Undifferentiated pelvic sarcoma 5 years post-radiochemotherapy for rectal adenocarcinoma: A case report

YANG YANG[1] and YUBIN LONG[2]

[1]Department of Oncology, The Central Hospital of Shaoyang, Shaoyang, Hunan 422000, P.R. China;
[2]Department of Spinal Surgery, The Central Hospital of Shaoyang, Shaoyang, Hunan 422000, P.R. China

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Abstract. Radiation-induced sarcoma (RIS) is a rare, life-threatening late complication of radiotherapy, typically diagnosed 3-5 years post-treatment. It is associated with a poor prognosis and poses diagnostic challenges due to non-specific imaging and clinical features that mimic tumor recurrence. As the long-term survival rates of patients with rectal cancer improve, recognizing late-onset treatment-related malignancies becomes increasingly paramount. The present study describes the case of a 62-year-old male patient who presented to Shaoyang Central Hospital (Shaoyang, Hunan, China) in February 2019 and underwent neoadjuvant chemoradiotherapy followed by radical resection for rectal adenocarcinoma in June 2019. After 5 years, he presented with buttock pain in December 2024. Computed tomography (CT) imaging revealed a sacrococcygeal soft tissue mass within the prior radiation field. A biopsy confirmed an undifferentiated sarcoma, histologically distinct from the primary carcinoma, fulfilling modified Cahan criteria for RIS. Despite diagnosis, the patient declined further treatment due to concerns about toxicity and exhibited disease progression. A follow-up examination in June 2025 showed that the masses had increased in number and size. The present case underscores the importance of long-term surveillance in rectal cancer survivors. A re-biopsy of suspicious masses is key to differentiate RIS from recurrence. Early recognition may improve patient outcomes, although current therapeutic options, such as standard radiotherapy and chemotherapy, remain limited. Immunotherapy and targeted therapy are considered as second-line treatment options. However, due to factors such as physical and financial conditions, this patient did not choose to continue the treatment.

Correspondence to: Dr Yubin Long, Department of Spinal Surgery, The Central Hospital of Shaoyang, Hongqi Street, 360 Baoqing Road, Daxiang, Shaoyang, Hunan 422000, P.R. China
E-mail: spineyb07@tmu.edu.cn

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Introduction

Colorectal cancer (CRC) is the third most commonly diagnosed malignancy globally, with its incidence continuing to rise. There were ~1.93 million new CRC cases and 904,000 deaths worldwide in 2022 (1). Concurrently, advancements in multimodal screening and treatment strategies have improved survival rates, resulting in a growing population of long-term CRC survivors (2). As this population expands, precise perioperative risk stratification and structured long-term follow-up have become increasingly key to optimize patient outcomes and proactively manage late treatment-associated complications (3,4). For locally advanced rectal cancer, neoadjuvant chemoradiotherapy followed by total mesorectal excision constitutes the standard of care, effectively decreasing local recurrence (5). While radiotherapy is integral to achieving tumor control, its potential long-term sequelae cannot be overlooked. Among these sequelae, the development of radiation-associated second primary malignancies (SPMs) represents a rare, yet severe complication, affecting 5-10% of long-term cancer survivors (6-8). The association between pelvic irradiation and SPMs risk in rectal cancer survivors has been debated, with population-based studies yielding conflicting conclusions (9,10). Certain evidence suggests a marginally elevated overall risk (4-8%) of SPMs in this population compared with matched controls, with specific increases noted in cancer of the uterus and bladder (10-13).

Radiation-induced sarcoma (RIS) arising within the treated field is a rare event following rectal cancer treatment, with only a few cases reported to date (14). Its clinical and radiological presentation typically mimics local recurrence or metastasis, creating a diagnostic dilemma that necessitates histological confirmation (15). This underscores the importance of long-term vigilance, systematic follow-up and a low threshold for re-biopsy of new masses within the irradiation field to prevent misdiagnosis and guide appropriate management. The present study describes a case of high-grade undifferentiated sarcoma occurring in the sacrococcygeal region 5 years following curative radiotherapy for rectal adenocarcinoma. The present case report aimed to enhance clinical awareness, illustrate the application of diagnostic criteria and emphasize the role of biopsy in differentiating this entity from tumor recurrence.

