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Case Report

Metastatic Pancreatic Adenocarcinoma Presenting as Primary Ureteral Tumor

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Summary

Ureteral obstruction as a presenting finding of pancreatic adenocarcinoma is a rare occurrence,with only 8 prior reported cases. In this case report, we present the clinical and pathological diagnosis of a rare pancreatic adenocarcinoma presenting as ureteral metastasis.

Introduction

Pancreatic cancer is the fourth leading cause of cancer mortality with a 5-year survival rate of 6%[1]. This is largely explained by its aggressive behavior, with only 15-20% of local pancreatic cancers able to be resected at time of diagnosis[2]. Pancreatic cancer often spreads to distant sites, most commonly the liver and peritoneum [3]. Spread to the ureter, however, is rare. In fact, ureteral obstruction as a presenting finding of any distant primary tumor metastasis has only been reported in 400 cases, most discovered post-mortem [4]. To date, there have been 8 reported cases of pancreatic adenocarcinoma that presented as ureteral obstruction[4]. In this report, we describe the clinical presentation and the diagnostic challenges which ensued.This is followed by the histologic characteristics and follow-up. This case demonstrates that expedient diagnosis of metastatic pancreatic cancer of the ureter is difficult due to the inability of standard diagnostic techniques to confirm metastasis.

Case Review

A 61-year-old femaleformer smoker presented to primary care with a 10-day history of right abdominal pain and right flank pain that radiated to the right lower quadrant, worse after eating. Gallbladder Ultrasound (US)and Hepatobiliary Iminodiacetic Acid Scan (HIDA) scanwere normal. CT scan of the abdomen demonstrated multiloculatedrim-enhancing fluid surrounding the right kidney, right hydronephrosis, obstruction at the right Ureteropelvic Junction (UPJ) and slight right urothelial thickening.Perinephric fat-stranding was minimal.Acute kidney injury with creatinine = 1.87, up from baseline of 0.8, was diagnosed.Urinalysis (UA) was negative.Voided urine cytology was negative.Cystoscopy and right ureteroscopy revealed a narrow ureteral segment 2-3 cm distal to the UPJ, which was unable to accommodate the ureteroscope.Aspiration of renal pelvis fluid using open-ended ureteral catheter was used to obtain culture and cytology specimens.Renal barbotage was also performed.Brush biopsy was obtained of the narrowed segment, which had normal-appearing mucosa. Upon retrograde contrast injection, areas of proximal ureteral narrowing, a dilated renal pelvis and contrast extravasation at the level of narrowing was noted.Ureteral stent was placed. Previous right abdominal and flank pain resolved and creatinine normalized to 0.73 by post op day 3. Brush biopsy pathology was negative for cancer. Six-week follow up CT scan was similar to prior findings. With retroperitoneal fibrosis or benign ureteral stricture on the differential, a trial of stent removal was performed.Four days later,the patient reported intermittent abdominal and right flank pain and CT scan showed right renal obstruction. One week after removal, a new stent was placed. The etiology of ureteral obstruction was unclear, due to non-specific CT findings, negative brush biopsies and renal barbotage, requiring an open abdominal exploration. A very fibrotic retroperitoneum with ureteral adhesions and stricture in the proximal ureter was notedand frozen sections of the ureter showed inflammation but no evidence of cancer. Partial ureterectomywas attempted. Tension-free anastomosis was not feasible, andcompletion right nephronureterectomy was performed. Fur-

ther analysis of theureteral sections indicated duct-like infiltrating carcinoma.Neoplastic cellswere immune reactive for CEA, CK7, high molecular weight keratin and beta-catenin, and were negative for CK20, CD10, PAX8, GATA-3, ER, Breast Cocktail, CDX2, HER-2/Neu (Figure1). These findings suggested metastasis from a non-genitourinary source. Magnetic Resonance Cholangiopancreatography (MRCP) demonstrated bile duct narrowing at the head of the pancreas and abdominal MRI revealed a soft tissue mass at the head of the pancreas (Figure 2 C,D). PET/CT confirmed the hypermetabolic abnormality at the head of the pancreas(Figure 2 A,B).Fine needle aspiration (FNA) of the pancreatic mass revealed pancreatic adenocarcinoma.

