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Unusual presentation of squamous cell carcinoma of the middle ear and mastoid

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Abstract
An unusual case of squamous cell carcinoma of the middle ear and mastoid in which syncope was a major presenting feature is reported. No such case has been reported in the literature. A possible explanation is offered.

Introduction

Squamous cell carcinoma of the middle ear and mastoid, even though the most common form of carcinoma in the region, is very rare. The incidence is said to range from 1:4000 to 1:50 000 patients (Conley, J. 1965).

It is often diagnosed late, an average of six months from the initial symptom (Lewis, 1973). This is because the main symptoms which are otorrhoea and vertigo are not specific to carcinoma but rather to chronic suppurative otitis media. The changes which may arouse suspicion include blood-stain otorrhoea, severe pain, and cranial nerve paralysis.

When it presents late, the disease is often extensive and spreads by transdural invasion along the VIIth and VIIIth nerves in the internal auditory meatus but affects the IXth, Xth, XIth and XIIth nerves extracranially along the base of the skull (Stell, 1987).

Treatment of the disease is very difficult, especially when it extends beyond the confines of the middle ear and mastoid.

Case history

A 76-year-old man was admitted via the Casualty Department with a three-month history of recurrent loss of consciousness. These episodes, which lasted for a few seconds, were without warning and were unrelated to posture, exercise or head movements. The patient usually fell to the ground and the limbs became flaccid. Recovery occurred usually very quickly.

