Post-operative accidental diagnosis of intra-cerebellar astrocytoma in an emergency appendicectomy case

INTRODUCTION

Incidental brain abnormality diagnosed by magnetic resonance imaging (MRI) is 15% among adult research volunteers and 25% in children.[1,2] Central nervous system tumours constitute the second most common types among tumours of childhood with the incidence of 2.5/100,000.[3] Gliomas are the most common type of brain tumour with the mean age of presentation being 9 years.[4] Clinical presentation depends on the size and site of the tumour in addition to the factor which affect the intracranial pressure. We report a case of undetected intra-cerebellar astrocytoma, whose clinical presentation was accelerated by anaesthesia and perioperative clinical factors.

CASE REPORT

A 14-year-old female with no other significant medical or surgical history was admitted to the hospital with a history of abdominal pain, vomiting and fever since 1 day. Computed tomography (CT) scan of abdomen and pelvis revealed a well-defined thick walled collection with air fluid level measuring 5.5 cm × 5 cm in size in right iliac fossa, suggestive of ruptured appendicular abscess with associated hydro pneumoperitoneum. Investigations showed haemoglobin (Hb) of 10 g/dl, total white cell count of 21,600 cells/mm^3 with neutrophils 91%; rest of the investigations were within normal limits.

She was scheduled for emergency laparotomy under general anaesthesia. The patient was pre-medicated with injection glycopyrrolate 0.2 mg, injection midazolam 1 mg, injection pantoprazole 40mg, injection ondansetron 4 mg, all intravenous (IV). Anaesthesia was induced with propofol 50 mg after injection pentazocine 30 mg. Tracheal intubation was facilitated with injection atracurium 25 mg IV. Anaesthesia was maintained with oxygen:nitrous oxide (50:50), 0.8 to 1% isoflurane and intermittent bolus of atracurium. She was ventilated to maintain an end-tidal carbon dioxide value of 30–35 mm Hg. Physiological parameters including blood pressure, electrocardiogram, oxygen saturation, end-tidal carbon

dioxide and airway pressures were stable throughout the procedure. At the end of the surgery, neuromuscular blockade was reversed with injection neostigmine and glycopyrrolate. Trachea was extubated after the return of adequate motor power and consciousness. The patient was moved to post-operative Intensive Care Unit with stable haemodynamics for further monitoring with maintenance normal saline IV infusion at 75 ml/h.

Patient was haemodynamically stable for the first 14 h post-operative period. After this, patient complained of dizziness on semi-recumbent positioning with a systolic blood pressure of 80 mm Hg. A fluid challenge of 150 ml was given and the IV fluid infusion was increased to 150 ml/h for next 2 h; this resolved the postural symptoms, and she was moved to the surgical ward. Next morning, when the patient was made to sit up on bed (semi-recumbent), she had another episode of dizziness. On examination, she was found to be conscious and oriented with normal motor and sensory reflexes and blood pressure of 100/65 mm Hg. Investigations showed Hb of 9.5 g% for and 1 unit of blood was transfused. Next day, when child again had multiple episodes of dizziness, a neurological opinion was sought and a two-dimensional (2D) echocardiography, CT brain with contrast was advised. ENT surgeon advised for vestibular exercise and tablet cinnarizine 25 mg. 2D echocardiography was normal. CT brain showed an ill-defined mass in the left cerebellum with calcification causing midline mass effect on the 4th ventricle with mild hydrocephalus suggesting a differential diagnosis of medulloblastoma [Figure 1a] requiring MRI for further evaluation. Patient was subsequently treated with injection mannitol (initial bolus of 1 g/kg, then 0.5 g/kg, q 6 h up to 48 h) and injection dexamethasone IV 0.5 mg/kg/day, q 6 h. Response to treatment was good. Later MRI showed astrocytoma in the left cerebellum [Figure 1b] for which she was advised decompressive craniotomy and excision of the tumour, but due to financial constraints, patient refused surgery. Patient showed improvement with medical management and was able to ambulate without any symptoms.

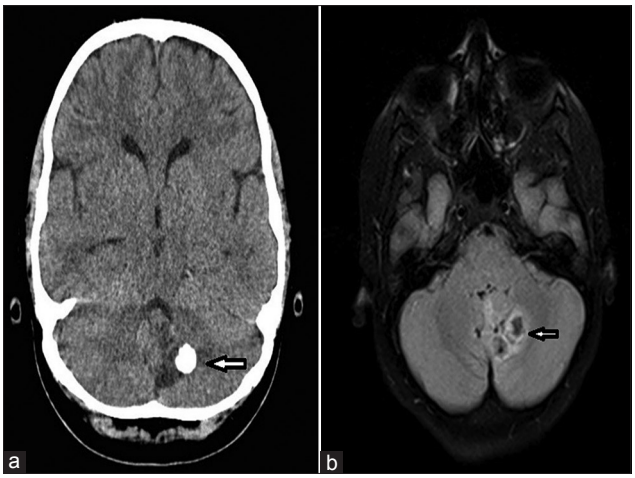

Figure 1: (a) Computed tomography scan of brain showing left cerebellar tumour with calcification. (b) Magnetic resonance imaging scan showing left cerebellar tumour extending into vermis

