Clinical Reports

Myxoedema presenting with seizures

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Summary: The case of a 32 year old female who presented with her first epileptic fit and took over 36 hours to recover is described. Investigations led to a diagnosis of hypothyroidism as the cause. Following treatment with thyroxine she has remained well with no further fits for over 2 years. Hypothyroidism should be considered as a possible underlying cause of epilepsy especially when there is a prolonged recovery time after a fit.

Introduction

Patients are often brought to Accident and Emergency departments after having had a seizure in a public place. We describe such a patient who presented in this fashion but had an unusually long post-ictal period. The final diagnosis was one of hypothyroidism.

Case report

A 32 year old woman had been seen cycling across some wasteground when she fell from her bicycle and appeared to have a grand mal type seizure. There was some doubt as to whether the fit preceded the fall or vice versa.

On arrival she was very restless, agitated and confused and needed to be restrained. Following 15 mg diazepam intravenously her agitation settled. Her pupils were dilated but reacting, eye-opening was spontaneous and she was responding to pain but with no verbal response. There were no localizing neurological signs and her plantar reflexes were equivocal. There were no external signs of a head injury, she had no neck stiffness, Kernig's sign was negative and fundoscopy was normal. Apart from obesity and psoriasis, general examination was unremarkable, as was her previous medical history and family history, later obtained from a relative. Results of urea and electrolytes, full blood count, blood sugar, drug screen and skull X-ray were all normal.

The patient was admitted for observation and a

lumbar puncture performed which showed a slight increase in protein (0.4 g). She remained unresponsive, though later that evening she had a further fit and vomited. Overnight she developed a mild pyrexia and blood cultures, thyroid function tests, and viral titres were done.

The following morning, still unconscious, she had a cranial computerized tomography scan which was normal. Electroencephalogram showed diffuse slow activity indicative of an encephalopathy of unknown origin.

Thirty-six hours after admission, having remained in a comatose state throughout, she suddenly sat up and requested paracetamol for a headache. She continued to improve and she was discharged one week later.

Results of her blood cultures and viral titres were entirely normal. However, serum thyroxine of 27 nmol/l (normal 60–145) and thyroid stimulating hormone of 93.7 mU/l (normal 0.4–4) indicated significant hypothyroidism. Following treatment with L-thyroxine on an outpatient basis, she has begun to lose weight and has become biochemically euthyroid. Serial electroencephalograms have demonstrated rapid improvement in excess of that expected in a case of encephalitis. She has had no further fits.

Discussion

Some of the associations between hypothyroidism and neurological function are well recognized. Carpal tunnel syndrome, the psychotic manifestations of myxoedema psychosis and the often terminal myxoedema coma are mentioned in most

standard textbooks. However, the association with seizures has only been noted sporadically.

As long ago as 1922 it was shown that there was an increased susceptibility of rabbits¹ and cats² to induced convulsions following thyroidectomy. Later the administration of synthetic thyroxine was shown to reduce the incidence of photically induced epileptic responses in baboons *Papio papio*.³

There have been few cases of myxoedema in human patients presenting *prima facie* with epileptic-type seizures but in all instances the patients had several episodes of convulsions before the connection was made and appropriate treatment commenced.

Evans⁴ described one such case and postulated various explanations. He suggested that impaired liver glycogenolysis and reduced adrenocortical function caused by absence of thyroxine produced a mild hypoglycaemia. Alternatively, it could be that the increased interstitial and intracellular fluids and reduction in plasma volume seen in patients with myxoedema, with alterations in the blood brain barrier, results in increased cerebrospinal fluid and cerebral oedema which may cause the fits. Atheroma producing a reduced cerebral blood flow was also postulated but unsubstantiated as a cause.

Thrush and Bodie⁵ reported one patient with Hashimoto's thyroiditis and another with myxoedema who had recurrent seizures and encephalo-

pathy attributed to their thyroid disease. Like the patient described here, these had normal routine haematological, biochemical and virological investigations. Also, they had raised cerebrospinal fluid proteins and diffuse encephalopathic changes on electroencephalogram. They postulated that in the patient with Hashimoto's disease, an associated cerebral angiitis, also of auto-immune origin, was the cause. In the patient with myxoedema, like ours, they suggested that the encephalopathy was a direct result of the hypothyroidism. Following treatment with thyroxine their patient had no further seizures.

Convulsions of an epileptic type are an uncommon complication of myxoedema, whether as a direct effect on the neurones of the central nervous system, an episode of cerebral oedema, or secondary to peripheral metabolic alterations. It is recommended that thyroid function tests should be performed in any patient presenting with a fit when there is a prolonged period of recovery or if the fits are of a recurrent nature and thought to be idiopathic.

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