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Rare Case Of Acute Pancreatitis Revealing Multiple Myeloma : Case Report And Review Of Literature

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Abstract :
Hypercalcemia is a rare cause of acute pancreatitis ; it is in most cases due to primary hyperparathyroidism. Hypercalcemia of metastatic tumors or multiple myeloma is an atypical etiology of acute pancreatitis. We report a case of acute pancreatitis revealing multiple myeloma by emphasizing the interest of imaging and biological parameters in diagnostic orientation.
Case :
This is a 65-year-old patient admitted to the emergency for acute pancreatitis, in whom the abdominal CT scan showed and enlargement of pancreas pancreas and multiple vertebral osteolytic lesions. The etiological investigations showed major hypercalcemia and high serum protein . A bone marrow biopsy revealed infiltration of 25% plasma B cells confirming the diagnosis of multiple myeloma . The patient received specific treatment for hypercalcemia ; however she died following a multi-organ failure before she began chemotherapy for multiple myeloma.
Conclusion :
This case exhibits an interesting presentation of multiple myeloma as acute pancreatitis. It should be considered in patients with multiple myeloma who develop vomiting and upper abdominal pain .
Key Words : *Acute pancreatitis – Multiple myeloma – Hypercalcemia*

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I. Introduction :

The etiology for acute pancreatitis is mainly biliary or alcoholism [1] . Although the association of acute pancreatitis with hypercalcemia of various etiology is well established [2], it remains rare . We report an interesting case of acute pancreatitis secondary to hypercalcemia of multiple myeloma . In literature, only three other cases have been published . We report a fourth one by showcasing the interest of biological parameters and imaging in diagnostic orientation and discussing it clinical impact .

II. Case :

We report 65 -year-old female without any medical history or medication ; presented to the emergency department with epigastric pain and vomiting . The symptoms had worsened over the last week . The patient also reported generalized weakness. She was conscious with body mass Index of 28. She was febrile with blood pressure 10/06 mm Hg, pulse 102 /min , and skin turgor . There was marked epigastric tenderness without abdominal distension. No lymphadenopathy or hepatosplenomegaly was noted nor any symptom of a neurologic deficit**.**

His lipase level was five time upper limit . The white blood cell count was 15,200 and haemoglobin was 10,7 g/dL . Serum calcium , calciuria and serum protein levels were high at 140 mg/l (corrected), 131 mg/24h , and 90 mg/l , respectively with a normal parathyroid hormone level. There was a rise of serum creatinine to 15mg/l . Liver function tests , lipid profile and the serum parathyroid hormone level were normal .

An abdominal ultrasound showed normal liver texture, a bulky pancreas, a normal walled gallbladder and had not found any hepatobiliary ductal gallstone. CT Scan was performed in the third day after the onset of pain , it shows an enlargement of pancreas without any pancreatic necrosis (Balthazard stage B , CTSI : 1) , and multiple lytic lesion in spine . **(Figure1).**

The existence of such lesions associated with biological disorders has oriented the etiological assessment of hypercalcemia towards a mechanism of bone demineralization. Serum protein electrophoresis showed an M-protein in the β2 and gamma regions **(Figure 2)** ; and a bone marrow biopsy revealed clusters of plasma cells; and 35% of plasma cells infiltrating the marrow.

For her the management of her acut pancreatitis we interrupted oral feeding until. Analgesic treatment and rehydration with ringer's lactate 5mg/kg were also started.

We started treatment with bisphosphonates and corticosteroid, once the diagnosis of hypercalcemia was made . According to CRAB criteria she was a candidate for chemotherapy treatment of her multiple myeloma. The evolution was marked the persistence of pain despite a well-conducted analgesic treatment, as well as hypercalcemia. Moreover she installed a cerebral confusion. Brain CT- scan showed multiple "punched out " lytic lesion in the skull (**Figure 3**) without any brain lesion , she was shifted to the ICU where she died following a multi-organ failure .

III. Discussion :

We herein describe a case of an elderly female who presented with a clinical picture of acute pancreatitis with reanal failure and hypercalcemia . On further evaluation , multiple myeloma was diagnosed as the underlying cause of her myriad of problems . This presentation was similar to the previous three cases reported in the literature [1,2,3], where the pancreatitis was the main clinical manifestation, and later a multiple myeloma was revealed .

The most common causes of AP are gallstones (40 – 70 %) and alcohol (25 – 35 %) [4]. Our patient was not alcoholic , also liver function tests were normal , and abdominal ultrasoud and CT- scan did not reveal any lesion or obstruction in common bile duct and gall bladder .

Several drugs can clearly cause pancreatitis. The most commons are azathioprine, 6-mercaptopurine, sulfonamides, tetracyclines, valproic acid, and 5-aminosalicylic acid . Although some chemotherapy drugs (bortezomib and vincristine, doxorubicin and dexamethasone) used in the treatment of multiple myeloma could induce pancreatitis [4]. Hypertriglyceridemia can cause AP for only 1 – 4 % of cases [4].Our patient was not under any treatment. She also had a normal lipid profil . Thus, the possibility of biliary pancreatitis , hypertriglyceridemia or drug-induced pancreatitis was unlikely in our case .

