Mixed carcinoma transdifferentiation of bladder urothelial carcinoma after chemotherapy: A case report

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Abstract. Both small cell carcinoma (SCC) and sarcomatoid carcinoma are rare malignancies of the bladder, with their simultaneous presence being exceedingly rare. The present study reports the case of a patient who underwent multiple transurethral resections for bladder tumors, with initial diagnoses consistently indicating urothelial carcinoma (UC). However, the pathology from the fourth surgery revealed a complex presentation of mixed bladder cancer, primarily characterized by sarcomatoid carcinoma, but also including SCC and traditional UC. After the operation, it was recommended that the patient should receive neoadjuvant chemotherapy combined with radical cystectomy as a treatment strategy, but due to the poor physical condition of the patient, this was refused; therefore, the patient was instead followed up closely for a long period. However, the patient died of multiple organ failure 9 months after the operation. After conducting an extensive review of the literature, it was concluded that UC undergoing transdifferentiation into SCC of the bladder and sarcomatoid carcinoma following chemotherapy is well founded. Immunotherapy represents a promising new treatment avenue for these highly malignant and advanced-stage tumors, offering substantial potential for research. Additionally, studies investigating the mechanisms underlying promising therapeutic regimens need to be conducted in greater depth in the near future.

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Abbreviations: SCCB, small cell carcinoma of the bladder; UC, urothelial carcinoma; TURBT, transurethral resection of a bladder tumor; BCG, Bacillus Calmette-Guerin

Key words: transdifferentiation, bladder cancer, SCC, sarcomatoid carcinoma, chemotherapy

Introduction

Small cell carcinoma of the bladder (SCCB), also known as neuroendocrine carcinoma, is a rare malignant tumor of bladder, accounting for ~0.35-0.70% of all bladder malignant tumors. Sarcomatoid carcinoma of the bladder has similar rarity and a high degree of malignancy, and the pathogenesis is still elusive. Both diseases are more common in men, and the ratio of men to women in SCCB is ~3.3:1 (1). The presence of SCCB or sarcomatoid carcinoma of the bladder usually indicates an advanced stage in the disease course. In total, 94% of patients with SCCB have already been initially diagnosed with an invasive malignant tumor, and 67% of them have metastasis. The 5-year survival rate is ~8% and the overall median survival time is 9.8 months (2). According to the literature, the survival time of 12 patients with sarcomatoid carcinoma of the bladder was 1 to 48 months (mean, 17 months), and most of them died of metastasis in the lymph nodes, lungs, chest, brain, liver and bone (3).

In the present case, the patient's pathology from the initial three transurethral resections of a bladder tumor (TURBT) indicated urothelial carcinoma (UC). However, subsequent pathology revealed a transition to a mixed carcinoma comprising UC, SCC and sarcomatoid carcinoma. The coexistence of these distinct carcinomas is extraordinarily uncommon, and the underlying mechanisms and reasons for such pathological transitions remain poorly understood. The present study reviews the current literature and discusses the advancements in treatment options for both SCCB and sarcomatoid UC, as well as examining the potential causes of such pathological transdifferentiation.

Case report

An 81-year-old patient was admitted to Hangzhou First People's Hospital (Hangzhou, China) in August 2022 due to a newly discovered bladder lesion by regular cystoscopy. Pre-operative examinations were followed by holmium laser resection of the bladder tumor. A solid, irregularly shaped

