CASE REPORT

Upper ureteric transitional cell carcinoma, extending to the renal pelvis, presenting as duodenal obstruction

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SUMMARY

A 61-year-old man presented with weight loss, dysphagia and vomiting. A barium swallow revealed a duodenal obstruction at D3. CT of the abdomen and pelvis showed a left upper ureteric tumour extending to the renal pelvis compressing the duodenum and causing left-sided hydronephrosis. Cystoscopy and left-sided ureteroscopy proved difficult and were unable to visualise or biopsy the mass, but a left ureteric stent was placed. Laparoscopic biopsy of the mass was completed and histology revealed transitional cell carcinoma. The patient went on to receive palliative chemotherapy, which relieved the small bowel obstruction, and the patient was able to eat solid food 8 weeks later. This case highlights a previously unreported cause of duodenal obstruction.

BACKGROUND

Transitional cell carcinoma (TCC) is the most common malignancy of the ureter and commonly presents with haematuria, but can present as loin pain or a palpable mass. Small bowel obstruction typically presents with severe abdominal pain and vomiting, which can be secondary to adhesions, hernias, volvulus or compression from pancreatic masses. To the best of our knowledge of the authors, there are no reports of duodenal or small bowel obstruction presenting secondary to ureteric or renal pelvis TCC.

CASE PRESENTATION

A 61-year-old man presented to the emergency department with a 9-day history of dysphagia to solids and epigastric abdominal pain. The dysphagia was worsening and he had four episodes of vomiting. No haematemesis was seen. The patient described one stone of weight loss over one month. He was an ex-smoker but had no other significant medical history of note.

On examination, his chest was clear and abdomen was soft, with epigastric and left upper quadrant tenderness. An oesophagogastroduodenoscopy was organised as an urgent outpatient in 3 days and he was discharged.

The patient presented again the next day with continued vomiting, dysphagia and epigastric pain. On examination, his abdomen was soft, with epigastric tenderness but no signs of peritonism. Oesophageal malignancy or achalasia was initially suspected and barium swallow requested.

INVESTIGATIONS**Blood test results**

Haemoglobin was 17 g/dL, white cell count $11.1 \times 10^9/L$, sodium 136 mmol/L, potassium 3.7 mmol/L, urea 10.5 mmol/L and creatinine 162 $\mu\text{mol/L}$. A degree of acute kidney injury was seen as creatinine tested 14 days prior at his general practitioner was 111 $\mu\text{mol/L}$.

Radiological investigations

Urgent barium swallow revealed a normal oesophagus and gastro-oesophageal junction. Inferior X-rays showed complete duodenal obstruction at D3 with dilated proximal duodenum and stomach (figure 1). An urgent CT of the abdomen and pelvis with contrast showed a soft tissue mass involving the duodenojejunal flexure and left ureter extending to the renal pelvis causing left-sided hydronephrosis (figures 2 and 3), which was confirmed by a specialist urological radiologist. There was some atrophy of the left kidney. Normal appearances were seen of bowel, right kidney, liver, pancreas, spleen and both adrenals. No intra-abdominal free gas or fluid was seen. A nasojejunal (NJ) tube was placed to relieve duodenal obstruction.

Obtaining histology

The patient underwent oesophagogastroduodenoscopy, which demonstrated generalised inflammation but no masses. Cystoscopy revealed no masses

Figure 1 Dilated duodenum up to D3 visualised on barium swallow.

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Figure 2 Axial view of CT showing duodenal compression secondary to upper ureteric mass.

but areas of inflammation were evident. Ureteroscopy proved difficult and it was not possible to obtain tissue diagnosis. A ureteric stent was placed to relieve hydronephrosis.

Urine cytology was taken twice, which showed some cells with atypia and inflammation but was inconclusive. Following multidisciplinary team discussion between the urology, histopathology and radiology teams, it was decided to perform a laparoscopic biopsy of the mass.

DIFFERENTIAL DIAGNOSIS

The primary differential was a urinary tract malignancy such as a TCC causing duodenal obstruction, but a primary mesenteric mass such as carcinoid or small bowel primary malignancy could not be excluded.