The association of acute pancreatitis with hypercalcemia of hyperparathyroidism is well established, Its incidence ranges between 7% and 12% [5] . In the present case, we ruled out hypercalcemia of hyperparathyroidism since the serum parathyroid hormone level was normal.

Pancreatitis secondary to the hypercalcemia of metastatic tumors or multiple myeloma are extremely rare. Only three cases were reported. The diagnosis of pancreatitis was made on the postmortem examination [6,7]. Meltzer et al described a patient with multiple myeloma with hypercalcemia and abdominal pain ; the diagnosis of pancreatitis was also made on the autopsy[8].

Kyoo Hyung Lee, reported a case of an acute pancreatitis occurring in a patient newly diagnosed with multiple myeloma. In whom other etiologic factors for pancreatitis were excluded.The patient received chemotherapy for multiple myeloma , abdominal pain and vomiting improved under IV hydration and, IV furosemide 20 mg every 2 hours for 6 days and parenteral hyperalimentation . [9]

Hypercalcemia triggered acute pancreatitis probably by the deposition of intraductal calculi in the pancreas, favoring to ductal obstruction . Further , the high concentration of calcium ions in pancreatic secretion and pancreatic tissue could favors activation of trypsinogen to trypsin [9]

Although we could explain the clinical finding by hypercalcemia of multiple myeloma ; we can´t exclude infiltration of the pancreas by the myeloma itself . Extramedullary spread of multiple myeloma to the pancreas can occur in two ways : Extension from skeletal tumor or hematogenous metastatic spread. However it remains rare , gastrointestinal involvement is seen in about 10% of cases, wherein 2.3% of the cases had pancreatic involvement in per autopsy reports[3]. The most cases of plasma cell infiltration are microscopic . Endoscopic ultrasound guided - fine needle aspiration cytology (EUS-FNAC) is one of the best modalities to diagnose such cases .

CT-scan is useful to confim the diagnosis of AP. It provides 90 % sensitivity and specificity for the diagnosis of AP . Still , routine use of CT-Scan in patients with AP is unwarranted, when the diagnosis is apparen. It´s recommended assessing local complications [4]. Our patient was febrile and unable to begin oral feeding over the 4th day , CT-Scan was performed to look for necrosis in the pancreas and the presence of any collections in the abdomen . It also helped in finding the etiology for this patient given the osteolytic lesions found in spin .

IV. Conclusion :

This case exhibit an unexpected presentation of multiple myeloma as acute pancreatitis , highlighting the myriad of clinical manifestations that can be associated with this pathology and showcasing the role of biological and radiological exams in the diagnosis .

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Figures :

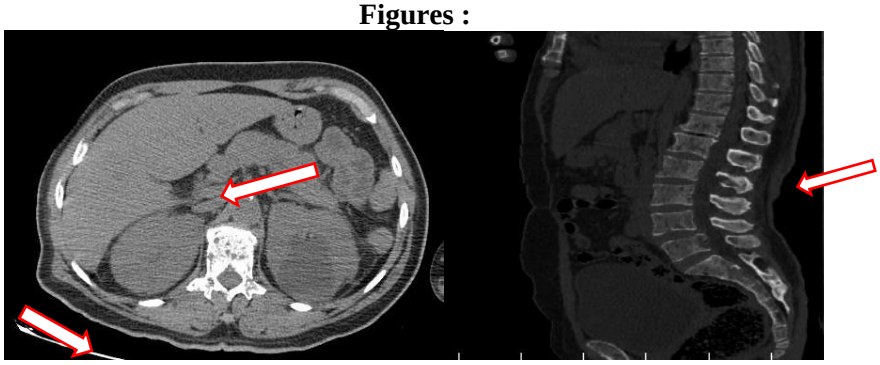

Figure 1A **Figure 1B**

Figure 1 : CT Scan showcasing acute pancreatitis, and multiple lytic lesion in spine

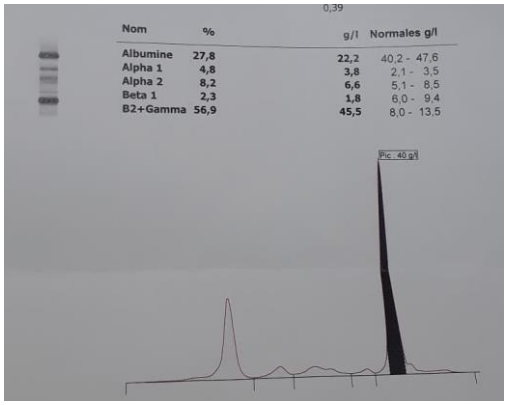

Figure 2 : Serum protein electrophoresis showed an M-protein in the B2 and gamma region

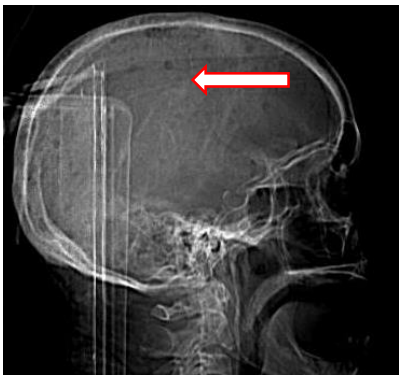

Figure 3 : Imaging of the skull has revealed multiple "punched out " lytic lesion