Isolated Meningeal Recurrence of Transitional Cell Carcinoma of the Bladder

Catherine Butchart a  Asa Dahle-Smith b  Donald Bissett b  James M. MacKenzie c  David J.P. Williams d

a Department of Medicine for the Elderly, Woodend Hospital, and Departments of  
b Oncology and c Pathology, Aberdeen Royal Infirmary, Aberdeen, UK; d Department of Geriatric Medicine, Royal College of Surgeons in Ireland, Beaumont Hospital, Dublin, Ireland

Key Words
Meningeal carcinomatosis · Recurrence · Transitional cell carcinoma · Bladder

Abstract
Meningeal carcinomatosis occurs in 1–18% of patients with solid tumours, most commonly carcinomas of the breast and lung or melanomas. There are relatively few reports of meningeal carcinomatosis in transitional cell carcinoma of the bladder. Isolated meningeal recurrence is particularly uncommon, and we present an unusual case of this in a 58-year-old man. The case was further complicated by the somewhat atypical presentation with a confirmed ischaemic stroke. The patient died one month after presentation.

Case Report
A 58-year-old man was admitted with a 2-month history of headache and a 2-day history of ataxia. There was no photophobia or neck stiffness, but he had vomited occasionally. Physical examination was unremarkable other than ataxia affecting all 4 limbs.

The patient had a previous history of a high-grade transitional cell cancer (TCC) of the bladder diagnosed earlier that year after presenting with dysuria, urinary frequency and renal failure with bilateral hydronephrosis. Bladder biopsies had revealed grade 3 muscle-invasive TCC of the bladder. The tumour was staged as T2 N1 M0 on CT imaging (with small pelvic nodes). The patient had completed 4 cycles of neoadjuvant chemotherapy with gemcitabine/cisplatin with good response and subsequently had pelvic radiotherapy (52.4 Gy in 20 fractions) completed 5 months prior to this most recent admission. A check cystoscopy 2 months after treatment showed no evidence of tumour recurrence. He had also had a previous pulmonary embolus and was receiving prophylactic low-molecular-weight heparin.

The patient underwent a CT brain scan, which showed a probable infarct in the left parietal lobe. He then developed an acute onset of left-sided weakness, left homonymous hemianopia, and left-sided
neglect in keeping with a right-sided total anterior circulation stroke. A repeat CT brain scan showed the previous lesion in the left parietal lobe, but also an early infarct affecting the caudate, the internal capsule and the lentiform nucleus on the right side (fig. 1). An MRI brain scan subsequently confirmed infarction in these areas, extending into the right posterior temporal and lower parietal regions (fig. 2).

Carotid Doppler examination, Holter monitor, and transthoracic echocardiogram with agitated saline were all normal. The patient’s blood results showed a normal full blood count, urea and electrolytes, erythrocyte sedimentation rate, glucose and lipid levels. His liver function tests were abnormal with alkaline phosphatase 193 and gamma GT 117. An abdominal ultrasound scan was normal.

The patient’s severe generalised headache persisted, and a lumbar puncture was performed. The sample was bloodstained, with red cells 11,097/μl and white cells 124/μl. Microbiological and virological investigations were negative. The cerebrospinal fluid (CSF) cytology showed malignant cells which were strongly positive for CK7 and CK20 on immunocytochemistry. This was the same immunostaining pattern as that of the original TCC of the bladder (fig. 3).

A CT scan of the chest and abdomen showed some fat stranding around the bladder, but no solid tumour mass or lymphadenopathy was identified. Unfortunately, the patient’s general condition continued to deteriorate, and he was not fit for any further active treatment. He was treated symptomatically and died 1 month after his admission.

Discussion

Meningeal carcinomatosis (MC) occurs in 1–18% of patients with solid tumours, most commonly carcinomas of the breast and lung or melanomas [1–3]. Presenting neurological features depend on whether the cerebral hemispheres, cranial nerves, spinal cord or nerve roots are involved [4]. The most common presenting symptoms are headache (which occurs in up to 50% of cases), nausea and vomiting, limb weakness and radicular pain. The most frequent clinical features are cauda equina syndrome, polyradiculopathy, cranial nerve deficits and alteration in mental status [1]. Seizures occur in 20% of the cases, and stroke-like symptoms, cerebellar signs and encephalopathy have also been reported [1, 4, 5].

Contrast-enhanced MRI scanning is the most sensitive and specific imaging modality in the detection of MC but has an estimated sensitivity of 34–71%. False positives can be due to meningitis (this is particularly important if the patient is immunosuppressed), recent surgery or radiotherapy causing dilatation of the spinal arteries. Cerebral infarction may also mimic MC [4, 6].

A small number of reports of MC in TCC of the bladder exist in the literature. Ten cases have been reported in patients with more than one site of metastatic disease and 2 cases with isolated meningeal recurrence. All cases demonstrated poor outcome, with a survival of 3–5 weeks following the diagnosis of MC [7]. It may be that the increasing use of neoadjuvant systemic chemotherapy for muscle-invasive bladder cancer will be associated with more frequent relapses with central nervous system disease, as has been the case, for example, with breast cancer.

Acknowledgement

With thanks to Dr. E. Ramage, Consultant Radiologist, Aberdeen Royal Infirmary, Aberdeen, UK.
**Fig. 1.** CT brain scan.

![CT brain scan](image1)

**Fig. 2.** MRI brain scan.

![MRI brain scan](image2)
**Fig. 3.** Cytology and immunocytochemistry (ICC) from the bladder and CSF.

- Bladder histology
- CSF cytology
- Bladder ICC for CK20
- CSF ICC for CK20
- Bladder ICC for CK7
- CSF ICC for CK7
References


