Renal pelvic cancer with spleen invasion arising in horseshoe kidney; a case report

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1. Introduction

Horseshoe kidney is the most common of all renal fusion anomalies, occurring in 0.25% of the population. The incidence of renal pelvic cancer is reported as 7.7%, whereas the incidence associated with horseshoe kidney increases to approximately 19.8%. We report a case of renal pelvic cancer in a horseshoe kidney with spleen invasion and review the cases that have been previously documented.

2. Case presentation

A 75-year-old man presented with horseshoe kidney and hydronephrosis. He had a history of cerebral infarction that had been treated with an antiplatelet drug. His body mass index (BMI) was 18.8 (kg/m²). He noticed asymptomatic gross hematuria three times within the previous three years. At each of the first two times, no apparent malignancies were detected. However, the third urine cytology examination revealed possible carcinoma. Pyuria was also detected using urinalysis. Enhanced computed tomography (CT) imaging showed a solid mass 50 mm in diameter in the left renal pelvis, and the mass had directly invaded the spleen (Fig. 1a–c). Retrograde pyelography showed no filling defect, and a separate urine cytology examination revealed no malignancy. Therefore, we performed a CT-guided biopsy of the left renal pelvic mass; the pathological diagnosis was urothelial carcinoma (high grade, G3). Based on these results, the clinical stage was determined to be cT4N0M0 (splenic invasion).

He received three cycles of gemcitabine plus cisplatin (GC) chemotherapy. As the tumor shrank 48% from the baseline size, we conducted a left nephroureterectomy with transection of the isthmus in the horseshoe kidney and splenectomy. The kidney isthmus was transected using an automatic anastomosis device, and we sewed the urinary tract using the intussusception method. The left kidney adhered strongly to the chest wall, spleen, and quadratus lumborum. No swollen lymph nodes were found. The operation time and amount of blood loss were 351 minutes and 1746 ml, respectively.

Macroscopically, the pelvic tumor had invaded the perinephric fat tissue. Moreover, it seemed to extend to the edge of the spleen directly. A final pathological diagnosis was urothelial carcinoma (G3, pT4, pR0, pL0, pV1, pN0) with spleen invasion (Fig. 2a and b). His perioperative course was uneventful. However, the tumor recurred within three months after the surgery and he was treated with paclitaxel, and ifosfamide plus nedaplatin as second-line chemotherapy. Thereafter, his condition worsened and died 14 months after surgery due to disease progression.

3. Discussion

It is reported that the incidence of renal pelvic cancer increases at least three-fold in the horseshoe kidney compared with the “normal” kidney. Buntley et al. hypothesized that urinary tract infection or urinary stasis could generate carcinogenesis more easily in the horseshoe kidney. To our knowledge, more than 48 cases of renal pelvic cancer in horseshoe kidneys have been previously reported, as summarized in Table 1. Among the 48 cases, 22 were evaluated on the basis of pathological stages (pT) as pTa-1, pT2, and pT3 (nine, three, and six cases, respectively). Including our case, pT4 was identified in three cases, with all three cases demonstrating invasion into the perinephric fat tissue. However, we did not identify invasion into the spleen in the other pT4 cases.
The third patient had bladder invasion and died 12 days after surgery due to increased metastasis to the lungs. All pT4 cases had a poor prognosis, and the development of combined modality therapy is required.

We propose two hypotheses as to why the renal pelvic cancer could directly invade the spleen based on pathological and diagnostic imaging. First, this case had high-grade cancer. Pathological findings revealed that this tumor showed an infiltrative growth pattern of minute alveolar, and the marginal range of the lesion appeared to be indistinct. Accordingly, this case was considered to be a high-grade invasive urothelial carcinoma. Second, the anatomical difference between a horseshoe kidney and a normal kidney is a factor. The major axis of the horseshoe kidney faces inward, cranially, which in turn forms upside down. Moreover, the horseshoe kidney has a morphologic feature in which the hilus of the kidney faces cranially because of the anomaly of the axis. We postulate that the tumor exceeded the blockage of the renal cortex, and the expanded pelvic cancer could invade the spleen directly. Based on postoperative chemotherapy; the third patient had bladder invasion and died 12 days after surgery due to increased metastasis to the lungs. All pT4 cases had a poor prognosis, and the development of combined modality therapy is required.

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Fig. 1. a Weak enhanced solid mass 5 cm in diameter in the left renal pelvis of the horseshoe kidney. The tumor seemed to invade the spleen. (Yellow arrow). b. Coronal section of enhanced CT image showed: The renal pelvic tumor could invade the spleen directly. (Yellow arrow). c. Horseshoe kidney.

Fig. 2. a Histological findings: Splenic invasion is recognized from the invasive front of the tumor. Original magnification ×100. b. Histological findings: Splenic invasion is recognized from the invasive front of the tumor. Original magnification ×200.
these hypotheses, the renal tumor in this case could invade the spleen directly.

4. Conclusion

We report the clinical course of a patient with renal pelvic cancer with invasion of the spleen associated with a horseshoe kidney. This could be associated with several morphological features unique to the horseshoe kidney, or to the chronic inflammation associated with this anatomical anomaly. To assess the prognosis of pelvic cancer arising in the horseshoe kidney accurately, we should accumulate and analyze more cases.

Conflicts of interest

None.

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Table 1

| Year       | Number of cases | pT stage |
|------------|----------------|----------|
|            |                | pTa-1 | pT2 | pT3 | pT4 | unknown |
| Buntley1   | 1895–1976      | 22    | –   | –   | –   | 22      |
| Matsushita et al.3 | 1975–2012 | 25    | 9    | 3   | 6   | 4        |
| Our case   | 2016           | 1     | –   | –   | –   | 1       |
| Total      | 48             | 9     | 3   | 6   | 4   | 26      |

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