Calcified occipital glioblastoma

Sir,

A rare case of a glioblastoma having areas of calcification is reported. The relevant literature is briefly reviewed.

An 18-year-old boy was admitted with a one-month history of bifrontal headache, intermittent vomiting and diplopia. He had history of seizures since the age of four years. The seizures were not preceded by any aura and the ictus consisted of a blank expression lasting for a few seconds. His scholastic performance had been average. Clinical examination revealed bilateral abducens paresis and mild papilledema. There was right-sided homonymous hemianopia. There was no cognitive deficit, disconnection syndrome or motor weakness. Skull radiograph and CT (Figure 1) done three years earlier had revealed calcification at the left occipital pole, without any mass effect. CT done at the time of present admission showed a large, mixed, attenuating and partly enhancing lesion at the left occipital pole having areas of calcification.

Left occipital craniotomy and a radical resection of the tumor-bearing occipital pole was carried out. The tumor was firm, solid and gritty and was not very vascular. The postoperative period was uneventful. Histopathology revealed a highly cellular tumor made up of anaplastic and pleomorphic cells with several mitotic figures and bizarre giant cells. Astrocytic proliferation was seen in the peripheral areas. There were areas of necrosis with palisading of nuclei and angioendothelial proliferation. Many areas of calcification were seen. The tumor was positive for glial fibrillary acid protein (GFAP). A diagnosis of glioblastoma multiforme with calcification was made. The lesion was then subjected to radiotherapy and chemotherapy. The patient was free from recurrence for three years after surgery, after which he was lost to follow-up.

Calcification may be seen in gliomas, especially in oligodendrogliomas and in mixed gliomas that have a benign histological appearance. It is unusual in high grade astrocytomas and glioblastomas; it is likely that some part of the previously low grade tumor may dedifferentiate into glioblastoma. Histological markers suggesting a better prognosis in glioblastomas include presence of giant cells and differentiation. The presence of calcium deposits has rarely been recorded.

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References

Acute inflammatory demyelinating polyneuropathy following plasmodium vivax malaria

Sir,

Acute Inflammatory Demyelinating Polyneuropathy (AIDP), seen following viral, bacterial infections or immunization, is uncommon following parasitic infection. We could locate 11 cases of Guillain Barre Syndrome (GBS) following malarial illness from the literature. Eight of these cases were following P. Falciparum infection and three were following P. Vivax infection. We report a case of AIDP / GBS following P. Vivax malaria that needed ventilatory support.

A 39-year-old male developed fever with chill and rigor. His hematological examination showed ring forms of P. vivax. He was treated with chloroquine (total 1500mg base) and pri-
maquine (total 210 mg). He became asymptomatic in about five days. On the eleventh day following onset of fever, the patient developed tingling sensations in both the lower limbs from the feet up to the knees and both hands. The next day he noticed weakness of both the lower limbs, he could not get up from the squatting position and could not climb stairs. In the night he developed retention of urine. On the following day he was bed-bound and could not move his fingers adequately and could not raise his arm above the bed. He was admitted to the hospital where he developed difficulty in speaking, swallowing and had nasal regurgitation. He was not dyspnoeic at that stage. There was no history of any other illness preceding or following malaria and there was no history of recent immunization.

On examination, his speech volume was low. There were bilateral VII, IX and X nerve palsies. Secretion accumulated in the throat. There was hypotonia in all the muscles of all four limbs. Muscle power was 2/5 in the upper limbs and 0/5 in the lower limbs. The sensory system was normal. Superficial reflexes were normal and all deep tendon jerks were absent. Chest expansion was 5 cm on deep inspiration and single breath count was 26. Chest examination was normal. Peripheral smear examination showed no malarial parasite. Falciparum antigen test was negative and urinary porphobilinogen was absent. CSF study revealed: cells –3/cmm (all lymphocytes), sugar 78mg%, protein 208 mg%. Conduction studies in both median, ulnar, peroneal and posterior tribal nerves revealed gross reduction in motor nerve conduction velocity (MNCV) and CMAP amplitude in all the nerves; distal latency grossly prolonged in all the nerves; F waves absent in all the nerves; temporal dispersion seen in all four limbs; H-reflexes absent in both sides; and SNAP absent in both median, ulnar and sural nerves.

The weakness progressed and the patient developed respiratory difficulty. He was ventilated. He was given IV immunoglobulin 24g/day for five days. The patient started improving 2 days after IVIG was completed and could be weaned off the ventilator after one week. He went home after about one month and could walk unaided.

AIDP / GBS following malaria is rare. It is important to rule out other neurological syndromes that may be unmasked by a febrile episode. A review of 12 cases of GBS (11 previously reported and the present one) revealed that eight patients had preceding falciparum malaria and four had vivax infections. All but three patients (including the present one) had distal symmetric sensory deficits. Paralysis was mild in seven cases (three due to P. Vivax and four due to P. Falciparum) and recovered completely in 2-6 weeks without any specific treatment. Four patients with falciparum malaria developed severe paralysis with respiratory failure, and three patients died. This appears to be the first case report of severe GBS following P. Vivax malaria requiring ventilatory support and IVIG therapy.

The pathogenesis of GBS following malaria infection is not known. This is likely to be immunogenic like that occurring after viral or bacterial infections. Other mechanisms suggested for the development of polyneuropathy following a parasitic infection include parasitic emboli obstructing vasa nervosum, release of neurotoxins, associated metabolic and nutritional disturbance, immune-mediated capillary damage, release of free radicals and tumor necrosis factor.²

A rare cause for mononeuritis multiplex

Sir,

Paraneoplastic peripheral neuropathy is a well-described entity.¹ Amongst the various manifestations, mononeuritis multiplex is relatively unusual and is usually associated with hematological malignancies.

A 72-year-old man presented with eight months history of progressive symmetric burning paraesthesias over both legs. Later he noticed tingling paraesthesias over the dorsum of the right hand. Four months later he noticed weakness in the left foot in wearing slippers and clearing the ground, which gradually worsened over two months. After one month, he noticed weakness of the right hand, with difficulty in writing, holding objects and buttoning his shirt. Eight months back he developed edema of the feet. He was not a known hypertensive or diabetic. On examination, he had bilateral pedal edema and non-tender hepatomegaly extending 2 cm from the costal margin. There were no hypesthesic patches. In the right upper limb, he had clawing of the medial two fingers, wasting of first dorsal interosseous muscle and weakness of the adductor pollicis, interossei, lumbricales and opponens digitii minimi. In the left leg he had weakness of the dorsiflexors of the ankle, evertors of subtalar joints and extensors of toes. Both ankle jerks were absent. He had sensory loss over the right little finger and medial aspect of the palm, the right medial forearm, and the lateral aspect of the left leg and dor-

References