Abdominal and Scrotal Mass Associated with Calcium Replacement Therapy in L-Asparaginase-Induced Pancreatitis

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ABSTRACT

Although it has many causes in adults; such as alcohol, cholelithiasis, hyperparathyroidism, drugs, and trauma, acute pancreatitis is an uncommon disease at childhood period. Pancreatitis, resulting from any reason, lead to autodigestion of this organ. Pancreatitis following administration of asparaginase has been reported for over 30 years, treating these patients is a dilemma. Here, reporting an 8–years-old patient with abdominal and scrotal mass due to calcium replacement in L-asparaginase induced pancreatitis, we want to emphasize that sometimes, correction of plasma calcium levels with replacement therapy, would be problematic, as calcium will deposite in necrotic tissue of pancreas and omentum, mimicking a mass.

Key words: *Calcium therapy, L- asparagines, Pancreatitis*

INTRODUCTION

Asparaginase toxicity is varied and ranges from acute hypersensitivity and hyperglycemia to hepatocellular dysfunction and acute pancreatitis.[1] Pancreatitis has been noted to be a complication in 2-16% of patients undergoing L-asparaginase treatment for a variety of pediatric neoplasms [1]. Most of cases associated with L-asparaginase-induced pancreatitis are self-limiting that treatment with nasogastric decompression, and parenteral nutrition provide good results, but in rare cases hemorrhagic pancreatitis may occur.[2, 3]

CASE REPORT

An 8-year-old boy was diagnosed as T-ALL. Therapy according to BFM-95 protocol, consisting of intravenous vincristine, daunomycin, L-asparaginase, prednisone, and cytarabine, was started. Complete remission was provided

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after 2 weeks of therapy. At the 28th day of therapy, he suffered from epigastric pain with nausea and vomiting. Gastroendoscopic examination revealed an antral ulcer and duodenitis. First ranitidine and after than lansoprosol treatments were started. At the 33rd day of protocol, L-asparaginase was given for the eighth time due to regimen (E.coli asparaginase, 5000 U/m^2 IV). After treatment, the patient deteriorated, abdominal pain and vomiting began. Physical examination revealed a tender epigastrium, Cullen and Gray Turner signs were noticed. Laboratory analyses showed white blood cell count (WBC) 1200/mm^3, hemoglobin 10.7 gr/dl, platelet 29000/mm^3, amylase 309 IU/L (N: < 25 IU/L), C-reactive protein 3 mg/dl (N: < 0.6 mg/dl), AST 112 IU/L (N: < 40 IU/L), ALT 160 IU/L (N: < 40 IU/L), GGT 813 IU/L (N: < 25 IU/L), Ca 7.1 mg/dl (N: 8.5-10.4 mg/dl). Abdominal ultrasonography (USG) showed peripancreatic edema and abdominal CT revealed diffusely increased pancreas dimension without any mass or cyst. Acute pancreatitis was diagnosed. Nasogastric decompression and partial parenteral nutrition were started. Teicoplanin and meropenem treatments for febrile neutropenia and calcium replacement for hypocalcemia, because of tetanic contractions, had to be given (2mg/kg). After 9 days of therapy serum amylase was normalized and serum calcium was increased to 10.5 mg/dl. Abdominal ultrasonography revealed regression in pancreas dimension. The pancreatitis recovered after 10 days and oral feeding was begun, while than hyperemia and tenderness on the left scrotal skin with a mass 1x1, 5 cm in diameter was seen. The abdominal USG revealed an increased density of adipose tissue which extended to bilateral inguinal canals (Figure 1, 2). Being T- cell leukemia, in order to rule out testis infiltration, biopsy from the mass of the scrotum was performed to reveal the definitive diagnosis. Dystrophic calcification was detected in biopsy which was thought to be as a complication secondary to rapid and intensive calcium replacement. Calcium therapy was stopped and afterwards abdominal and scrotal mass diminished in size and disappeared very slowly, totally after four months. Chemotherapy was interrupted for one month period during pancreatitis. After complete recovery of laboratory values and clinical remission, treatment was restarted without L-asparaginase. He completed his ALL treatment, being still in remission about one year period, developing neither diabetes nor exocrine pancreatic insufficiency.

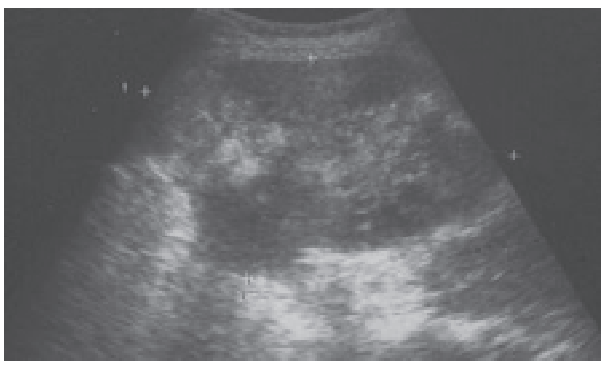

Figure 1: Ultrasound evaluation; a heterogenous well-defined mass was detected in the left anterior pararenal space. The mass was mainly hypoechoic, containing hyperechoic areas. On Doppler US the mass was mainly avascular in nature.

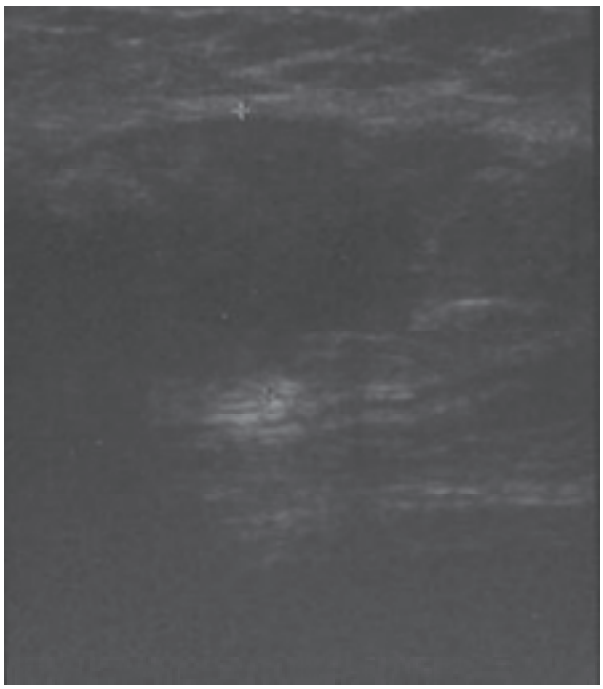

Figure 2: In both inguinal regions, more prominent on the left side similar mass lesions were present.

DISCUSSION

Hypocalcemia is a poor prognostic factor according to Ranson Criteria in patients diagnosed as pancreatitis.[4] Calcium level below 7.5 mg/dl may demonstrate severity of necrosis, exudation and multi organ failure retrospectively.[5] Because cytosolic Ca overload is a crucial feature, inhibition of Ca entry will be important in pancreatitis. Nowadays, enhancements of Ca extrusion or inhibition Ca release from the ER are all attractive targets for development of pharmacological tools for clinical therapy.[6] It has been suggested that, individuals with normal calcium serum levels, likely have a better prognosis when they were diagnosed pancreatitis. However, excess calcium replacement should be avoided due to possibility of accumulation in various sites like abdominal wall and repeated measurements of serum calcium level may prevent such complications.

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