NASOPHARYNGEAL CARCINOMA WITH AUTONOMIC EPILEPSY

Y T Wang

T L Tjia

S C Poh

Department of Medicine III
Tan Tock Seng Hospital
Moulmein Road
Singapore 1130

Y T Wang, MRCP (UK)
Senior Registrar

S C Poh, FRCPE
Head and Senior Physician

Department of Neurology
Tan Tock Seng Hospital

T L Tjia, M Med (Int Med)
Head

INTRODUCTION

We report a unique patient with nasopharyngeal carcinoma and autonomic epilepsy.

CASE REPORT

A 56 year old Chinese man was admitted to this hospital on 16.7.83 for attacks of giddiness. He had had 10 such attacks in the previous two months, each lasting a few minutes. He described them as spells of giddiness and profuse sweating which occasionally led to loss of consciousness. No involuntary movements were observed by the relatives. He had developed cervical lumps 5 years before and these had persisted despite self-medication with traditional Chinese applications. He smoked cigarettes but denied alcohol abuse. On admission, during an attack, he was drowsy, sweaty and cold. Systolic blood pressure was 80 mmHg. Bilateral hard cervical lymph nodes were present, some of which had ulcerated after topical applications. The pulse was 60/min and regular. He appeared to respond to a bolus of intravenous glucose, and blood pressure rose to 130/80 mmHg. However, the initial blood sugar level was 103mg% (5.7 mmol/l). He suffered a further 15 such episodes in the next 3½ weeks. In between attacks, he was well and his blood pressure and electrocardiogram were normal. Neurologic examination was negative except for a mild left ptosis which was noted on 23.7.83 and which developed into a Horner's syndrome a week later.

The attacks were typically heralded by giddiness, retching and vomiting. The pulse then became weak and was impalpable on two occasions. The pulse rate was variable but slowed down to 50/min in one attack, and increased to 148/min in another. The blood pressure almost always fell to a systolic of 50-80 mmHg., and was unrecordable once. He then broke out in profuse sweating all over the body, his bed clothes typically being soaked with perspiration. Sometimes, he lost consciousness and was transiently apnoeic. He had urinary incontinence during 3 attacks and bowel incontinence in one. Each attack lasted about 3 minutes. During recovery, he appeared tired and slept. Blood sugar and serum electrolytes were repeatedly normal. Other investigations including blood urea, serum calcium, serum phosphate, haemoglobin concentration and white cell counts were also normal. Twenty-four hour urinary vanillyl mandelic acid excretion taken on 3 occasions following the attacks gave normal values (26.5, 22, 29 umol per day). Urinary 5-hydroxyindoleacetic acid excretion was also normal (23 umol per day). A chest film showed old tuberculous scars in the right upper lobe.

In view of the possibility that the attacks were of cardiac origin, a transvenous pacemaker was inserted into the right ventricle on 5.8.83. However, the patient suffered further attacks, with hypotension occurring despite a pacing rate of 70/min. The pacemaker was later removed. The electroencephalogram during an attack showed a sudden onset of generalised polymorphic delta wave activity which lasted for 40 msec. However, no epileptiform discharge was seen.

Associated motor convulsions were absent until 12.8.83, when two such seizures were accompanied by biting of lips, uprolling of eyes, and clenched fists. On the same day, he developed 3 generalised seizures. CT scan of the brain showed no abnormalities. He was then started on carbamezepine 200 mg.t.d.s. No attacks of autonomic dysfunction or generalised seizures occurred after commencement of carbamezepine. A cervical lymph node biopsy showed metastatic carcinoma and biopsy of the posterior nasal space revealed a nasopharyngeal carcinoma. A repeat CT brain scan on 30.8.83 showed some fullness in the left posterior nasopharynx, but no intracranial abnormalities. A chest radiograph two months after hospitalisation showed multiple pulmonary metastases. He remained seizure-free and comfortable, and finally died on 21.12.83, 5 months after presentation. Autopsy was not performed.

DISCUSSION

The diagnosis of autonomic epilepsy was made on the basis of the stereotypic and episodic attacks of autonomic dysfunction (vomiting, hypotension, diaphoresis, sphincter disturbance, cardiac arrhythmias) in the absence of a motor component. The appearance of motor convulsions with the later episodes of autonomic dysfunction strongly favours their epileptic nature. Finally the dramatic response to carbamezepine further supports the diagnosis of autonomic epilepsy.

Autonomic epilepsy was first described in 1929 by Penfield in a patient with a choroid plexus tumour (1). A few years later McLean reported another case, this time due to a hypothalamic astroblastoma (2). Subsequently, more cases of tumour associated autonomic dysfunction were reported: cerebellar tumours (3), astrocytoma (4,5) and astroblastoma (6). It is noteworthy that all these were primary brain tumours. Excessive autonomic discharge has also been reported to occur in temporal lobe epilepsy (7,8), stroke (9), thalamic degeneration (10), agenesis of the corpus callosum (11), or without any associated disorder (12,13).

Nasopharyngeal carcinoma is noted for its neurological complications, the majority of which involve the cranial nerves (14). To the best of our knowledge, autonomic epilepsy has never been described in association with tumours outside the cranium. Our patient had a nasopharyngeal carcinoma, and presented to us only when it was complicated by autonomic epilepsy. We could not ascribe the seizures to any electrolyte or metabolic disturbances, nor did we detect any intracranial metastases on 2 separate CT brain scans. However, as he had widespread pulmonary metastases and autopsy was not performed, micrometastases to the hypothalamus or brain stem cannot be excluded. The resemblance of these attacks to hypoglycaemia, phaeochromocytoma and even Stokes-Adams attacks may delay diagnosis and treatment. Although the patient finally succumbed from the malignancy, the last months were free from these disabling seizures.

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