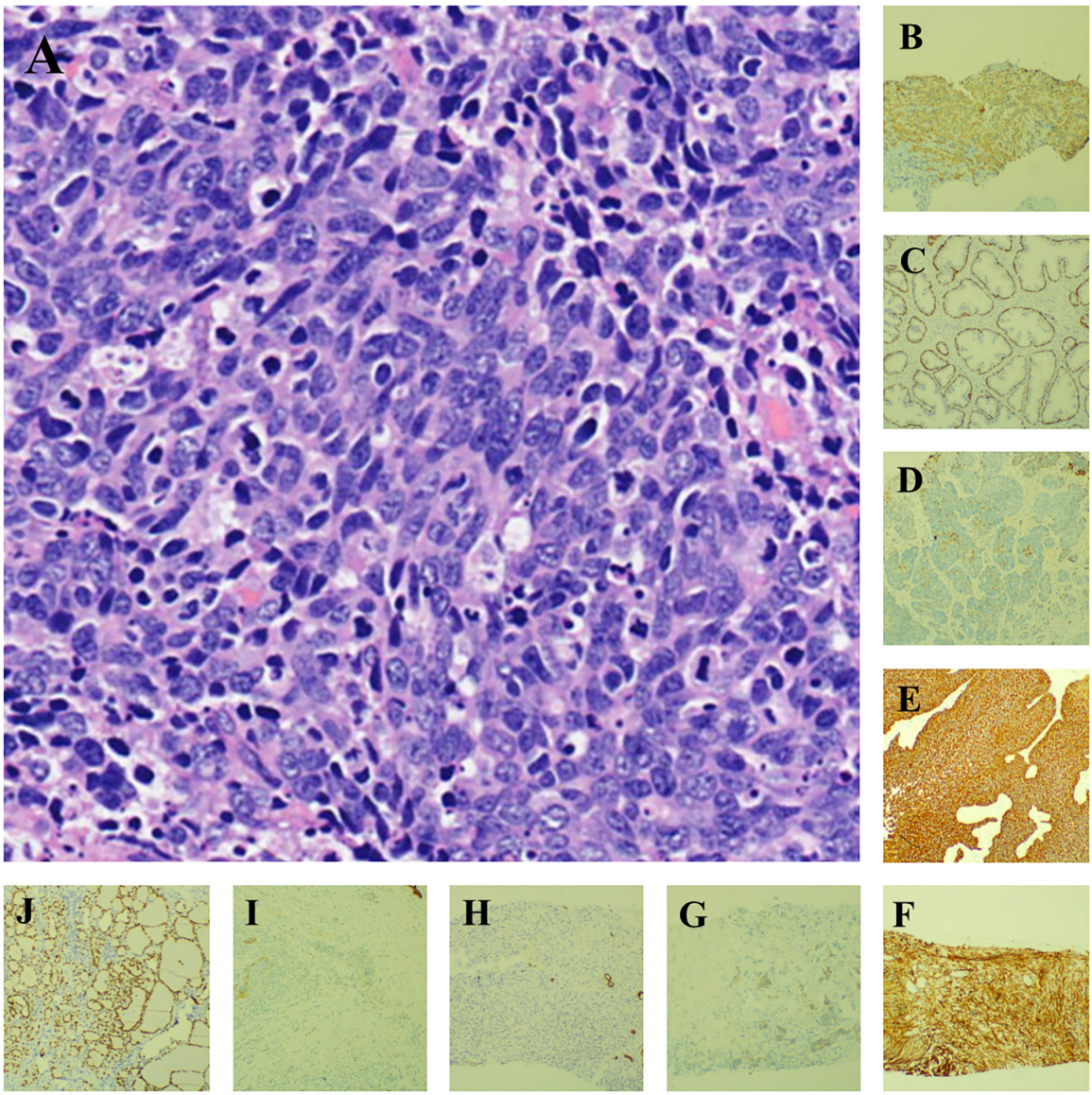

Figure 1. H&E staining and immunohistochemical analysis of SCCB. (A) Microscopic manifestations of SCCB. The cancer cells are diffuse and may be cord-like or trabecular. Most of the cells are rhombic or oat-shaped, and the nuclei are deeply stained and rich in morphology, which can be in various forms. There is abundant chromatin in the nuclei, mostly in fine particles. Nucleolar staining shows mitotic figures. Immunohistochemical analysis: (B) CK(+), (C) 34βE12(+), (D) GATA binding protein 3(+), (E) CK7(+), (F) CD56(+), (G) synaptophysin(+), (H) chromogranin A foci(+), (I) neuron-specific enolase(-) and (J) thyroid transcription factor 1(slightly +). SCCB, small cell carcinoma of the bladder; CK, cytokeratin.

