A Rare Case of Methotrexate Induced Pancreatitis in Acute Leukemia Patient.

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Introduction

Acute pancreatitis is recognized with increasing frequency in the pediatric population as a result of trauma, biliary tract disease, viral illness, states of intracranial hypertension and steroids. Children with acute lymphoblastic leukemia (ALL) treated with L-asparaginase represent an additional group at risk to develop pancreatitis. Pancreatitis, which is usually mild and self limited may begin any time during treatment or even up to 16 weeks after discontinuation of treatment with L-asparaginase. Corticosteroids are capable of inducing acute pancreatitis in children. The incidence of clinical pancreatitis seems much higher in patients receiving combination chemotherapy comprising L-asparaginase, corticosteroids and other agents. Methotrexate has never been reported to be linked to pancreatitis. Here we report a case where methotrexate seems to be the most likely agent to have caused acute pancreatitis.

Case Report

A 10 year old girl was diagnosed to have acute lymphatic leukemia. Drugs used during induction were vincristine, daunorubicin, L-asparaginase, intrathecal methotrexate, cyclophosphamide, and oral 6 MP. Child tolerated the chemotherapy well and underwent remission. The induction phase was repeated uneventfully. Subsequently during consolidation phase, the above drugs in addition to cytarabine were used which child tolerated well. Following this child was started on maintenance phase of chemotherapy. During intra venous phase of maintenance therapy she was treated with inj. vincristine, L asparaginase, daunorubicine and oral prednisolone. During oral phase she was treated with oral 6MP and weekly oral methotrexate. During third oral phase of third maintenance regimen, child presented with acute abdomen. On investigation serum amylase level was 2195 iu/l and serum lipase was 930 iu/l. Ultrasound showed bulky pancreas, with isoechoic texture, free fluid in Morrison pouch and pouch of Douglas. She was diagnosed to have acute pancreatitis. Child recovered with conservative management and over four weeks serum amylase, lipase levels were normalized. L-asparaginase was withheld from further treatment. During second oral phase of fifth maintenance cycle, that is at around six months later, again patient developed features of acute pancreatitis. On investigation serum amylase was found to be elevated (549 iu/L). She was managed conservatively and recovered. Methotrexate was withheld from future treatment schedule in addition to L-asparaginase. Other drugs including Vinristine, steroids, daunorubicin and oral 6-MP were continued subsequently during fifth and sixth maintenance regimen without any complications. Child is presently in remission and off chemotherapy for the last one year.

Discussion

Here pancreatitis occurred six months after stopping L-asparaginase, hence it was unlikely to be the cause for pancreatitis. Though steroids can cause pancreatitis, in this child they were used throughout the course without any complications. Daunorubicine and Vinristine could be continued during the course of treatment and are not known to be associated with pancreatitis. From this, it is likely that pancreatitis was due to methotrexate. In the oncology setting, concomitant treatment and the underlying disease make specific attribution of a reaction to methotrexate difficult. Methotrexate has never been reported to be linked to pancreatitis. In a sixteen year old girl with systemic lupus erythematosus acute necrotizing pancreatitis was attributed to a combination regimen involving methotrexate. Another case report of 36 year old woman who received methotrexate along with etoposide, Actinomycin D, cyclophosphamide and Vinristine for suspected hydatiform mole developed acute pancreatitis. We believe this is the first case report of methotrexate induced pancreatitis in an acute leukemic child.

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