are highly effective anticonvulsants against seizures induced in animals by a variety of mechanisms including kindling. Adenine receptor antagonists exert protective actions indicating tonic adenosinergic control of ictal susceptibility. Animal studies have shown a rapid and substantial release of adenosine and its metabolites inosine and hypoxanthine after experimental seizures. The physiological actions and metabolism of adenosine in the human CNS remain to be fully elucidated. We report that acute increases of adenosine and its metabolites can be detected in lumbar CSF after clinical status epilepticus in humans.

Seven patients with new onset status epilepticus were studied. Five patients (aged 12 to 67) developed generalised tonic-clonic seizures with three or more clonic or tonic-clonic seizures occurring in less than one hour without regaining consciousness beforehand. Seizures were not preceded by ephelais, dystasia, or adduction of the right face and arm. One patient (82 years old) had a complex partial status epilepticus documented by electroencephalography and was sedated by diazepam withdrawal and drug toxicity.

One patient (48 years old) with diabetes and chronic renal failure developed epilepsia partialis continua (EPC). This patient (aged 22 to 53) evaluated for other neurological conditions (demyelinating neuropathy, headaches, cranial neuropathy, multiple sclerosis) were used as controls.

Cerebrospinal fluid concentrations of adenosine and its metabolites after status epilepticus

<table>
<thead>
<tr>
<th>Seizure Time (h)</th>
<th>Adenosine (µM)</th>
<th>Inosine (µM)</th>
<th>Hypoxanthine (µM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls (n = 11)</td>
<td>0.063 (0.039-0.106)</td>
<td>0.41 (ND-0.79)</td>
<td>2.36 (1.7-4.08)</td>
</tr>
</tbody>
</table>

ND = none detected, GTC = generalised tonic-clonic status epilepticus, EPC = epilepsy partialis continua, CPSE = complex partial status epilepticus, time estimated time from last seizure to sampling of CSF. Patient 7 was sampled during continuous seizure activity. Patient 6 had multiple partial motor seizures and time from last seizure is not available.

Bilateral cavernous sinus thrombosis causing Korsakoff's amnesic syndrome

Korsakoff's amnesic syndrome (Korsakoff's psychosis) is a condition in which learning and memory are affected out of proportion to other cognitive functions. It has not previously been reported as a complication of cavernous sinus thrombosis. Our patient developed Korsakoff's amnesic syndrome after bilateral medial tempo-occipital infarcts as a complication of cavernous sinus thrombosis.

A 44 year old man was admitted with fever of 15 days duration, and altered sensorium of 10 days duration preceded by a day of recurrent vomiting. Three days before admission he developed painful swelling of his left eye associated with redness and sudden total loss of vision in that eye. The fever was high grade, intermittent, and associated with sweating. The altered sensorium was in the form of restlessness, incoherent speech, disorientation, and an inability to recognise his relatives properly. He had hypertension for four years duration, was on irritable treatment, and was a past smoker. He had no history of alcohol intake.

On examination he was drowsy, febrile (99°F), and had a blood pressure of...
134/90 mm Hg. General and systemic examinations were otherwise unremarkable. Examination showed a proposed left eye, circumcorneal congestion, absent perception of light, and direct light reflex with a pale optic disc; findings on the right side were normal except for a sluggish direct light reflex. Other cranial nerves were normal. Motor system examination was unremarkable. A mild neck rigidity was present. The next day the left sided ptosis was more prominent and associated with chemosis, pronounced lid oedema, and severe restriction of all movements of the left eyelid. The intracranial pressure was 40 mm Hg on the left side and 14 mm Hg on the right.

Relevant laboratory data included mild normocytic, normochromic anaemia (Hb-12.1 g/dl) with mild neutrophilia and a normal platelet count with normal concentrations of blood glucose, creatinine, serum urea nitrogen, sodium, and potassium. Liver function tests, prothrombin and activated partial thromboplastin times, blood culture, ECG, chest radiograph, and two dimensional echocardiogram were all normal. A CSF examination showed normal pressure, clear acellular fluid with 77 mg/dl of sugar, and 50 mg/dl of protein.

Plain head CT showed bilateral asymmetric medial temporo-occipital infarctions, the left being larger. Repeat CT two days later with contrast showed enhancement of the cavernous sinus with filling defects and bulging of the lateral margins of both cavernous sinuses (fig 1)—more so on the left, and the infarcts seemed more prominent. The paranasal sinuses were normal.

The patient was treated with cefotaxime and amikacin for the next two weeks, with intravenous mannitol and local application of steroids and atropine for the eyes. Anticoagulants were not given. He became apyrexial with gradual improvement of his sensorium. A third plain CT two weeks after admission showed bilateral haemorrhagic transformation of the infarcts, more prominent in the left medial temporo-occipital area.