mass ~2x2 cm was discovered on the left side of the bladder during surgery. Postoperative pathology revealed a mixed carcinoma of the bladder, consisting of 40% SCC and 60% sarcomatoid carcinoma, with two small areas of UC *in situ*. H&E staining and immunohistochemical analysis identified the following results for SCC (Fig. 1A): Cytokeratin (CK) (+) (Fig. 1B), 34βE12(+) (Fig. 1C), GATA binding protein 3 (GATA-3)(+) (Fig. 1D), CK7(+) (Fig. 1E), CD56(+) (Fig. 1F), synaptophysin (Syn)(+) (Fig. 1G), chromogranin A (CgA) (focal +) (Fig. 1H), neuron-specific enolase (NSE)(-) (Fig. 1I) and thyroid transcription factor-1(slight +) (Fig. 1J). Detailed staining methods are provided in the Supplementary material and primary antibodies are listed in Table SI. H&E staining and immunohistochemical analysis identified the following results for sarcomatoid carcinoma (Fig. 2A): CK(-) (Fig. 2B), GATA-3(slight +) (Fig. 2C), p63(+) (Fig. 2D), 34βE12(-) (Fig. 2E), CD56(-) (Fig. 2F), S100(-) (Fig. 2G), cyclin-dependent kinase 4(partial +) (Fig. 2H), E3 ubiquitin-protein ligase Mdm2(+) (Fig. 2I), smooth muscle actin(-) (Fig. 2J), Ki-67(+) (Fig. 2K) and vimentin(++) (Fig. 2L).

The patient had a history of bladder cancer treatment, including a TURBT at Ruijin Hospital (Shanghai, China) in May 2010, with postoperative pathology indicating non-invasive high-grade UC (Fig. 3). The H&E-stained section showed urothelial cells growing in papillary/proliferative architectures, with markedly increased cell layers and disordered arrangement. The tumor cells exhibited a significantly elevated nuclear-to-cytoplasmic ratio, marked nuclear pleomorphism with variation in size and shape, coarse and hyperchromatic chromatin, visible nucleoli and mitotic figures, as well as occasional tumor giant cells, consistent with the morphological features of high-grade

