erature varied from 13.2 grams\textsuperscript{[3]} to 228 grams\textsuperscript{[4]}. The duration of therapy after which neuropathic symptoms developed varied from 11 days\textsuperscript{[3]} to 6 months\textsuperscript{[4]}. The 3 days of latency and 3.6 g of cumulative dose observed in our patient are the smallest reported so far. It is noteworthy that, in the case reports from India\textsuperscript{[3,5,6]}, the cumulative dose of metronidazole was low (13.2-18 grams) and the latency to symptom onset very short (11 days to 18 days) when compared to patients from the West. This may reflect a genetic susceptibility to the neurotoxic effects of metronidazole or a genetic variation in the metabolism of metronidazole in Indian patients.

To conclude, we report an unusually rapid development of peripheral neuropathy after starting metronidazole. Its early recognition and rapid withdrawal of the drug are important, as the neuropathy can be disabling and persistent.

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Idiopathic primary pan intraventricular hemorrhage in a child

Sir,

Primary intraventricular hemorrhage (PIVH) is defined as hemorrhage restricted to the ventricular system without apparent brain parenchymal involvement. Idiopathic PIVH (IPIVH, whereupon no clear evidence of a source is found) has been reported in only six cases, all of whom were adults\textsuperscript{[1,2]}. We present the case of a 7-year-old female with IPIVH, with pertinent discussion.

With a past history of mild headaches over 6 months, this girl awoke with a severe headache and nausea, becoming subsequently unresponsive (with no apparent seizure activity noted). She was rushed to an adjacent hospital where a computed tomography (CT) scan of the head revealed a pan-intraventricular hemorrhage with ventricular dilatation [Figure 1]. An external ventricular drain (EVD) was placed with an extremely high opening pressure and the CSF was drained continuously. The patient improved post-operatively and over the next 24 hours was able to interact with her parents and follow some commands. A post-EVD placement CT revealed no new bleed, adequate EVD placement, and a right basal ganglia hypodensity. Approximately 36 hours post-presentation, she became acutely unresponsive to central stimulation with fixed and dilated pupils, with the EVD still functional and draining bloody fluid. The patient’s pupils became fixed and dilated, and she was unresponsive to deep central stimulation. No significant vital sign change was noted leading up to the event, and the phenytoin level was slightly supratherapeutic, with no evidence of coagulopathy. Subsequent CT over 8 hours showed no new findings. No seizure

Figure 1: CT scan (non-contrast) of the head on admission

Figure 2: Autopsy findings. Gross specimens showing herniation with small amount of subarachnoid blood
activity was ever noted, and the patient never improved. After failing apnea and nuclear medicine blood flow studies, the patient was pronounced brain dead and support withdrawn. At autopsy, findings were consistent with herniation [Figure 2] and histopathology revealed no underlying vascular malformation, tumor, or other pathology.

“Spontaneous” PIVH describes all non-traumatic cases of PIVH and includes hemorrhage related to entities such as vascular malformations, vascular tumors, moyamoya disease, aneurysms, fibromuscular dysplasia, stroke, coagulopathy, and hypertension, along with some idiopathic cases. Survival has ranged from 40-100% according to various series.[3,4] Idiopathic hemorrhagic stroke has been described in 46% of young patients.[5] Other than EVD placement, few guidelines exist for treatment.

The causes of the PIVH and subsequent deterioration remain unclear. An intracranial pressure wave, subsequent hemorrhagic stroke and “suicidal vascular malformation” are all possible but unlikely. The cause of the PIVH in our case remains a mystery in spite of an autopsy.

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Spinal intramedullary metastasis from intracranial germinoma

Sir,

Germinomas in the central nervous system (CNS) usually occur in the pineal or the suprasellar regions; less frequent sites include the thalamus, basal ganglia and cerebellar vermis. Metastases to the spine from brain tumors, ‘drop metastases’, are usually intradural extramedullary masses. However, they rarely present as intramedullary tumors.[1]

A 30-year-old female with amenorrhea for 1.5 years and galactorrhea for 6 months had a magnetic resonance image (MRI) of the brain that showed an 8 mm enhancing lesion in the pituitary stalk and hypothalamus [Figure 1A]. Her hormonal tests were suggestive of panhypopituitarism and she was given thyroid and steroid replacements. The possibilities of a hypothalamic hamartoma or granuloma were considered. She had been started on antituberculosis therapy (ATT) elsewhere. After a discussion with the patient and family regarding the risks and benefits of surgery in obtaining a diagnosis, it was decided to follow her up with a repeat image after 6 months and the ATT was continued. Six months later, she had paraesthesiae in both upper limbs, progressive weakness in all four limbs and difficulty in walking. Her gait was spastic but she could walk unaided. An MRI of the cervical spine and brain showed an intramedullary lesion from C1 to C6 levels [Figure 1B] and slight increase in the size of suprasellar lesion. Cerebrospinal fluid (CSF) analysis showed a total count of 18/cumm, protein 47 mg%, and sugar 55 mg%. CSF beta-hCG was 67 mIU/ml (normal levels 0.0–5.0 mIU/ml) and alpha-fetoprotein was <2 mIU/ml (normal level 0.0–5.5 mIU/ml). She underwent a C1–C7 laminectomy and subtotal excision of the intramedullary tumor. At surgery, there was an exophytic component of the tumor at C2 level and there were areas of necrosis and haemorrhage with a poor tumor–cord interface.

Microscopic examination revealed sheets and alveolar clusters of large polygonal cells with abundant eosinophilic to clear cytoplasm and central, round mitotically active nuclei containing prominent eosinophilic nucleoli [Figure 2]. Immunohistochemical staining was positive for placental alkaline phosphatase and negative for cytokeratin, S100 protein and CD30. A histological diagnosis of a germinoma was made. She underwent three cycles of combination chemotherapy with ‘cisplatin’, etoposide and bleomycin followed by craniospinal irradiation (Cobalt 60). A total dose of 36 Gy in 18 fractions was delivered using two parallel opposing fields for the cranial and direct posterior field for the spine. In addition 14 Gy in seven fractions was given as a boost to the cranial and spinal lesion. Contrast enhanced cranial and spinal MRI,