SHORT REPORTS

Serum prolactin in epilepsy and hysteria

Differentiating between hysteria presenting in an epileptiform manner and epilepsy can present diagnostic difficulties. Differentiation is important, however, since management of the two disorders differs. Electrochemical stimulation of the medial basal hypothalamus in animal models increases prolactin release.[1] Therefore if the abnormal electrical activity in epilepsy passes through the midbrain it should raise the serum prolactin concentration. This paper reports an investigation of this hypothesis.

Patients, methods, and results

Initial studies of neuroendocrine abnormalities after epileptic seizures showed that the optimal time for observing serum prolactin changes after a fit was 15-25 minutes. Blood was therefore taken from patients with epilepsy and from patients with a diagnosis of hysteria 20 minutes after a clinical fit. All the hysteria patients had presented initially with a diagnosis of epilepsy, and the pattern of their seizures resembled major tonic-clonic seizures. Hysteria was diagnosed on positive criteria as well as negative neurological signs. Serum prolactin concentrations were also measured in patients after non-dominant unilateral electric convulsion therapy (ECT) with standard anaesthetic procedures. The patients were divided into the following four groups: (1) those with generalised tonic-clonic seizures lasting more than 30 seconds with generalised interictal electroencephalographic (EEG) abnormalities; (2) those with hysteria (two had coexisting epilepsy and generalised EEG abnormalities); (3) those given unilateral ECT; and (4) those with "minor convulsions" with brief periods of altered consciousness and focal or generalised EEG abnormalities.

The results are shown in the table. After a generalised tonic-clonic seizure the serum prolactin concentration rose sharply compared with baseline levels. In all but one patient the concentration rose to 1000 μU/ml or more postictally. Such rises were not seen after hysterical seizures. In three hysteria patients the concentration was lower than the baseline level. Serum prolactin was raised in all patients after unilateral ECT. No clear pattern was seen in patients in group 4 but two, one with complex partial epilepsy, had postictal concentrations exceeding 1500 μU/ml.

Baseline serum prolactin concentrations (μU/ml) and concentrations 20 min after a fit in patients with generalised epilepsy and patients with hysteria, and in patients before and 20 min after unilateral ECT

Generalised epilepsy (n = 9)		Hysteria (n = 7)		Unilateral ECT (n = 11)	
Baseline	After fit	Baseline	After fit	Before	After
400	3400	140	480	395	2890
250	3000	460	360	280	2820
480	2100	425	345	320	2520
400	1800	350	300	290	2040
640	1400		261	380	1920
650	1200	160	240	230	1830
415	1120	220	222	360	1440
	1000			140	1000
240	680			355	790
				480	740
				169	315

Comment

These results suggest that the serum prolactin concentration rises after a generalised tonic-clonic seizure and is maximal 15-25 minutes after an attack. In all patients but one it was above 1000 μU/ml. Patients with a clinical diagnosis of hysteria, presenting as major epilepsy, had no such rises in serum prolactin after an attack. The serum prolactin concentration after a seizure may therefore be useful in differentiating hysteria from epilepsy. This study indicates that when the prolactin concentration is above 1000 μU/ml, in the absence of other causes such as a high baseline level or medication, the attack is epileptic rather than hysterical. Since similar rises occur after unilateral ECT with anaesthesia and a muscle relaxant they are unlikely to be due to either muscular activity or anoxia. Probably a rise in serum prolactin concentration will occur only when there is abnormal neurophysiological activity in the midbrain hypothalamic region. After minor seizures the serum prolactin levels were not always high, presumably because the spread of seizure activity differs depending on the characteristics of the discharge. Since serum prolactin was considerably raised after a fit in one patient with complex partial epilepsy prolactin concentration may be an indicator for detecting "limbic" partial seizures. Further studies of neuroendocrine changes after epileptic seizures and ECT are in progress and may lead to a better understanding of epileptic mechanisms, particularly of some of the behavioural and somatic consequences of recurrent abnormal electrical activity in the brain.

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[1] Clemens, J A, *et al*, *Experimental Brain Research*, 1971, **12**, 250.

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Vipoma: localisation by percutaneous transhepatic portal venous sampling

The watery diarrhoea syndrome of Verner and Morrison[1] is usually associated with a non-β islet cell tumour of the pancreas producing vasoactive intestinal peptide (VIP).[2] But tumours in sites other than the pancreas—particularly the adrenal medulla, sympathetic ganglia, and lung—may secrete VIP.[3] Cases of the watery diarrhoea syndrome have also been reported in which a tumour was present but did not secrete VIP or, indeed, in which there was neither tumour nor increased secretion of VIP.[4] When a pancreatic vipoma (tumour secreting vasoactive intestinal peptide) is present the syndrome is theoretically cured by removing the tumour, provided it has not metastasised. Diagnosis is often delayed, however, the average duration of symptoms before diagnosis being three years, and preoperative localisation of the tumour often difficult or impossible. We report here a case of watery diarrhoea syndrome produced by a vipoma which was localised by transhepatic venous sampling even though conventional diagnostic methods had failed to detect it. The syndrome was completely suppressible with corticosteroid—a feature of this disease often not appreciated. The interval between first symptoms and surgical cure was five months, the shortest recorded to our knowledge.

Case report

A 50-year-old man with no relevant history developed sudden, severe watery diarrhoea. On admission he was dehydrated, hypokalaemic, hypercalcaemic, and uraemic. He was treated empirically with intravenous fluid replacement and corticosteroids and rapidly recovered both clinically and biochemically (figure). Over the next four months he had several more attacks of watery diarrhoea, some very severe, which responded on each occasion to high doses of prednisolone. The figure shows their severity, episodicity, and response to corticosteroids. While in remission taking prednisolone 20 mg/day his serum biochemical values were normal. During an attack of severe diarrhoea investigations showed K^+ 1·9 mmol(mEq) l, urea 22·3 mmol/l (134 mg/100 ml), Ca^{++} 3·71 mmol(7·4 mEq)/l, plasma VIP 450 pmol/l (normal < 20 pmol/l). Other investigations were normal including serum gastrin, calcitonin, and parathormone concentrations; thyroid function tests; rectal and jejunal biopsy specimens; and barium meal, follow-through, and enema examinations.

A vipoma was considered the probable diagnosis, and localisation was attempted by conventional methods including coeliac, mesenteric, and hepatic arteriography; thyroid scan; intravenous pyelogram; and abdominal