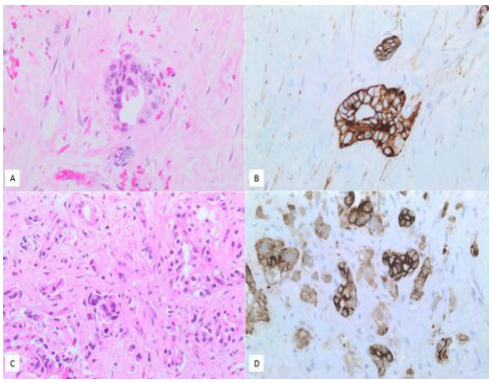

Figure 1: (A) Segment from the uteropelvic junction showing infiltrating glands within the smooth muscle of the right ureter.The glands are composed of pleomorphic tumor cells demonstrating irregular nuclear boarders and prominent nucleoli.Individual tumor cells are also seen.

(B) Tumor cells strongly express CK7.

(C) Pancreatic head mass, cell block.Glands are composed of tumor cells with irregular nuclear contours, irregular chromatin and marked variability in size within a desmoplastic background.Many infiltrating individual tumor cells are also identified.

(D) The tumor cells strongly express CK7.Further immune histochemical stains performed on both specimens showed similar staining patterns, with strong expression of MUC1 and no expression of CK20.

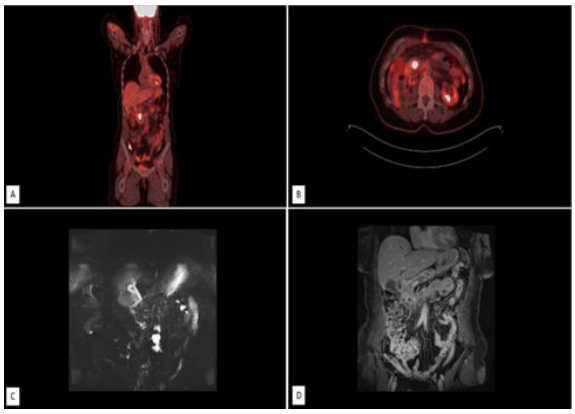

Figure 2: (A,B) Coronal and axial reconstructed images from PET/CT scan demonstrates increased metabolic activity at the head of the pancreas.

(C) Coronal T2 MRCP demonstrates bile duct narrowing at the head of the pancreas.

(D) Coronal T1 weighted imaging following intravenous gadolinium demonstrates a hypoenhancing mass measuring 2.2 cm at the head of the pancreas.

Comments

This case was complicated by the inability of standard diagnostic techniques to recognize pancreatic metastasis to the ureteral wall. The patient's initial right abdominal and flank pain, worse after eating, presents a vastdifferential diagnosis that includes gastrointestinal, genitourinary, infectious and metabolic etiologies. Initial HIDA, US and abdominal CT ruled out gastrointestinal, gallbladder and appendiceal pathology and suggested genitourinarydamage due to the multiloculated perirenal fluid and thickened urothelial wall. The patient was afebrile and had a normal white count suggesting a non-infectious source. Differential included post-stone urinoma, upper tract malignancy and retroperitoneal fibrosis, resulting in a thorough urologic workupand completion right nephroureterectomy. Metastasis from the pancreas was not

initiallyconsidereddue to the rarity of distant primary cancers metastasizing to the ureter, with only 8 cases of pancreatic adenocarcinoma presenting as ureteral obstruction[4]. In addition, there was low clinical index of suspicion for a pancreatic tumor as the patient did not report classic symptoms of asthenia, weight lossor anorexia, and did not present with jaundice nor hepatosplenomegaly[5].Finally, abdominal CT did not initially show a pancreatic mass. The paucity of cases, combined with the clinical symptoms and imaging indicating genitourinary pathology, led us away from the idea that the ureteral obstruction had a pancreatic origin. The diagnosis of pancreatic metastasis was made more difficult by the fact thata standard urologicworkupusing cystoscopy, voided urine cytology, ureteral lavage, ureteroscopy, and ureteral brush biopsy often does not detect the metastatic cells.According to Arvind et al,metastasis to the ureter most commonly involves theperiureteral adventitial layer, rather than the mucosal layerthat is visualized,brushed and washed in the urologic evaluation[4].Therefore, the only way to definitively diagnose pancreatic metastasis is viafull thickness tissue biopsy and histopathological examination, which was completed post-nephroureterectomy.

In summary, ureteral obstruction secondary to pancreatic metastasis is very rare. Standard urological diagnostic techniques do not often readilyidentify pancreatic metastasis to the ureter, making diagnosis difficult without adequate imaging and tissue biopsy.This makes it important to consider metastasis from a distant primary tumor when ureteral obstruction presents without obvious genitourinary etiology.

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