Fatal Intestinal Perforation in a Pediatric Neurosurgical Patient

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Intestinal perforation is rare in neurosurgical population despite risk factors such as surgical stress, concomitant use of nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids, chemotherapy, and infection. We report fatal duodenal perforation in a child and discuss the possible factors contributing to this complication.

A 4-year-old male child weighing 15 kg underwent re-exploration and decompression of a recurrent ependymoma (►**Fig. 1A**). The child received cefazolin 400 mg/day, pantoprazole 10 mg/day, and dexamethasone 8 mg/day during the perioperative period. He was operated earlier for a fourth ventricular anaplastic ependymoma at 1 year and had received 6 cycles of chemotherapy and 31 cycles of radiotherapy.

External ventricular drain was placed in view of neurological deterioration secondary to hydrocephalus on the third postoperative day (POD), along with elective ventilation. The child was started on enteral feeds on the second POD and tolerated the same until seventh POD, when the child developed melena (preceded by a decrease in hemoglobin to 9 g/dL the previous day) and coffee-ground aspiration with abdominal distension and guarding, suggestive of acute abdomen. Neurological status further worsened with subsequent onset of features of septic shock requiring change in antibiotics and noradrenaline infusion. Emergent treatment was started and one unit of red blood cells was transfused. Abdominal radiograph revealed free air under the diaphragm (►**Fig. 1B**). An emergent exploratory laparotomy and primary repair were performed followed by peritoneal lavage for duodenal perforation (►**Fig. 1C**). Bilateral transverse abdominis plane block was performed for postoperative analgesia to avoid opioids and NSAIDs. However, despite continued supportive measures, the child succumbed 30 hours after surgery to multiorgan dysfunction.

A combination of surgical stress, corticosteroid use, and sepsis is the likely cause for duodenal perforation in this previously asymptomatic child. As steroids can mask inflammatory signs of peritonitis, diagnosis should be considered if patient has abdominal discomfort, fever, leucocytosis, and melena or gastrointestinal bleeding with guarding and rigidity of abdomen. Abdominal tenderness, the earliest sign of peritonitis, was masked by the poor neurological status, and dexamethasone further delayed its manifestation, leading to sepsis and poor outcome despite an emergency laparotomy. This emphasizes the need for a daily auscultation for adequate bowel sounds in neurocritical care patients, particularly if on prolonged steroids and have a high index of suspicion of an abdominal emergency in the presence of the above-mentioned signs. Mortality is high (85%) in patients receiving high-dose glucocorticoids.[1] Hence, strategies for prevention (early enteral nutrition, prophylactic H2 receptor antagonists, and judicious use of corticosteroids and NSAIDs by daily scrutiny and de-escalation) and prompt recognition of this complication by assessment of abdominal tenderness and radiograph of the abdomen should be adopted early.

There are very few reports of intestinal perforation in neurosurgical population, mostly subsequent to ventriculoperitoneal shunt surgery.[2] Perforation due to NSAIDs or corticosteroids is extremely rare. One study documented intestinal perforation in 5/719 neurosurgical patients who received steroids as compared with none in 3,749 patients who did not receive steroids. Age > 50 years and pre-existing diverticular disease were identified as risk factors for perforation.[3] In pediatric population, fatal hemorrhage from duodenal perforation 10 days after surgery was described in a 10-year-old child with medulloblastoma receiving high-dose dexamethasone.[4] Authors of a recent report of two cases of brain stem anaplastic ependymomas attribute duodenal ulceration to autonomic dysfunction-induced gut ischemia, gastric hypersecretion, and adrenal cortical hormones-induced decreased gastric mucus secretion.[5] Perforation can complicate 2 to 14% of duodenal ulcers in general population,[6] contributing to approximately one-tenth of all acute abdomen diagnosis in medical intensive care units.[7]

Despite perioperative steroids being routinely used in the perioperative period for brain tumors, literature search reveals that guidelines for optimal treatment duration of steroid therapy are lacking. A recent cohort study of 131 newly diagnosed

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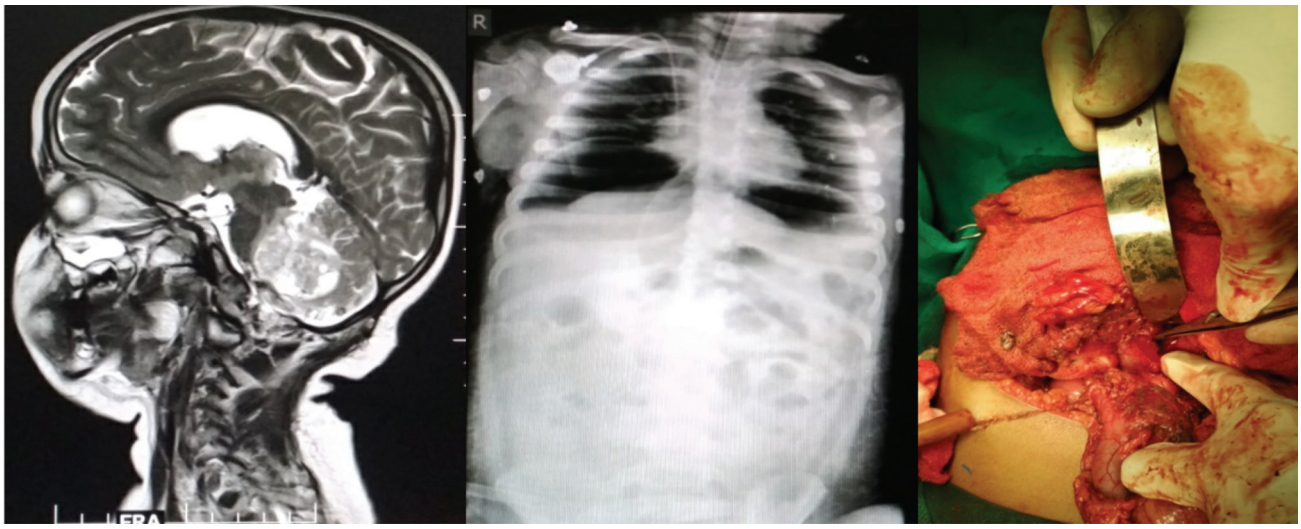

Fig. 1 (**A**) Recurrent posterior fossa tumor; (**B**) Radiographic image of free air under the diaphragm; (**C**) Exploratory laparotomy revealing duodenal perforation.

neurosurgical patients with glioblastoma provides evidence of scope for successful steroid tapering within 2 weeks after surgery with improved survival rates.[8]

To conclude, steroids can mask the symptoms of peritonitis and hence close monitoring and high degree of suspicion are essential. Perioperative steroids should be tapered and discontinued as soon as possible. Safer alternatives to NSAIDs such as paracetamol should be preferred for perioperative analgesia in patients receiving steroids.

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Conflict of Interest
None declared.

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