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Case Report

Multiple myeloma presenting with acute pancreatitis and renal failure

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ABSTRACT

Multiple myeloma is the disease of elderly male with medullary and extramedullary involvement. Pancreatitis is rarely reported in such cases. Here, we report a case of an elderly male who presented with a clinical picture of acute pancreatitis and when investigated, was found to have multiple myeloma and acute renal failure. He was further evaluated for various causes of pancreatitis and was finally concluded to have a possibility of a direct invasion of pancreas by plasma cells.

Key words: *Multiple myeloma, Acute renal failure*

According to the literature available, very few cases of pancreatitis in association multiple myeloma have been reported [1-5]. The most common cause being hypercalcemia induced pancreatitis and incidence of hypercalcemia is about (30%) in cases of multiple myeloma. Others causes include drug-induced pancreatitis and direct invasion by malignant cells.

CASE REPORT

A 68-year-old male presented to our hospital with complaints of pain abdomen, nausea, and vomiting for 10 days. He also had a history of low backache for the past 3 months and easy fatigability, and progressive weight loss of approximately 15 kg in the past 1 year. In the past, patient drank alcohol occasionally and stopped 5 years ago. On general physical examination, the patient was emaciated (body mass index - 16.8 kg/m^2), and pallor was present. On per abdominal examination, tenderness was present in the epigastric region, flanks were full and shifting dullness was present.

His routine laboratory investigations revealed hemoglobin of 7.2 g/dl, total leukocytes count 9200/mm^3, platelet count of 1,08,000/ml blood urea of 84 mg/dl and serum creatinine of 3.7 mg/dl. Erythrocyte sedimentation rate was 112 mm at the end of 1st h, and peripheral smear examination showed microcytic, hypochromic picture with mild anisocytosis and poikilocytosis. Blood and urine cultures were sterile. His total serum protein was 13.82 g/dl with serum albumin of 2.26 g/dl, serum globulin of 11.56 g/dl, and A: G ratio of 0.20. His serum calcium was 6.7 mg/dl, and serum triglyceride level was 67.8 mg/dl. His blood sugar, liver enzymes, and bilirubin were all within the normal ranges. Viral markers including HBsAg, anti-hepatitis C virus, and human immunodeficiency virus were also non-reactive. He had elevated levels of serum amylase (1954 U/L, normal: 23-88 U/L) and serum lipase (217.7 U/L, normal: <60 U/L).

Ultrasonography abdomen and kidney ureter bladder (KUB) showed altered echotexture of normal sized pancreas suggesting acute pancreatitis. Contrast enhanced computed tomography (CECT) of abdomen showed evidence of bulky tail of pancreas with evidence of multiple hypodense ill-defined lesions in body and tail of pancreas suggestive of necrosis (<30%). There were peripancreatic fluid and necrosis with evidence of diffuse mesenteric fat stranding and moderate ascites. Common bile duct and gall bladder were normal. On the basis of these features, diagnosis of acute necrotizing pancreatitis was made with modified CT severity index score of 8 (pancreatic inflammation: 4 points, pancreatic necrosis <30%: 2 points, and extrapancreatic complications: 2 points). The patient was managed conservatively, with parenteral nutrition, and intravenous fluids and was maintained nil orally till pain abdomen subsided with supportive therapy, his renal functions improved.

His X-ray lumbosacral spine showed generalized osteoporotic changes with collapse of L3 vertebra. X-ray skull and pelvis also showed osteoporotic changes. His urine examination was negative for bence jones proteins. Serum electrophoresis showed: Total protein - 9.2 g/dl, albumin - 1.55 g/dl, α1 proteins - 0.24 g (normal), α2 globulin - 0.49 g (low), β globulin - 0.40 g (low), γ globulin - 6.53 g (high), and M spike were detected: 5.88 g/dl. However, immunoelectrophoresis and flow cytometry could not be performed due to its unavailability in our setup and cost constraints. Bone marrow biopsy showed plasma cell infiltration (30% plasma cells) suggestive of multiple myeloma. Patient satisfied the diagnostic criteria of multiple myeloma [1], and chemotherapy was planned; hence, was being referred to higher oncological center. Despite the best efforts patient succumbed to his illness and the chemotherapy could not be initiated (Fig. 1).