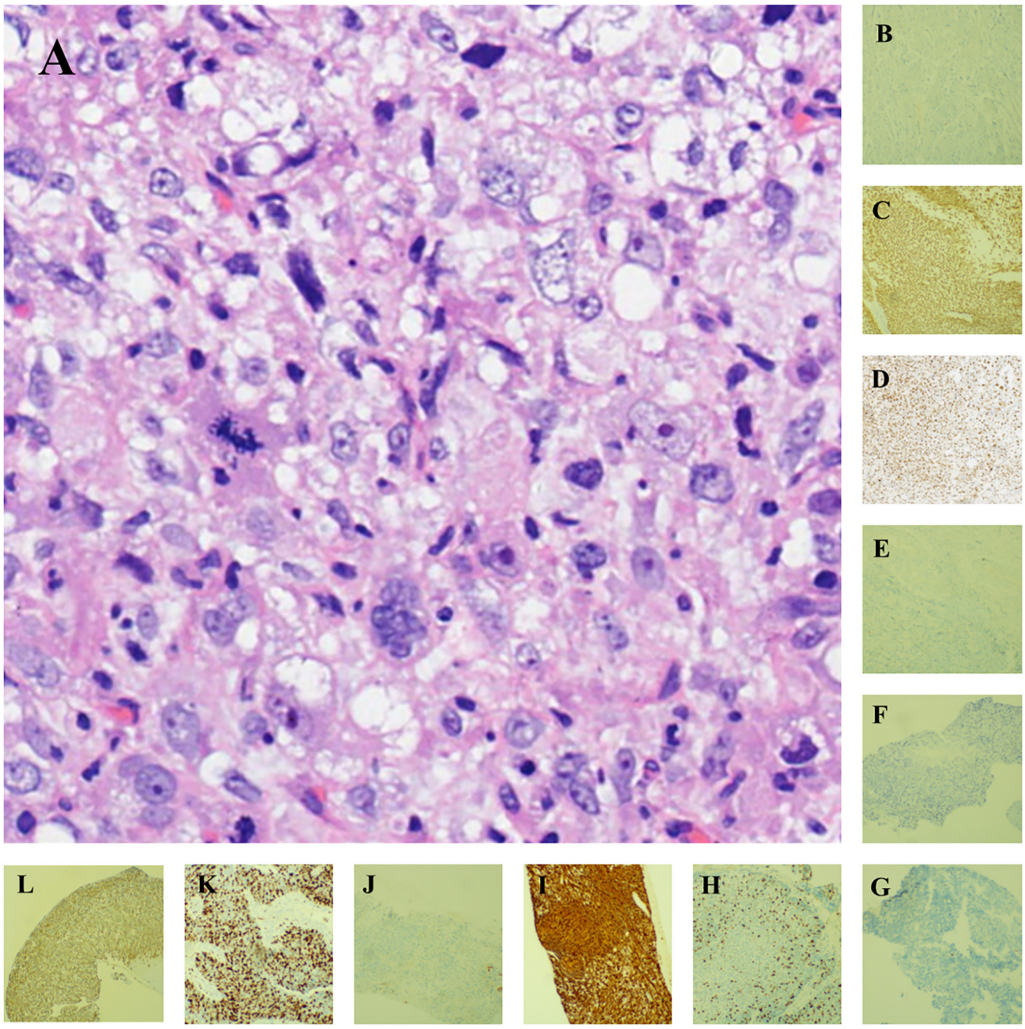

Figure 2. H&E staining and immunohistochemical analysis of sarcomatoid carcinoma. (A) Microscopic findings of sarcomatoid carcinoma (x400 magnification; hematoxylin and eosin staining). The cells are spindle-shaped, polygonal and triangular, with unclear boundaries. The nuclei are mostly spindle-shaped, round-like and irregular, with different sizes and significant atypia. The chromatin is thick, and singular nuclei, megakaryocytes and multinucleated nuclei are present in mulberry, flower-ring or irregular shapes. Immunohistochemical analysis: (B) Cytokeratin(-), (C) GATA binding protein 3(slightly +), (D) p63(+), (E) 34βE12(-), (F) CD56(-), (G) S100(-), (H) Cyclin-dependent kinase 4(partially +), (I) E3 ubiquitin-protein ligase Mdm2(+), (J) smooth muscle actin(-), (K) Ki-67(+) and (L) vimentin(++).

UC. Post-surgery, the patient received intravesical Bacillus Calmette-Guerin (BCG) chemotherapy. A recurrence in May 2015 led to another TURBT and the pathology then showed high-grade invasive UC. The patient underwent three cycles of intravenous chemotherapy, which were discontinued due to intolerance. A 2016 biopsy during a re-examination via cystoscopy at Hangzhou First People's Hospital confirmed the presence of invasive high-grade UC with chemotherapy-induced changes (Fig. 4A). The patient declined a radical cystectomy, opting for another TURBT. Immunohistochemistry from this last procedure showed the following results: Anti-CK1/anti-CK 3(+) (Fig. 4B), GATA-3(+) (Fig. 4C), CK7(+) (Fig. 4D), CK20(-) (Fig. 4E), clone KP1(poor) (Fig. 4F), Ki-67(~20%) (Fig. 4G), phosphoglucomutase 1(+) (Fig. 4H) and p63(+) (Fig. 4I).

