Comments on chronic subdural hematoma in a child with acute myeloid leukemia after leukocytosis

Sir,

We would like to make the following comments about the recently published case report in Indian Journal of Critical Care Medicine.[1]

Role of Acute Leukemia in Bleeding

We would like to refine the authors’ introductory statement as ‘Hemorrhage in acute leukemia may occur due to the underlying disease (thrombocytopenia or hyperleukocytosis due to acute leukemia per se or Disseminated Intravascular Coagulation (DIC) as observed in acute promyelocytic leukemia (APML)), chemotherapy (drugs causing thrombocytopenia and/or DIC), infections (causing sepsis and DIC) and in peritransplant setting (multifactorial).[2] This statement includes the majority of causes of bleeding in acute leukemia. The role of acute leukemia in causing bleeding cannot be underestimated. The conditions associated with bleeding risks in acute leukemia have been reviewed in detail by Kwann HC.[2]

Drugs for Acute Lymphoblastic Leukemia in Acute Myeloid Leukemia

L-asparaginase and prednisolone are used in the induction therapy of acute lymphoblastic leukemia. We find it difficult to understand the reason for mentioning these drugs in a case report pertaining to acute myeloid leukemia (AML). L-asparaginase is associated with bleeding as well as thrombosis. Bleeding (except gastro-duodenal ulcers) due to prednisolone is almost never seen. It can cause thrombosis by increased plasma levels of prothrombin, von Willebrand factor and antithrombin, with decreased plasminogen.[3] References 5-7 cited by the authors do not support their view.

Type of Acute Myeloid Leukemia

The authors mention that the patient was diagnosed as AML. However, there is no clarification on the French-American-British (FAB) type of AML. FAB M3 (APML) is notorious for causing bleeding including Central Nervous System (CNS) bleeds due to DIC.[2] AML M4/M5 subtypes are associated with leukostasis and cause thrombosis and bleeds in CNS.[3]

Leukostasis or Hyperleukocytosis

What were the presenting clinical features in this patient? What was the duration of vomiting, agitation, and seizure? Were the other features of leukostasis present at the time of diagnosis of chronic subdural hematoma? Leukostasis in acute leukemia is diagnosed in a symptomatic patient. Presence of other features of leukostasis will help to label this child with this complication (leukostasis). Patients with AML and White Blood Cells counts more than 100,000/cu.mm may be asymptomatic and are referred to as hyperleukocytosis.

It is surprising that the respiratory complaints, which are seen in leukostasis, are mentioned in the discussion part of the case report and not in the presenting features under the heading of case report.

Coagulation Parameters

In acute leukemia, CNS bleed due to hyperleukocytosis per se is reported to be as low as 3%. [4] Bleeding in acute leukemia is multifactorial. The authors should have mentioned the reports of prothrombin time (PT)/activated partial thromboplastin time (APTT) and plasma fibrinogen to rule out DIC. This becomes more significant as the patient was operated. Authors have not mentioned anything about platelet transfusion. In patients with intracerebral bleeding and during and following neurosurgical procedures, it is recommended to maintain the platelet count >100,000/μL.[5]

We admire the authors’ efforts to evacuate the chronic subdural hematoma in this child. The case report lacks in the description of the associated AML and the necessary steps to be taken prior to neurosurgery in acute leukemia. For a detail and complete case report, we suggest including the views and opinions of the hemato-oncologists looking after the AML component.

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Unrestricted prescription of dabigatran: Is it safe in a resource-limited setting

Sir,

A 71-year-old grossly underweight male (about 52 kg), on dabigatran (110 mg twice daily) for paroxysmal atrial fibrillation, was admitted to our intensive care unit (ICU), with hemorrhagic shock following lower gastrointestinal bleeding. He was bedridden for past 3-months following right middle cerebral artery stroke and was on tracheostomy. He had recently undergone radical cystectomy with ileal conduit for bladder malignancy. His other comorbidities include hypertension and a history of stable coronary artery disease. He was also on aspirin, levetiracetam, and atorvastatin.

He was resuscitated with crystalloids, four units of packed red blood cells, and brief period of noradrenaline infusion. Six units of fresh frozen plasma were also transfused empirically (questionable benefit). Initial laboratory investigations showed hemoglobin: 5.7 g/dl, activated partial thromboplastin time: 49 s, international normalized ratio: 2.3, thrombin time >60 s (report received only after 4 days), and normal platelet count and hypoalbuminemia 2.1 g/dl.

Hemodialysis was planned at the beginning but it took another 6 h in family counseling (and Nephrologist!), to arrange a dialysis machine, and finally start the dialysis (12-h after last dabigatran dose). In the following 24 h he continued to have deranged coagulation parameters and intermittent bleeding per rectum (albeit hemodynamically stable) requiring further transfusion. Colonoscopy showed diverticular disease with large ulcers and active ooze [Figure 1]. At 36-h in the ICU, he suffered cardiac arrest with monitor showing ventricular tachycardia. Repeat electrocardiogram done post-resuscitation showed new ST-segment elevation in anterior leads [Figure 2]. When explained, family refused further treatment and decided to take him home. On a telephonic follow-up 2 weeks after his discharge from the ICU, he was alive with apparently no change in his preadmission cognitive state.

Reversal agent to dabigatran, that is, a neutralizing monoclonal antibody aDabi-Fab is limited to research setting.[1] Some of the measures suggested to stop dabigatran-induced bleeding[2] are either not available in

References