CASE REPORT

Pancreatic adenocarcinoma presenting with sudden onset bilateral deafness secondary to metastatic leptomeningeal infiltration

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Abstract

Deafness is a very common problem in older persons. We present a case of metastatic adenocarcinoma of the pancreas with deafness being the patient’s only reason for seeking medical attention. The patient had bilateral vestibulocochlear nerve palsies with associated lower motor neurone facial nerve palsies and a bulbar palsy. Magnetic resonance imaging of his brain was unremarkable. It was only on post-mortem histology that tumour infiltrating the leptomeninges was demonstrated. Leptomeningeal metastases are rarely associated with adenocarcinoma of the pancreas. A review of the literature reveals only two other case reports of metastatic pancreatic carcinoma presenting with deafness but both had demonstrable temporal bone lesions on MRI as opposed to the meninges.

Keywords: meningeal carcinomatosis, pancreatic carcinoma, bilateral deafness, older people

A 72-year-old male presented to us with a history of sudden onset bilateral deafness. In addition the patient preferred not to respond verbally and this meant that all communication was written. A collateral history was obtained from the patients’ partner who noted the he had needed the sound turned up very high on the television, that his voice had become more nasal and that he had become unsteady on his feet. He was a previously fit and healthy male with no medical problems and was not on any regular medications. He had no relevant family history. He did not smoke or drink alcohol.

Examination revealed a well-nourished Caucasian male with no clinical evidence of jaundice, anaemia or clubbing. Neurological examination revealed bilateral sensorineural deafness, a right-sided lower motor neurone facial nerve palsy and a bulbar palsy. His gait was ataxic but there were no other cerebellar or peripheral neurological signs. Respiratory, cardiovascular and abdominal examinations were unremarkable.

His initial full blood count, urea and electrolytes, liver function, CRP, calcium and blood glucose were all within normal limits and surprisingly remained so during his admission. A chest X-ray was performed on admission that demonstrated a large left hilum which was further investigated with a computed tomography scan of his thorax. This showed multiple enlarged mediastinal and bilateral hilar lymph nodes only.

To investigate his neurological symptoms, a magnetic resonance scan of his brain was performed which demonstrated only a mild degree of small vessel ischaemia. A lumbar puncture showed a cerebrospinal fluid (CSF) red cell count of 356 cells/mm³, a white cell count of 18 cells/mm³, decreased glucose of less than 0.3 mmol/l (serum 4.2 mmol/l) and increased protein of 2.24 g/l (0.1–0.45). CSF gram stain and bacterial culture were negative, CSF cytology demonstrated reactive macrophages but no malignant cells.

After 1 week, he developed a left-sided lower motor neurone cranial nerve VII palsy (making it bilateral) and his mobility progressively declined. After 2 weeks his level of consciousness began to fluctuate and it was felt that despite negative cytology and imaging, the clinical signs were highly suspicious of meningeal carcinomatosis. A trial of intravenous dexamethasone was commenced but no significant
improvement was seen. The patient passed away peacefully 3 weeks after his admission to hospital.

Consent was obtained from the next of kin for a hospital post-mortem with the retention of the brain. Gross pathological findings included mild jaundice, diffuse lymphadenopathy above and below the diaphragm including hilar, mediastinal and periportal lymph nodes greater than 3 cm in dimension. A 5 cm, ill-defined partially necrotic mass was identified in the head of the pancreas. Macroscopically no meningeal thickening or parenchymal masses were identified. Histology demonstrated adenocarcinoma of the pancreas that had metastasised to the liver, multiple lymph nodes and the leptomeninges (see Figure 1). The dense infiltration of the leptomeninges, especially in the posterior fossa explains the broad spectrum of neurological signs and symptoms, including deafness, that he presented with.

Discussion

Metastases of pancreatic adenocarcinoma to the brain and meninges is a rare event with only a scattering of cases reported in the literature [1–5], none of which presented with deafness. Two cases of metastatic pancreatic adenocarcinoma presenting with deafness have previously been reported [6, 7] but the cause in these cases was infiltration of the temporal bone by metastatic tumour.

This presenting complaint, deafness, is a common symptom in older persons and in a high proportion of cases the cause is benign [8] (e.g. presbycusis). This case demonstrates the need for heightened clinical suspicion of a more malignant pathology when a patient presents with a short history of deafness associated with rapid progression of other cranial nerve palsies.

Key points

- Pancreatic carcinoma with brain/meningeal metastases is a rare occurrence.
- Pancreatic carcinoma can metastasise to the brain/meninges and cause deafness and/or other cranial neuropathies.
- Deafness is a common complaint in elderly patients and an open mind should be kept when considering the diagnosis.

References


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