Following the surgical procedure, the clinical team suggested a treatment plan comprising neoadjuvant chemotherapy followed by radical cystectomy. Owing to the patient's debilitated physical state, this approach was declined. As an alternative, the patient was placed under rigorous long-term

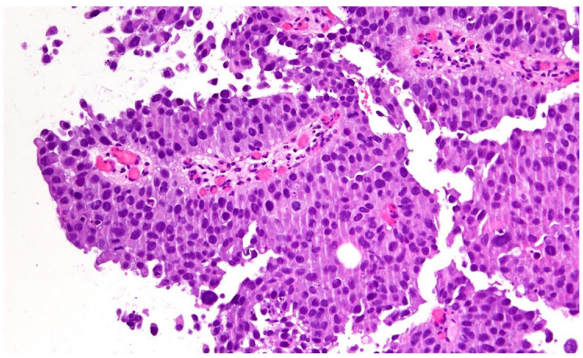

Figure 3. H&E staining analysis of non-invasive high-grade UC in May 2010. Non-invasive high-grade urothelial carcinoma of the bladder detected in May 2010 (x200 magnification; hematoxylin and eosin staining). This H&E-stained section shows urothelial cells growing in papillary/proliferative architectures, with markedly increased cell layers and disordered arrangement. The tumor cells exhibit a significantly elevated nuclear-to-cytoplasmic ratio, marked nuclear pleomorphism with variation in size and shape, coarse and hyperchromatic chromatin, visible nucleoli and mitotic figures, as well as occasional tumor giant cells, consistent with the morphological features of high-grade UC. UC, urothelial carcinoma.