He was not on any medication but had been treated by his

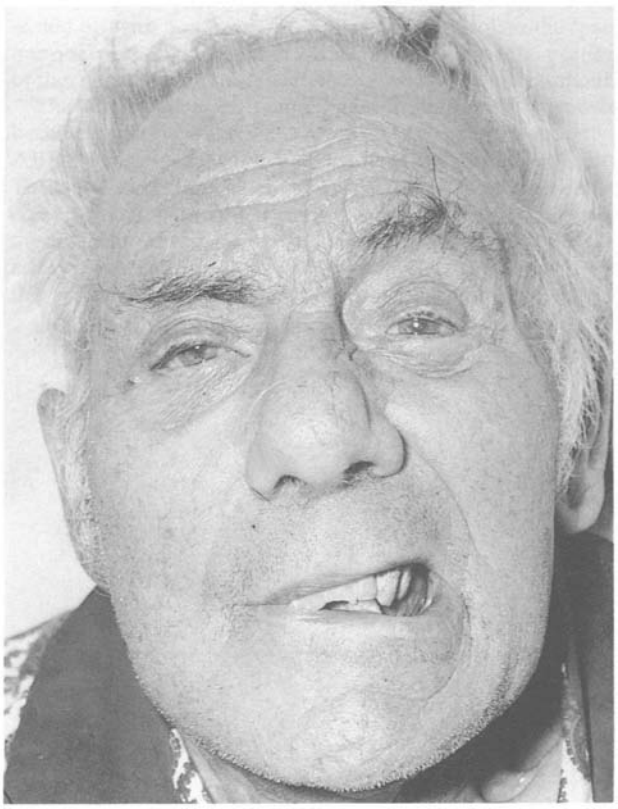

Fig. 1

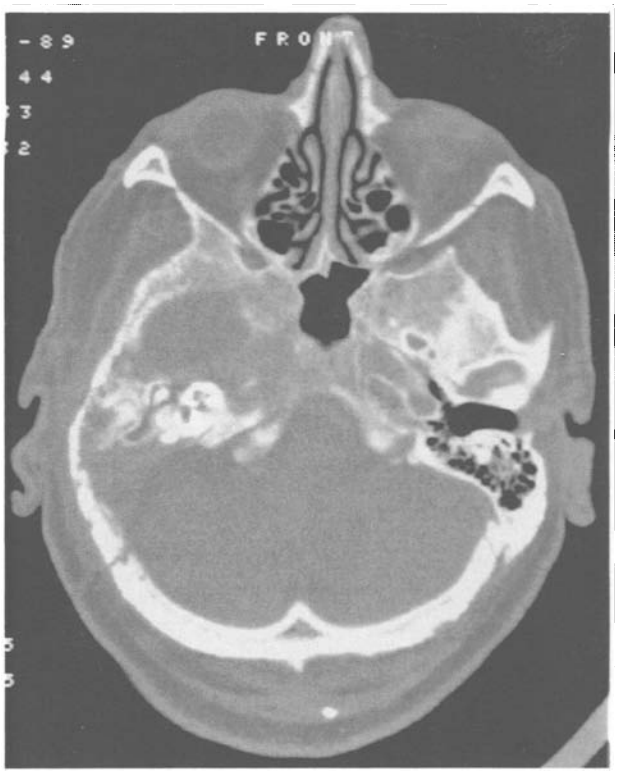

Fig. 2

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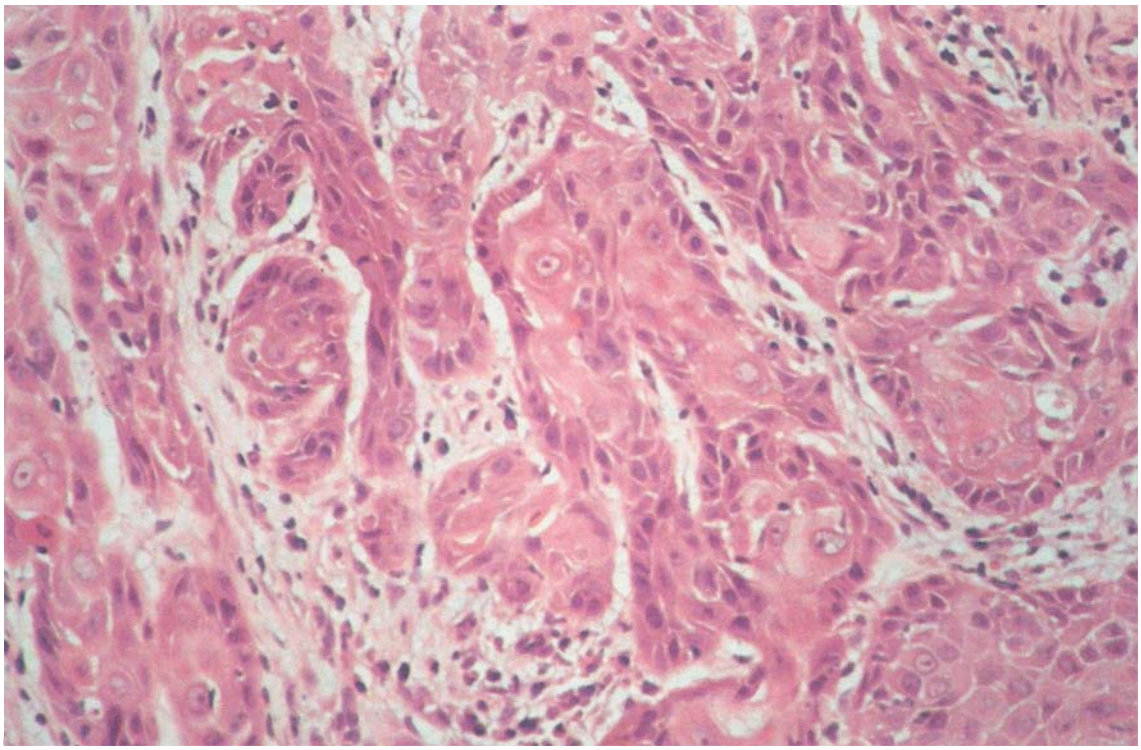

FIG. 3
An H&E stained section of the biopsy showing well differentiated squamous cell carcinoma (×250)

