. The patient was started on chemotherapy using CHOP (cyclophosphamide, adriamycin, oncovin and prednisone) in addition to Rituximab. Three days later the patient developed a spontaneous left colonic perforation and underwent a left hemicolectomy with end-ostomy. A 473 A 66 year-old white woman with a history of rheumatoid arthritis was hospitalized for the work-up of diffuse abdominal pain and a palpable abdominal mass. The patient reported worsening diffuse abdominal pain associated with nausea, recurrent vomiting, and early satiety. She denied any history of hematemesis, melena, pancreatic, or hepatobiliary disease. On physical examination, the patient was afibrile. The abdomen was distended, diffusely tender, and left upper quadrant fullness was noted. Significant laboratory data included a white blood cell count of 16000/ cubic millimeter ( normal: 4500–11500), a hemoglobin of 9.7 g/dl (12–15), albumin 2.3 g/dl (3.5–5), iron 6 mg/dl (50–160) and the iron saturation was 3% (20–55). Platelets, bilirubin, transaminases and pancreatic enzymes were within normal limits. Computed tomography (CT) of the abdomen and pelvis revealed diffuse soft tissue masses in addition to ascites. A large (8 cm) mass was noted to be compressing the distal stomach and causing marked gastric distention. A CT guided biopsy of one of the masses revealed amyloid deposition in addition to infiltration with plasma cells. An upper endoscopy showed a large, ulcerated antral mass causing complete gastric outlet obstruction. Hematoxylin/Eosin (H&E) and Congo Red staining of the endoscopic biopsies confirmed the presence of amyloid deposits. In view of the presence of plasma cells on biopsy, a bone marrow biopsy was performed and a diagnosis of non-Hodgkin’s B-cell lymphoma was made. The patient was started on chemotherapy using CHOP (cyclophosphamide, adriamycin, oncovin and prednisone) in addition to Rituximab. Three days later the patient developed a spontaneous left colonic perforation and underwent a left hemicolectomy with end-ostomy. After a complicated post-operative course, the patient was transferred to a long term care facility for comfort care.

Conclusion: In patients with inflammatory or hematologic disorders, systemic amyloidosis may lead to a myriad of gastrointestinal disorders. This includes malabsorption, delayed motility, intestinal ischemia, and luminal obstruction. Although amyloid deposits may regress with treatment of the underlying disorder, the outcome in patients with hematologic malignancies remains poor.