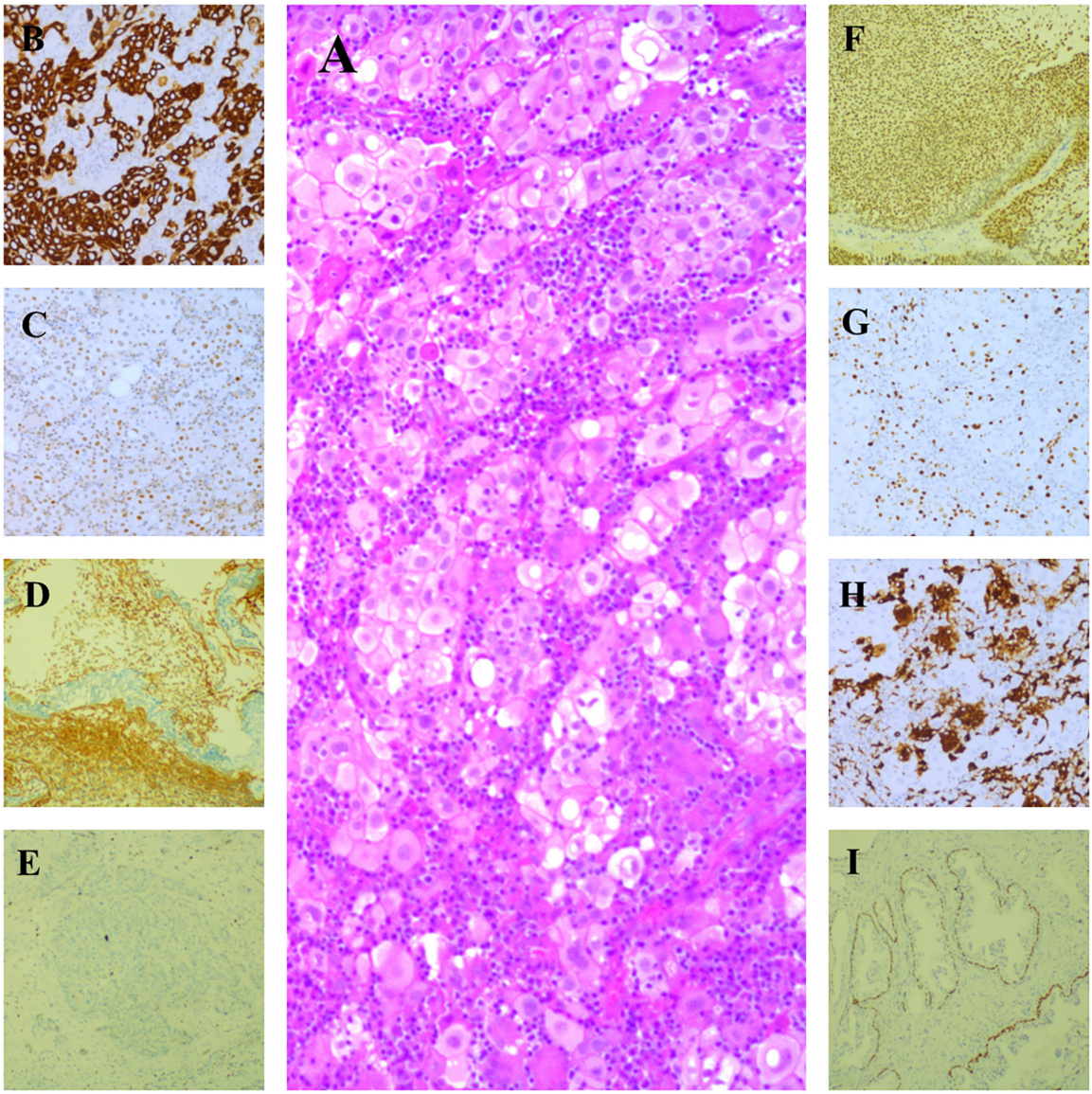

Figure 4. H&E staining and immunohistochemical analysis of non-invasive high-grade UC in May 2016. (A) Biopsy pathology of the patient showing invasive high-grade UC with chemotherapy-induced changes in May 2016 (x100 magnification; hematoxylin and eosin staining). Immunohistochemical analysis: (B) Anti-cytokeratin antibody 1/anti-cytokeratin antibody 3(+), (C) GATA binding protein 3(+), (D) CK7(+), (E) CK20(-), (F) Clone KP1(poor), (G) Ki-67(+; ~20%), (H) phosphoglucomutase 1(+) and (I) p63(+). UC, urothelial carcinoma; CK, cytokeratin.

surveillance. Nonetheless, the patient succumbed to multiple organ failure 9 months after the surgery.

Discussion

The present study reports a rare and highly aggressive case of mixed differentiation in the modern context of chemotherapy and immunotherapy, highlighting its clinical challenges and potential biological significance. The pathology from the initial three TURBT procedures indicated UC. However, subsequent pathology revealed a transition to a mixed carcinoma comprising UC, SCC and sarcomatoid carcinoma. This change occurred after the patient received intravesical BCG and intravenous chemotherapy treatments. Although a previous report documented SCC transdifferentiating from UC following chemotherapy (4), it is rare for UC to transdifferentiate into a mixed carcinoma that included both SCC and sarcomatoid carcinoma at the same time after chemotherapy.

SCCB is a rare and highly aggressive form of cancer, constituting <0.7% of all bladder tumors (5). Morphologically, it resembles small cell lung carcinoma. Immunohistochemical analysis typically reveals the presence of both epithelial and neuroendocrine markers. Epithelial antigens such as epithelial membrane antigen are present in ~80% of cases, and CK in ~60% (6). NSE is the most frequently observed neuroendocrine marker, found in 25-100% of cases, followed by CgA (22-89%) and Syn (67-76%) (7), among others, such as neurofilaments, lymphocyte-7 and S-100. Clinically, primary SCCB can be divided into simple and mixed types. The mixed type, which constitutes 29-66.7% of cases, often includes UC (58%) as the most common co-occurrence, followed by adenocarcinoma (14%), sarcomatoid carcinoma (4%), micropapillary carcinoma (4%), squamous cell carcinoma (3%) and plasma

cell carcinoma (1%). A recent study has indicated that there is no significant difference in survival rates between patients with simple and mixed SCCB, with survival predominantly influenced by the stage of the tumor rather than its type (8). SCCB typically presents at advanced tumor stages.