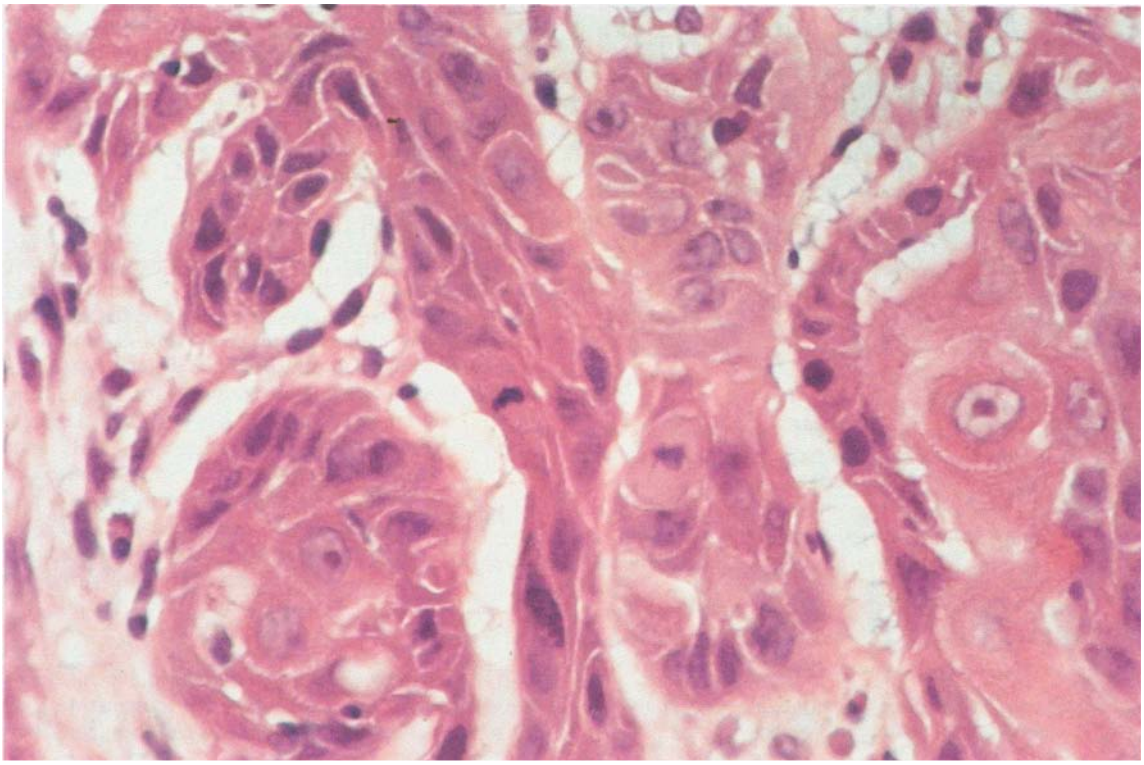

FIG. 4
A high power field from an H&E stained section showing mitotic bodies (×500)

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general practitioner for episodes of discharge from the right ear and Bell's palsy (Fig. 1). He also had hearing loss, markedly worse in the right ear, hoarseness and dysphagia. He had no earache and no vertigo and was very alert.

On examination, he had scanty blood-stained discharge from the right ear and paralysis of the VIIth, IXth, Xth, XIth and XIIth cranial nerves. There was no nystagmus.

The right ear was examined under the microscope and this revealed an absent tympanic membrane and posterior meatal wall, extensive granular tissue in the external meatus, middle ear and mastoid. There were no ossicles and the promontory was 'honeycomb'.

A biopsy was taken for histology and was reported as 'well differentiated keratinizing squamous cell carinoma' (Figs. 3,4).

Routine investigations such as urea and electrolytes, full blood count, blood sugar, VDRL, ECGs and chest X-rays were normal. Caloric and positional tests were considered unnecessary because of the state of the right ear.

He did have a few episodes while on admission but blood pressure measurements and ECG monitorings soon after the episodes were normal. A neurological opinion was sought and no cause was identified.

A CT scan (Fig. 2) revealed extensive bony erosion of the mastoid, petrous bone, middle and posterior cranial fossae, base of the skull and posterior wall of the maxillary antrum, all on the right side.

The disease was too extensive to treat and the patient had only symptomatic care.

Discussion

Syncope can be caused by a fall in the cardiac output, a fall in the peripheral vascular resistance of the systemic circulation, or due to local abnormality of the blood vessels supplying the brain (Shillinford, 1970). In the elderly, cardiovascular disorders such as dysrhythmias, heart blocks, transient ischaemic attacks are the most common causes. They can often be diagnosed by repeated or continuous ECG monitoring. Transient ischaemic attacks tend to have neurological disturbances such as hemiparesis, hemi-sensory disturbance or dysphasia although these are not always present (Spalding and Wollner, 1985). All these were excluded in the patient being discussed.

Other rare causes such as micturition, coughing and defaecation are usually easy to identify.

Hypoglycaemia is a known cause but is usually associated with sweating and low blood sugar while epilepsy is diagnosed by increased muscle tone. The patient had none of these.

The vasovagal group is usually more common in the younger age group (Isaacs, 1985).

Bradycardia from excessive vagal tone is associated with peripheral vasodilatation in muscles. Factors responsible include anxiety, pain, emotional stress and standing for long periods. These factors can be difficult to exclude.

Glossopharyngeal neuralgia is another documented cause of syncope. This is due to 'irritation' of the glossopharyngeal nerve. Several head and neck tumours have been known to cause syncope via this mechanism. These tumours include cerebellopontine angle tumour, pharyngeal abscess (Sobol *et al.*, 1982), carcinoma of larynx and nasopharynx with local spread (Giorgi and Broggi, 1984). Reddy *et al.* (1987) reported a case of glossopharyngeal neuralgia in which there was no associated pain. As previously stated, there has been no specific mention of carcinoma of the middle ear and mastoid as a cause.

The mechanism of syncope by these groups is that proposed by Gardner (1973) in which he suggested that there are artificial synapses formed between the glossopharyngeal and vagal fibres in the region of their ganglia. When there is 'irritating' sensory phenomenon, there is said to be a 'cross-talk' at these levels resulting in a decrease in the cardiac drive and/or peripheral vascular resistance resulting in the syncope.

We would like to suggest that this is a tenable explanation as the 'irritation' is the spread of tumour around the glossopharyngeal nerve at the jugular foramen shown by the CT scan (Figs. 2, 3).

It has also been suggested that transient ischaemic cerebral attacks can occur without neurological disturbances and therefore cannot be excluded.

Acknowledgement

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