Carbamazepine-responsive paroxysmal nausea and vomiting in a patient with meningeal carcinomatosis

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Case Report

Carbamazepine-responsive paroxysmal nausea and vomiting in a patient with meningeal carcinomatosis

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In neurology, paroxysmal syndromes are well-known, eg, as manifestations of multiple sclerosis. We report a patient with meningeal carcinomatosis, who presented with therapy-refractory nausea and vomiting. The clinical suspicion of a paroxysmal syndrome prompted a trial of carbamazepine, which resulted in complete cessation of the symptoms. In cancer patients with central nervous system (CNS) involvement and therapy-refractory symptoms with sudden onset, carbamazepine treatment should be considered. *Palliative Medicine* 2006; **20:** 549–550

Introduction

Therapy-refractory nausea and vomiting may result in unacceptable suffering in patients with advanced cancer. We report an unusual case of paroxysmal nausea and vomiting responding to anti-convulsant treatment in a patient with breast cancer. Paroxysmal syndromes are well-known in neurology, eg, as manifestations of multiple sclerosis. These syndromes present as sudden, short bouts of a specific symptom (eg, pain, vertigo), usually triggered by movement or external stimuli. The best known example is trigeminal neuralgia.[1] The literature reports a few cases of paroxysmal nausea and vomiting.[2–4] To our knowledge, this is the first reported case of a paroxysmal syndrome in a patient with meningeal carcinomatosis.

Case history

A 39-year-old patient with metastatic breast cancer presented with nausea and vomiting, headache, photophobia and painful neck stiffness. A NMR of the brain showed diffuse dural enhancements and plaques (Figure 1). A spinal tap revealed malignant cells and increased protein levels. The diagnosis of meningeal carcinomatosis was made and intrathecal chemotherapy was started.

Pain medication controlled the headache, but the patient still suffered from sudden, visually and movement-induced bouts of nausea and vomiting, which caused her to lie in bed with closed eyes for most of the time. These episodes were induced by almost every movement of the patient. Treatment with metoclopramide, 5-HT_3-receptor-antagonists, dexamethasone and levopromazine failed completely. The diagnosis of a postural vertigo was excluded. The clinical suspicion of a paroxysmal syndrome prompted a trial of carbamazepine. Nausea and vomiting stopped immediately after a first dosage of 100 mg. Interruption of medication resulted in symptom recurrence on the next day. An electroencephalogram (EEG) showed intermittent paroxysmal excitations. We re-started the medication with carbamazepine 400 mg/day. This allowed the patient to leave the bed and eat without nausea and vomiting. Despite intrathecal chemotherapy, the patient's condition worsened and death ensued three weeks later, without recurrence of the paroxysmal syndrome.

Discussion

We report an unusual, well-treatable clinical manifestation of meningeal carcinomatosis – paroxysmal nausea and vomiting. The level of suffering was high, because the patient had to lie immobile and close her eyes to suppress the symptoms.

Nausea and vomiting are associated with a chemoreceptor trigger zone in the area postrema and a vomiting centre, both located in the medulla oblongata. Common antiemetics, including serotonin antagonists, are usually

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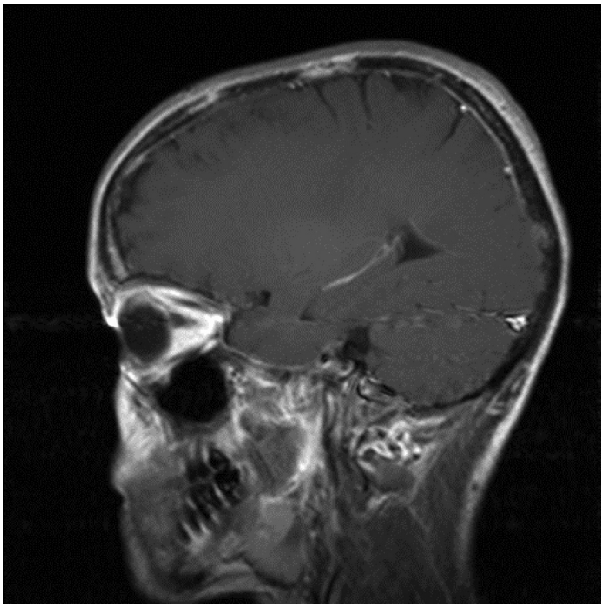

Figure 1

able to control nausea and vomiting in incurable cancer patients, but did not show any effect in our patient. The dramatic effect of the first application of carbamazepine, the recurrence of the symptoms after treatment interruption and the reproducible effect after re-administration of the drug confirmed the clinical diagnosis of a paroxysmal syndrome.

In central demyelinating disorders, paroxysmal syndromes are thought to be due to ephaptic (ie, non-synaptic) transmission of discharges between neighbouring axons within areas of demyelination. Brainstem-related paroxysms may present as paroxysmal dysarthria, ataxia, diplopia, tonic seizures, akinesia, sensory disturbances, or pains.[4] Visually-induced paroxysmal vomiting has been reported in multiple sclerosis.[2] Paroxysmal vomiting has also been described as a manifestation of temporal lobe epilepsy.[5] The exact pathophysiological mechanism leading to the paroxysmal syndrome in our patient is unclear, but was likely related to the meningeal carcinomatosis, which possibly caused local demyelination in the brainstem.

Carbamazepine is the drug of choice to suppress paroxysms and the response is usually dramatic.[4] Given the high frequency of central nervous system (CNS) involvement in tumour patients, currently, paroxysmal symptoms may be under-diagnosed. In this group of patients, therapy-refractory symptoms with sudden onset (pain, vomiting, vertigo and other unclear neurological symptoms) should prompt a treatment trial with carbamazepine.

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