Due to its uncommon occurrence, there is no established standard clinical treatment for SCCB. The National Comprehensive Cancer Network suggests that the treatment protocols for all SCCs should align with those used for small cell lung cancer (9). This includes chemoradiotherapy or neoadjuvant therapy for all patients, regardless of tumor stage, with radical cystectomy recommended when the cancer is localized to the bladder (9,10). Studies have shown that preoperative neoadjuvant chemotherapy in conjunction with radical cystectomy benefits patients with early-stage SCCB, enhancing their survival prospects (11,12). However, due to its aggressive nature, SCCB is susceptible to early recurrence. Immunotherapy could represent a promising treatment avenue for primary SCCB in the future. For example, Yang *et al* (13) noted that primary SCCB expresses the immune checkpoint adenosine receptor A2A, which can be targeted to reduce immune infiltration. Additionally, Wilde *et al* (14) reported a case in which primary SCCB was successfully treated with pembrolizumab, and Hatayama *et al* (15) also documented the successful application of pembrolizumab in treating recurrent SCCB. However, the aforementioned studies are only case reports, and large-scale studies are required to verify the efficacy of immunotherapy in treating primary SCCB.

Sarcomatoid carcinoma of the bladder is also a highly aggressive malignant tumor, representing only 0.1-0.3% of all bladder cancer cases (16). This cancer type is distinguished by the simultaneous presence of epithelial cells and sarcomatoid spindle cells (17), often accompanied by high-grade papillary/undifferentiated UC, and may include other epithelial tumor subtypes such as small cells or squamous cells (18). The presence of such varied histology tends to correlate with early disease progression, a high recurrence rate and reduced survival time (19). Therefore, cases of bladder sarcomatoid carcinoma are of high grade and advanced stage at diagnosis. In cases of sarcomatoid carcinoma of the bladder, vimentin is commonly positive in epithelial cells, and markers such as high molecular weight cytokeratin, p63 and GATA-3 may also be positive in the sarcomatoid components (20). In the present case, vimentin expression was marked as (++), GATA-3 was slightly positive (+), and p63 and Ki-67 were also positive (+).

Currently, the optimal treatment for sarcomatoid carcinoma of the bladder remains under debate due to limited patient data. This type of cancer is often diagnosed at an advanced stage, and is characterized by local invasion, lymph node involvement or distant metastasis (16). Current academic consensus supports surgical resection as the primary treatment, with options including cystectomy or TURBT, although cystectomy generally shows better outcomes regarding tumor survival (21,22). In different studies, neoadjuvant chemotherapy followed by cystectomy or a combined approach of surgery and chemoradiotherapy has been shown to enhance survival rates (23,24). Additionally, molecular targeted therapies have demonstrated significant potential in treating sarcomatoid carcinoma. Studies on mesenchymal non-small cell lung cancer reveal that sarcomatoid differentiated tissues are more likely to express programmed cell death protein-1/programmed death ligand-1 (PD-1/PD-L1), suggesting that patients with differentiated sarcomatoid tumors could be candidates for anti-PD-1/PD-L1 therapy (16,25). For instance, nivolumab, a PD-1 inhibitor, has been effective in treating brain metastases and other metastatic sites post-surgery in pulmonary sarcomatoid carcinoma (26). Furthermore, although renal sarcomatoid carcinoma differs from renal clear cell carcinoma, Başeskioglu *et al* (3) found that everolimus, a drug used for renal clear cell carcinoma, also shows efficacy in renal sarcomatoid carcinoma. The effectiveness of molecular targeted therapies for bladder sarcomatoid carcinoma requires further research and validation, but given their success in other sarcomatoid cancer types, they hold promising potential for application.

