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# Non-convulsive status epilepticus: a treatable cause of confusion in pituitary apoplexy

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#### Abstract

Confusion occurring in pituitary apoplexy is well described. We describe a case of pituitary apoplexy associated with confusion, occurring as a result of non-convulsive status epilepticus. Electroencephalography should be performed in pituitary apoplexy associated with confusion if this treatable and potentially serious complication is not to be missed.

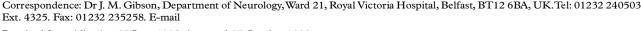
**Key words**: Confusion, non-convulsive status epilepticus, pituitary apoplexy.

## Introduction

Non-convulsive status epilepticus is a rare cause of a confusional state and is probably unfamiliar to many neurosurgeons. We describe a case of pituitary apoplexy in which this occurred. The confusion cleared with appropriate treatment, prior to surgical removal of the pituitary.

### Case report

A 60-year-old woman, with a 5-year history of chronic frontal headache, presented with increasingly severe generalized headache associated with vomiting for 6 days, and drowsiness for 2 days. On admission she was dehydrated, with a low grade fever (37.9°C) and tachycardia (12 min), but was normotensive (120/





60 mmHg). Glasgow coma scale (GCS) was 7/15 (M=5, V=1, E=1) and marked neck stiffness was present. All reflexes were pathologically brisk and the plantar responses were extensor. Otherwise examination was unremarkable. Intermittently, for up to half an hour, the GCS was noted to fall to 4/15. During these episodes continuous repetitive stereotyped 'plucking' movements of the hands, and occasional lip-smacking movements were observed.

Laboratory studies showed a mild peripheral leucocytosis and no other abnormalities. The serum sodium was 143 mmol/l on admission to hospital and remained normal throughout. Brain CT was normal apart from slight enlargement of the sella. Lumbar puncture revealed clear cerebrospinal fluid (CSF) at an opening pressure of 70 mm of CSF with  $9 \times 10^6$ /l lymphocytes,  $49 \times 10^6$ /l erythrocytes, a protein concentration of 0.63 g/l, a glucose concentration of 3.6 mmol/l (serum glucose 8.3 mmol/l) and no organisms on direct microscopy or culture. An electroencephalogram (EEG) demonstrated continuous high amplitude, repetitive, single sharp waves, consistent with status epilepticus (Fig. 1), which diminished with the administration of intravenous diazemuls.

The initial diagnosis was non-convulsive status epilepticus (NCSE), due to presumed infective meningoencephalitis and treatment was commenced with intravenous chlormethiazole and benzylpenicillin, cefotaxime, acyclovir and dexamethasone. She continued to be obtunded, however, EEG remaining

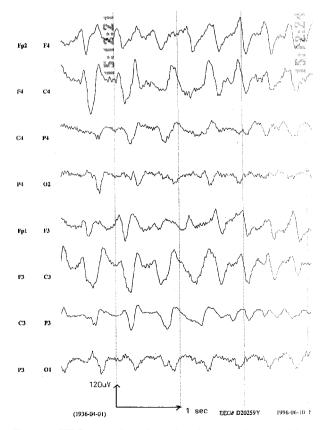


FIG. 1. EEG on admission showing continuous, highamplitude, single sharp waves, maximal anteriorly, consistent with status epilepticus.

unchanged. Intravenous clonazepam was substituted in place of chlormethiazole. Within hours she became fully orientated, but had a right-sided third nerve palsy. Visual acuity and fields to confrontation were normal. EEG at this stage did not demonstrate any epileptic activity. MRI showed a large mass arising from the pituitary fossa on T1 weighted images with well defined peripheral enhancement (Fig. 2). This extended superiorly to the optic chiasm and inferiorly into the sphenoid sinus. It was composed centrally almost entirely of low signal on T1 weighted images and high signal on T2 weighted images, indicating the presence of fluid within the lesion, in keeping with pituitary apoplexy.

The tumour was approached trans-sphenoidally and proved to be a null cell adenoma, which contained areas of necrotic, bloody material. Postoperatively the third nerve palsy resolved. The patient remains on hydrocortisone and thyroxine replacement therapy. No further seizures have occurred.

#### Discussion

Pituitary apoplexy occurs in approximately 2% of patients with pituitary tumours, 1,2 because of haemorrhagic infarction or necrosis of the tumour. The diagnosis of the condition can be difficult and is often delayed, as in our case, because in addition to its relative rarity, the presenting symptoms and signs can suggest more common conditions and the existence of an adenoma is usually unsuspected at the time of the ictus.<sup>3</sup> The features of the syndrome include headache of acute onset, vomiting, meningism, visual impairment, ophthalmoplegia and deterioration in the level of consciousness<sup>2</sup> Alteration in the level of consciousness, attributed to such diverse causes as brain-stem or hypothalamic dysfunction, vasospasm and metabolic derangement is not uncommon.<sup>3,4</sup> However, its occurrence because of



FIG. 2. Gadolinium-enhanced MRI showing pituitary tumour enhancing peripherally with contrast, with inferior hypodensity suggestive of tumour necrosis.



seizure activity is poorly documented, despite the recognized occurrence of epilepsy secondary to local mass effects from pituitary adenomata.<sup>5</sup>

The case presented illustrates the usefulness of EEG in defining NCSE as the cause of stupor in our patient. Although EEG changes including diffuse slowing have previously been reported in pituitary crises,6,7 EEG is rarely performed in this setting. We therefore predict that if the initial working diagnosis had been pituitary apoplexy, rather than meningo-encephalitis we would not have performed an EEG and NCSE would not have been identified, which might have been to the detriment of the patient.

NCSE accounts for up to 20% of all cases of status epilepticus<sup>8</sup> (the remaining 80% having convulsive status epilepticus) and is frequently missed.9 This is because, despite the usual presence of continuous epileptic activity on EEG, NCSE is essentially behavioural in presentation without prominent or any motor manifestations.10 Although considered by some to be a relatively benign condition, 11 certain types have been shown to be associated with serious morbidity and mortality.12 Treatment is often effective.10 We would suggest that EEG should be performed in pituitary apoplexy associated with confusion to avoid missing this treatable and potentially serious complication.

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# Primary petrous bone Ewing's sarcoma

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### Abstract

A case of primary Ewing's sarcoma involving a large portion of the petrous temporal bone in an eighteen month old child is reported. The lesion was treated by radical surgery and adjuvant chemotherapy and radiotherapy. An extremely rare age of presentation, an unusual location and an excellent response to treatment are the highlights of this case.

**Key words**: Chemotherapy, Ewing's sarcoma, petrous bone, skull base surgery.

## Introduction

Primary Ewing's sarcoma arising from the skull base in general and petrous bone in particular are extremely uncommon.<sup>1-4</sup> On our literature survey we located three cases of primary Ewing's sarcoma involving the petrous bone arising either primarily within it or by virtue of secondary extension.<sup>1,2</sup> An additional case of Ewing's sarcoma involving the petrous temporal bone is reported and the literature of the subject is reviewed.