Histology from the duodenum showed scattered intraepithelial lymphocytes, suggesting non-specific inflammation. Bladder histology showed chronic inflammation but no evidence of malignancy. Urine cytology was taken twice, which showed groups of atypical urothelial cells with large hyperchromatic nuclei and prominent nucleoli, but was inconclusive. Laparoscopic biopsy showed clusters of suspicious cells on H&E staining (figure 4) and was confirmed as malignant on pancytokeratin immunostaining (figure 5). These cells showed positivity for CK7 and CK20, which would have differentials of TCC, gastric adenocarcinoma and pancreatic adenocarcinoma.¹

Figure 3 Coronal view of CT showing duodenal compression secondary to upper ureteric mass.

Figure 4 Perirenal mass biopsy (H&E staining $\times 4$). Fibroadipose connective tissue. There is mild chronic inflammation and there are small clusters of suspicious cells (red arrow).

On the basis of presentation and radiological findings, a diagnosis of ureteric TCC was made.

TREATMENT

The patient was placed nil-by-mouth and a NJ tube inserted, with intravenous fluid replacement. Feeding was started via the NJ tube. The NJ tube got blocked and was replaced. Later, the patient had to start total parenteral nutrition (TPN) via a peripherally inserted central catheter line. Following multidisciplinary team discussion, he was diagnosed with left upper ureteric and renal pelvis TCC. He was transferred to the care of the oncologists and started on six 21-day cycles of gemcitabine 1 g intravenously on days 1 and 8 and cisplatin 70 mg intravenously on day 1.

OUTCOME AND FOLLOW-UP

The patient remained on TPN nutrition for a total of 8 weeks until he could tolerate food. He had no further episodes of small bowel obstruction. He responded well to palliative chemotherapy and suffered no side effects. After the third cycle of chemotherapy he had gained two stone in weight. A follow-up CT 6 months following presentation showed stable appearances of the upper ureteric TCC with no distant metastases.

Figure 5 Perirenal mass biopsy (Pancytokeratin immunostaining $\times 40$). Clusters of malignant cells highlighted by pancytokeratin immunohistochemistry. These cells were positive with CK7 and CK20.

DISCUSSION

To the best of our knowledge of the authors, this is the first case of ureteric or renal pelvis TCC presenting as duodenal or small bowel obstruction. However, urological causes of duodenal obstruction have been reported, including hydronephrosis secondary to pelviureteric obstruction and renal calculi.^{2 3}

The case presented multiple challenges, primarily in obtaining histology, which required laparoscopy due to failed ureteroscopy. A multidisciplinary approach was required involving general and urological surgeons, oncologists, radiologists and dieticians.

TCC is the most common malignancy of the ureter and up to 80% of patients present with visible haematuria.⁴ Synchronous ureteric TCC occurs in 0.7–4% of patients with primary bladder TCC and up to 17% of patients with upper tract TCC also have bladder cancer.⁵ Options for management of ureteric

TCC depend on size and location of tumour and include conservative, ureteroscopic, laparoscopic or open resection, and/or local chemotherapy using mitomycin C or BCG.⁶ Unfortunately, in contrast to bladder TCC, up to 60% of ureteric TCCs are invasive at presentation and, in such patients, 5-year survival rates are less than 50%. In T4 disease 5-year survival rates fall to less than 10%.^{7 8}

Contributors LAS and NS contributed to the writing of the manuscript. MS reviewed the manuscript. LAS, NS and MS were all involved in the clinical care of the patient.

Competing interests None declared.

Patient consent Obtained.

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Learning points

- ▶ Upper ureteric cancers can present as small bowel obstruction, however, this is very rare.
- ▶ Locally invasive upper tract TCC can present without loin pain or haematuria.
- ▶ Acute management is focused on relieving small bowel obstruction.
- ▶ Long-term nutritional support may be required using either an nasojejunal tube or total parenteral nutrition.
- ▶ Palliative chemotherapy to a ureteric tumour causing compression can reduce tumour size and relieve duodenal obstruction, allowing the patient to eat normally.

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