DISCUSSION

Astrocytoma represents up to 10% of all primary brain tumours and up to 25% of posterior fossa tumours in children, with low-grade gliomas forming the most common of the cerebellar tumours. Cerebellar astrocytomas commonly present with symptoms and signs of raised intracranial pressure due to obstruction of the fourth ventricle and may also be present with cerebellar signs such as ataxia and dysmetria. These tumours occasionally present acutely due to intra-tumoural haemorrhage.[4]

Slow growing tumour initially displaces the cerebrospinal fluid and the blood without causing an increase in the intracranial pressure during which the patient may be symptom-free with normal Glasgow Coma scale. Once this compensatory mechanism is overcome, a small increase in the size of the tumour will lead to compression of solid tissue within the skull and the compliance decreases exponentially with a significant raise in intracranial pressure.[5]

Anaesthesia technique suitable for a healthy individual may be dangerous in the presence of an intracranial tumour. Opioids produce respiratory depression with hypercapnia increasing the cerebral blood flow and consequently increased intracranial pressure. Inhalational agents such as isoflurane are known to increase intracranial pressure by increasing cerebral blood flow.[6,7] Perioperative fluid management may have a significant role in oedema and neurological decompensation. In addition, in the post-operative period pain, supine position, accumulated bronchial secretions or residual paralysis produce inadequate ventilation which can lead to carbon dioxide retention and raise in intracranial pressure.[8]

In any case of prolonged recovery or delayed neurological deficit post-general anaesthesia, a silent brain tumour should be suspected after exclusion

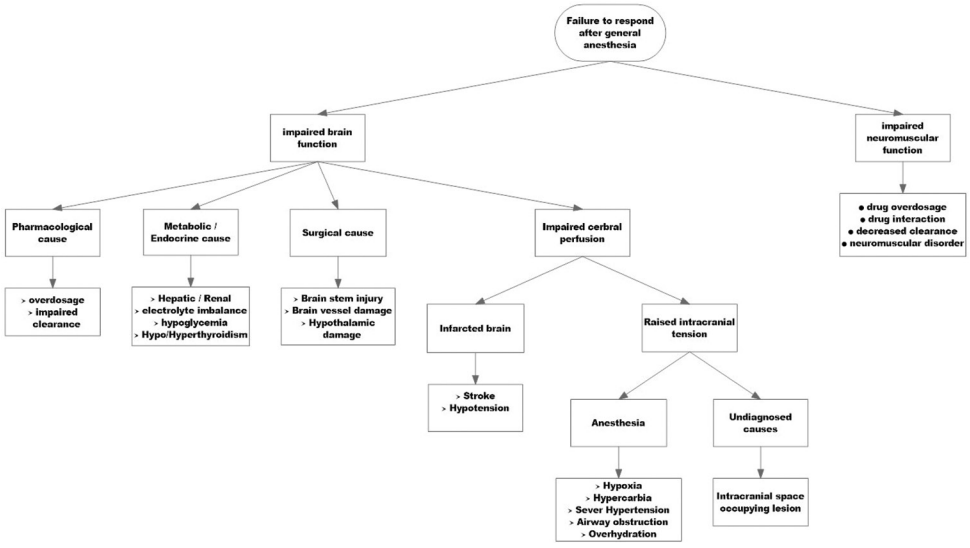

Figure 2: Causes of failure to respond after general anaesthesia

of the most common causes [Figure 2].[9] Brief neurological examination such as pupillary size, reactivity of the limbs to painful stimuli should be performed on every patient. Evaluation of prolonged post-anaesthetic coma should include a CT scan even in the absence of focal signs when no clear cause is evident.

Early administration of anti-oedema therapy with correct fluid balance is suggested in immediate management of post-operative neurological deterioration without clear aetiology. Both mannitol and dexamethasone are definitely beneficial, especially mannitol reducing the intracranial pressure by 26–34% with a dose of 0.5 g/kg with greater reduction with higher doses. Mannitol produces osmotic effect in intact blood-brain barrier and is therefore more likely to reduce cerebral oedema. Betamethasone and dexamethasone decrease secretion-excretion of substances that are probably vasoactive. Even though they do not decrease the brain water, the total intracranial elastance returns within 72 h with the greatest decrease being in the first 24 h.[10]

CONCLUSION

Neurological deficits post-general anaesthesia should always raise the suspicion of underlying intracranial tumour whose manifestations may be accelerated by anaesthesia and perioperative factors. Immediate anti-oedema measures might improve the neurological condition which should be followed by investigation such as CT scan and MRI for both definitive management of patient and for medicolegal purposes.

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Conflicts of interest

There are no conflicts of interest.

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