Several theories have been proposed in the literature to explain the origins of SCCB (27-31). The first theory suggests that SCCB arises from neuroendocrine cells within the bladder mucosa. These cells are part of the human amine precursor uptake and decarboxylation system, similar to K cells in the digestive system, C cells in the thyroid, chromaffin cells in the adrenal gland and Langerhans cells in the pancreas. These cells are capable of developing various benign and malignant tumors, such as adenomas, carcinoids, apudomas and SCC (27). The second theory posits that SCCB originates from pluripotent stem cells capable of differentiating into various tissue types, and the origin of sarcomatoid carcinoma is the malignant transformation of stromal cells, leading to a 'collision tumor' composed of both epithelial and stromal tumors. In the current literature, 66% of SCCB were mixed with other cancers, with the most common being UC at 40% and Urothelial Carcinoma *In Situ* (UCIS) at 32%, highlighting a close association between SCCB and UC (28). Certain studies suggest that sarcomatoid carcinoma may originate from monoclonal tumors with diverse differentiation potentials, with components deriving either from urothelial tumors or from pluripotent stem cells capable of differentiating into various cell types (29,30). Molecular genetic studies have demonstrated that SCCB, UC, sarcomatoid carcinoma and UC share a common clonal origin. Cheng *et al* (30) investigated the loss of heterozygosity and chromosome X inactivation patterns across the three components of SCCB, UC and sarcomatoid carcinoma. The findings revealed a high degree of similarity among these components, supporting the theory that SCCB and UC originate from a common clonal precursor. According to the third theory, SCCB originates from metaplasia of transitional epithelial cells (31), where abnormal proliferation can lead to the formation of Brunn's nests and buds. Glandular metaplasia can evolve into cystitis glandularis, and argyrophil cells with neuroendocrine properties have been identified within both Brunn's nests and cystitis glandularis. Indeed, a study showed that microRNA-145 can reprogram conventional UC cells into pluripotent stem cells, which can then differentiate into neuroendocrine, glandular and squamous cell lineages (32), which is not inconsistent with the view that SCCB, sarcomatoid carcinoma and UC have the same clonal origin. In the present case, the pathology of the patient's most recent sample revealed two small foci of UCIS, with three distinct tumor components present, whereas only UC components were identified in the previous three pathologies. This supports the likelihood that UC underwent

transdifferentiation into SCCB and sarcomatoid carcinoma following chemotherapy. Currently, the pathologies of SCC and sarcomatoid carcinoma are complex, and their pathogenesis remains poorly understood. Future studies should aim to further elucidate their molecular mechanisms using advanced techniques such as single-cell sequencing, spatial proteomics and metabolomics.

Finally, the temporal correlation between chemotherapy and the occurrence of mixed carcinoma suggests a potential association between therapeutic stress and transdifferentiation in this case, which requires further verification. At present, there is no definitive treatment protocol for mixed malignant tumors of the bladder, with treatment generally guided by the approaches used for the primary tumor. Immunotherapy represents a promising new treatment avenue for these highly malignant and advanced-stage tumors, offering substantial potential for research. For the present case, it was recommended that the patient should undergo preoperative neoadjuvant chemotherapy combined with radical cystectomy as a treatment strategy. However, the poor physical condition of the patient precluded the use of chemotherapy or radical cystectomy. Considering the high recurrence rate associated with this disease, stringent postoperative follow-up is essential to monitor for any signs of recurrence and to manage the patient's condition effectively. This comprehensive follow-up is crucial due to the aggressive nature of the tumor and the limited treatment options for the patient, aiming to ensure the best possible management and quality of life.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

LZ and JH contributed to the conception of the case report, collected clinical data, analyzed patient information, drafted the original manuscript, and approved the final submitted version. ZD, JS and RH contributed to the acquisition and interpretation of clinical data, participated in literature retrieval and critical revision of the manuscript for important intellectual content, verified relevant reference information, and approved the final manuscript. XW and KW designed the clinical research scheme, performed the relevant surgery on the patient, supervised the overall conduct of this case report, critically revised the manuscript, took accountability for all clinical and textual contents, and gave final approval for publication. LZ, JH, ZD, JS, RH, XW and KW have confirmed the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

All procedures were performed in accordance with the Declaration of Helsinki of the World Medical Association. The study was approved by the Ethics Committee of Hangzhou First People's Hospital (ethics approval no. 2026ZN265-1).

Patient consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Competing interests

The authors declare that they have no competing interests.

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