CASE REPORT

L-asparaginase induced fatal cortical venous thrombosis in acute lymphoblastic leukemia

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Abstract L-asparaginase has become an integral part in the treatment of acute lymphoblastic leukemia. The major worry of using L-asparaginase is thromboembolism. The case presented here is a 21-year-old lady who developed fatal cortical venous thrombosis during induction phase of treatment for ALL. Early recognition is very important to treat this potentially catastrophic yet treatable complication.

Keywords Acute lymphoblastic leukemia · L-asparaginase · Cortical vein thrombosis

Case

21-year-old Miss. X presented with fever and menorrhagia of 1 month duration. She was diagnosed to have acute lymphoblastic leukemia-L1 phenotype (high risk due to age). Flow cytometric analysis was suggestive of ALL with myeloid coexpression. She was initiated on BFM – 86 protocol. Baseline coagulation parameters including EULQRJHQ37377DQG’3ZHUHQRUPDO6KHUHFHLYHG 3 doses of vincristine, daunorubicin and 3 doses of LASP. Fourth day after 3rd dose of LASP, patient complained of headache. Three days later she developed convulsions. She was stabilized with antiepileptics. MRI of brain was obtained. (Fig 1) Picture was suggestive of cortical venous thrombosis with mass effect and it was confirmed with a MR venogram which revealed lack of opacification of left transverse sinus. (Fig 2). She was given low molecular weight heparin (LMWH) (Fraxiparine) but despite all efforts she had progressive deterioration of neurological status and we lost the patient 11 days later.

Discussion

Patient with ALL are at increased risk of thromboembolic events. The proposed mechanisms include activation of coagulation system, like increased tissue factor activity, or presence of a cancer procoagulant. Iatrogenic causes include use of LASP and steroids. Ulrike etal – reported an incidence as high as 10.4% in BFM (90/95) induction group.where predinsolone is used compared to 1.8% in dexamethasone group used in BFM 2000 [1]. This was suggested due to more pronounced decrease in plasma coagulaton proteins (including fibrinogen and ATIII) in the pred arm due to synergistic effect in lowering hepatic protein synthesis. In patients with pre-existing risk factor for thromboembolic
Fig. 1 MRI brain showing a large infarct in the left temporoparietal region with mass effect