A detailed mental status examination three weeks after admission showed that attention was mildly abnormal with mild anoma in naming objects and parts of objects; the remaining language functions were essentially normal. He was disoriented for time, place, and person. Immediate and remote memories were mildly impaired with gross recent memory (both verbal and visual, new learning abilities) impairment. Momentary confabulations were noted with normal constructional abilities. Abstraction was impaired with partially preserved insight and a grossly normal fund of information. Mild impairment of the frontal lobe (inability to do alternating motor patterns) and occipital dysfunction (prosopagnosia and a colour agnosia) were seen. Parietal lobe tests were normal.

Follow up 15 months after discharge showed a persistent confabulatory amnesic state with total blindness of his left eye. The right eye was normal. Visual evoked potentials were normal on the right side but absent on the left. He was unable to return to work or function independently due to the continued severity of his memory impairment.

An MRI at this time showed bilateral well delineated temporal infarcts, which were hypointense in T1 and hyperintense in T2 images (fig 2). There was evidence of bilateral temporal atrophy with dilatation of the temporal horns of the lateral ventricles. The left eyeball was atrophic.

A neuropsychological assessment 18 months after the onset showed that on the Weschler memory scale his raw score was 22 and his Lui weighted score was 64. Overall memory quotient was 69. Functional analysis indicated that disturbances persisted in all the memory functions, with recent and immediate memory the worst.

Korsakoff’s amnesic syndrome after cavernous sinus thrombosis has not been reported before. Damage to three relatively discrete neuroanatomical regions has been primarily implicated in the amnesic syndrome—namely, the medial temporal lobes, the medial thalamic nuclei, and the basal forebrain. Mishkin’s model proposes that the degree of impairment of recognition memory was a direct function of the amount of conjoint damage to the amygdala and hippocampus irrespective of the specific structure involved. In this case the bilateral medial temporo-occipital infarcts also resulted in bilateral hippocampal damage together with amygdalar involvement (fig 2), which is consistent with Mishkin’s model for amnesia. This finding is consistent with the hippocampal abnormalities in amnesic patients as shown by high resolution MRI.

Clinicopathologically this patient had infarctions bilaterally in the territory drained by the basal vein of Rosenthal (BVR). The BVR receives tributaries from the insula, cerebral peduncles, medial temporal lobe, and veins of the temporal horn. It communicates with the cavernous sinus either through the sphenoparietal sinus or through the superior petrosal sinus, via the lateral mesencephalic vein. In instances of hypoplastic posterior segment of the BVR, the vein drains anteriorly via the lateral mesencephalic vein. In this patient the thrombotic process may have extended to the BVRs by any one of these channels or, alternatively, the BVRs may have had hypoplastic posterior segments. The dien-cephalic structures, drained by the internal cerebral veins, were spared in this patient as was the basal forebrain, confirmed by MRI.

Levine et al recommend early anticoagulant treatment in cavernous sinus thrombosis to reduce morbidity, but the spontaneous haemorrhagic conversion as seen in our patient suggests that adequate...
imaging either with CT or MRI to rule out mesial temporal infarcts should be mandatory before giving anticoagulant treatment. We thank Dr K Niranjan Reddy for help in the neuropsychological assessment of the patient.

RUPAM BOROGOHAIN HARI RADHAKRISHNA HAY KUMAR SINGH SURATI MOHANAD Department of Neurology, Nizam’s Institute of Medical Sciences, Punjagutta, Hyderabad—500 482, India

JAGANMOHAN REDDY Department of Imagoiogy

Correspondence to: Dr Rupam Borogohain, Department of Neurology, Nizam's Institute of Medical Sciences, Punjagutta, Hyderabad-500 482, India.