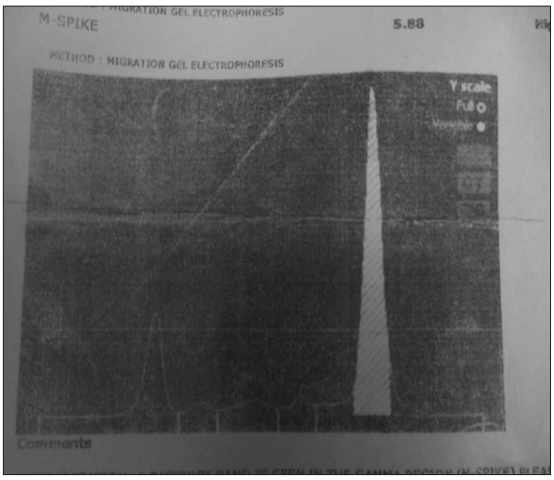

Figure 1: Serum protein electrophoresis showing M spike

DISCUSSION

Our patient presented with a clinical picture of acute pancreatitis of 10 days duration. On further evaluation, he was diagnosed to have concurrent multiple myeloma. This presentation was similar to the cases reported in literature previously, where pancreatitis was the first clinical manifestation, and later on multiple myeloma was detected. Pancreatitis has been rarely reported in association with multiple myeloma, and in most of the cases, the cited reason was hypercalcemia [2,3], initiation of chemotherapy [4,5], or direct infiltration of pancreas by plasma cells [6,7]. Multiple myeloma is usually associated with hypercalcemia and incidence of hypercalcemia is about 30% in cases of multiple myeloma [8]. However, our patient's serum calcium and triglyceride were within the normal range, so hypercalcemia induced pancreatitis was ruled out.

Several drugs can cause pancreatitis, and the common drugs are azathioprine, 6-mercaptopurine, sulfonamides, tetracyclines, valproic acid, and 5-aminosalicylic acid [1]. In cases with multiple myeloma, the drugs which were reported to cause pancreatitis are bortezomib and vincristine, doxorubicin and dexamethasone combination chemotherapy [4,5]. Bortezomib is a selective and reversible proteasome inhibitor, which is used for the treatment of multiple myeloma. Our patient was a reformed alcoholic and stopped drinking 5 years back, so possibility of alcoholic pancreatitis was ruled out. Detailed drug history of the patient was taken, he denied taking any medications in the past and was never given any chemotherapeutic drug for multiple myeloma. Thus, the possibility of drug-induced pancreatitis was unlikely in our case. Ultrasound and CECT abdomen also did not reveal any lesion or obstruction in common bile duct and gall bladder.

Multiple myeloma is a disease characterized by malignant proliferation of plasma cells. Although it typically involves medullary bones, extramedullary presentation also occurs in about 3-4% of cases, more common in males [6]. Extramedullary spread of multiple myeloma can occur in two ways that are either direct extension from skeletal tumor or hematogenous metastatic spread and is associated with poor prognosis [9,10]. 80-90% of these extramedullary tumors usually develop in head and neck area, but gastrointestinal involvement is seen in about 10% of cases, wherein 2.3% of the cases had pancreatic involvement as per autopsy reports. The most cases of plasma cell infiltration are microscopic with very few presenting with well-defined masses [6]. Endoscopic ultrasound guided - fine needle aspiration cytology (EUS-FNAC) is one of the best modalities to diagnose such cases. In our case, CECT abdomen showed evidence of ill-defined hypoechoic lesions in the body and tail of pancreas; therefore, possibility of direct invasion of pancreas by plasma cells can be suspected. However, confirmation could not be done due to lack of availability/expertise of EUS-FNAC and CT guided biopsy in our setup.

Renal failure is seen in almost 25% of the cases of multiple myeloma; although the most common cause of renal involvement in such cases is hypercalcemia, others being glomerular deposits, recurrent infections and use of nephrotoxic drugs. Prerenal azotemia with raised blood urea nitrogen levels has also been reported with acute pancreatitis, resulting from loss of plasma into retroperitoneal space and peritoneal cavity [1]. Therefore, renal impairment in our case could have been multifactorial.

CONCLUSION

This case highlights the myriad of clinical manifestations that can be associated with multiple myeloma. It is essential to evaluate a patient with multiple myeloma for other extramedullary manifestation as they are associated with poor prognosis and outcomes.

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