3 Stephenson RB, Stitwell DB. Arteries and veins of the human brain, Springfield, IL: CC Thomas, 1957:


5 Levine SR, Twyman RE, Gilman S. The role of anticoagulation in 24 year old woman presented to the eye casualty department with a week's history of headache—worse on bending—nausea with intermittent vomiting, and visual blurring. She had been taking oral contraceptives but there was no other relevant drug or medical history. Examination showed obesity (95 kg/155 cm), bilateral papilloedema, and normal visual acuity although testing fields by confrontation showed a small central scotoma affecting the right eye and decreased temporal fields in the left eye. Computed tomography, brain MRI, and MR angiography were unremarkable. Puncture produced clear and colourless CSF with an opening pressure of 36 cm CSF. The CSF constituents were normal, with a protein concentration of less than 0.1 g/l and 2 white cells per cubic mm. The CSF glucose to plasma ratio was normal. The following peripheral blood indices were also normal: full blood count, viscosity, urea and electrolytes, liver function tests, thyroid functions, creatine, protein, venereal disease research laboratory test, and anticoagulopathy antibody. OKP-Humphrey field analysis showed pronounced peripheral field constriction. The initial management was withdrawal of the oral contraceptive pill. Diuretic treatment with chlorothalidone was commenced and diuretic referral arranged. These measures initially resulted in considerable symptomatic improvement. However, several months, however, the headaches recurred with increasing visual obscurations. These symptoms progressed to daily bilateral obscurations of vision for up to a minute despite diuretic treatment. In association with this she had noted increasing impairment of hearing in the left ear, with no associated tinnitus, in the week before readmission. Repeat neurological examination on readmission showed chronic papilloedema, with peripheral constriction of the fields but normal visual acuity. Neuro-oto-logical examination confirmed clinical hearing loss in the left ear. The Rinne test was positive bilaterally. Pure tone audiometry showed a mild left sided conductive hearing loss, with an air bone gap ranging between 5 and 20 dB, at 0.5 and 1.0 kHz (250 to 4000 Hz). Tympanometry showed normal middle ear pressure and compliance bilaterally. Repeat lumbar puncture subsequently confirmed raised pressure of 29 cm CSF. On the second day of the above, after the patient noted normalisation of the hearing acuity in the left ear. Repeat audiometry and tympanometry were performed showing resolution of the previously noted mild conductive hearing loss and unaltered tympanometry. Abducens nerve palsies are described in between 9% and 36% of cases of benign intracranial hypertension as false localising signs. Other cranial nerve palsies occurring with this disorder are rare, but oculomotor, trochlear, trigeminal, and facial nerve lesions have recently been reported.1 It has been suggested that potential mechanisms for these occurrences are direct compression of the nerve root by cerebral tissue, traction of the nerve by cisternal displacement of the brainstem, or vascular disturbance as a consequence of the above. In this case there was not only a close relation between the worsening symptoms of raised intracranial pressure and the development of left sided hearing loss but also rapid normalisation of hearing acuity on reduction of the raised intracranial pressure, suggesting that the hearing loss may have been a pressure related phenomenon. In the current case, however, the audiometric pattern was indicative of a conductive hearing loss. A potential explanation is to infer an increase in the perilymphatic fluid pressure transmitted through the cochlear aqueduct as a result of the rise in CSF pressure. This might dampen the movement of the stapedial footplate and of the round window membrane giving a small conductive hearing loss. The alternative explanation of the cochlear damage leading to an effusion is unlikely given the normal tympanometry.

Although oto logical manifestations have been previously reported in raised intracranial pressure of varying aetiologies including benign intracranial hypertension,4 hearing loss is not normally considered in the context of false localising signs. It may be under recognised given the typically mild nature of the hearing loss3 and the wide prevalence of hearing loss in the normal population. In a series of 20 patients with benign intracranial hypertension the commonest otological manifestations were objective pulsatile tinnitus and low frequency hearing loss.3 Both these symptoms improved transiently in all patients after lumbar puncture. In the longer term, these symptoms responded well to weight reduction and treatment with diuretics. Wider awareness of this association will allow such patients to avoid unnecessary investigation and benefit from appropriate explanation and reassurance.

Letters to the Editor

Bromate intoxication with polyneuropathy

Acute bromate intoxication is a rare event in neurological clinics. Previous reports described renal failure and hearing deficit in addition to nausea, vomiting, haemolytic anaemia, depressed consciousness, and seizure. We report a woman who attempted suicide by taking a hair permanent wave preparation. This resulted in severe bromate intoxication and she developed renal failure, deafness, and toxic polyneuropathy. The 25 year old woman (body weight 57 kg) took 7.5 g sodium bromate in the suicide attempt in 1992. Nausea, vomiting, and diarrhoea developed rapidly. Stomach lavage and irrigation by activated charcoal were carried out at a local hospital. Tinnitus and dizziness occurred five hours later, followed by deafness. Blood urea nitrogen was 10 mg/dl (normal 7–20 mg/dl) and serum creatinine 1.4 mg/dl (normal 0.5–1.2 mg/dl for women). Oliguria was noted on the second day, with blood urea nitrogen (BUN) and creatinine rising to 16 mg/dl and 3 mg/dl respectively. The urine sediment disclosed 3–5 red blood cells per high power field (normal 0–2), and 45–50 white blood cells per high power field (normal <5 per high power field). Proteinuria was present (protein >3 mg/dl but without casts). Serum bromide (Br) concentration was 85 µg/ml (none present in normal subject). Oliguria was resistant to furosemide. On the third day, serum urea nitrogen reached 48 mg/dl and creatinine 9.3 mg/dl. Because of deteriorating renal function she was referred to the Veterans General Hospital, Kuala Lumpur and received three courses of haemodialysis.