Case report

In February 2019, a 62-year-old male patient presented to Shaoyang Central Hospital (Shaoyang, China) with altered bowel habits persisting for >1 month. A computed tomography (CT) scan performed using a Philips Ingenuity 64-row/128-slice CT scanner (Philips Healthcare) revealed a rectal mass located 4-13 cm from the anal verge (Fig. 1A and B). A colonoscopy confirmed a suspicious lesion and a biopsy revealed severe atypical hyperplasia with malignant transformation. Following multidisciplinary discussion, the patient received neoadjuvant concurrent chemoradiotherapy: External beam radiotherapy at 50 Gy in 25 fractions (Fig. 1C), synchronized with oral capecitabine (825 mg/m^2 twice daily on radiotherapy days). In addition, two cycles of post-operative adjuvant intravenous oxaliplatin (130 mg/m^2 on day 1) and oral capecitabine (1,000 mg/m^2 twice daily on days 1-14), followed by a 7-day rest period. In June 2019, the patient underwent a laparoscopic Miles procedure. Final pathology revealed well-differentiated rectal adenocarcinoma (4.5 cm; fixed in 10% neutral buffered formalin at room temperature for 24 h; 4-μm-thick sections; hematoxylin and eosin staining at room temperature, with hematoxylin staining for 5 min and eosin staining for 1 min; light microscopy), pathological T2N0 stage (pT2N0; tumor invasion into the muscularis propria without regional lymph node metastasis), with negative margins and no lymphovascular or perineural invasion according to the American Joint Committee on Cancer Staging Manual, 8th Edition (16). Immunohistochemistry yielded the following results: Cytokeratin (CK)7(-), CK20(-), villin(+), caudal-type homeobox 2 (CDX2) (+), mutS homolog 2 (MSH2) (+), MSH6(+), mutL homolog 1 (MLH1) (+), post-meiotic segregation increased 2 (PMS2) (+), indicating mismatch repair (MMR)-proficient status. Histopathological examination and immunohistochemistry were performed on formalin-fixed, paraffin-embedded tissue sections. Briefly, tissue samples were fixed in 10% neutral-buffered formalin at room temperature for 24 h, embedded in paraffin, sectioned at 3 μm and mounted on adhesive glass slides. The sections were baked at 58-60˚C for 2-3 h or overnight, deparaffinized in xylene three times for 5 min each, rehydrated through a graded ethanol series and washed with phosphate-buffered saline. Antigen retrieval was performed using citrate buffer (pH 6.0), or EDTA buffer (pH 8.0). Endogenous peroxidase activity was blocked with 3% hydrogen peroxide at room temperature for 10 min. The sections were incubated with ready-to-use, primary antibodies at room temperature for 60 min against CK (cat. no. Kit-0009), EMA (clone E29; cat. no. Kit-0011), S100 (rabbit polyclonal; cat. no. RAB-0150), CD34 (clone MX123; cat. no. MAB-1076), HMB45 (clone HMB45; cat. no. MAB-0098), Melan A (clone MX118; cat. no. MAB-1033), desmin (clone MX046; cat. no. MAB-0766), MyoD1 (cat. no. MAB-0822), myogenin (cat. no. MAB-0866), leukocyte common antigen (LCA; clone PD7/26 + 2B11; cat. no. Kit-0024), GATA-binding protein 3 (GATA3; clone EP368; cat. no. RMA-1067) and Ki-67 (clone SP6; cat. no. RMA-0542)at room temperature or 37˚C for 1-2 h, or at 4˚C overnight. All primary antibodies were purchased from Fuzhou Maixin Biotech Co., Ltd. The sections were then incubated with a ready-to-use MaxVision™ HRP-Polymer anti-Mouse/Rabbit IgG detection system (cat. no. KIT-5010; Fuzhou Maixin Biotech Co., Ltd.; enzyme conjugate: horseradish peroxidase) at room temperature for 20 min. Immunoreactivity was visualized using DAB for 3-10 min under microscopic control, followed by hematoxylin counterstaining at room temperature for 1 min, dehydration, clearing and mounting. The stained sections were examined using a light microscope. The patient did not receive further post-operative chemotherapy and attended irregular outpatient follow-up.

In December 2024, the patient reported a 1-week history of progressive left buttock pain. The patient had an Eastern Cooperative Oncology Group (ECOG) performance status of 1. A contrast-enhanced CT scan revealed a mixed-density soft tissue mass adjacent to the sacrococcygeal region, invading bone and abutting residual tissue (Fig. 2A). Pelvic magnetic resonance imaging (MRI) demonstrated heterogeneous T2-hyperintense signal with marked enhancement, suggestive of recurrent malignancy (Fig. 2B-E). The mass showed invasive osteolytic destruction of the sacrococcygeal bone with a large area of central necrosis. A bone scan ruled out distant osseous metastases.

An ultrasound-guided biopsy was performed in December 2024. Histopathological analysis revealed a high-grade spindle cell malignancy. Immunohistochemistry revealed CK(-), epithelial membrane antigen (EMA)(-), S100(-), CD34(-), human melanoma black 45 (HMB45)(-), melan-A(-), desmin(-), myogenic differentiation 1 (MyoD1)(-), myogenin(-), LCA(-), GATA3(-) and Ki67(+40%), supporting the diagnosis of undifferentiated sarcoma (Fig. 2F). Given the spatial concordance between the original radiation field and the new mass, >5-year latency, distinct histology and exclusion of recurrence/metastasis, the diagnosis of RIS was established according to the modified Cahan criteria (17). Due to anticipated treatment-associated toxicities and personal concerns, the patient declined surgical intervention or systemic therapy. At the most recent follow-up in June 2025, the patient's clinical status had significantly deteriorated. He presented with intensified sacral pain and marked weight loss, with ECOG performance status declining to 3. Follow-up contrast-enhanced CT imaging in June 2025 demonstrated interval growth and increased number of lesions, indicating progressive disease (data not shown).

Discussion

The diagnosis of RIS in the present patient was established by strictly and sequentially mapping the clinical findings to the modified Cahan criteria (17). Spatial concordance was confirmed, as the new sacrococcygeal mass developed entirely within the previous 50 Gy pelvic radiation field administered in February 2019. Second, a sufficient latency period was observed, with the new tumor emerging >5 years following the completion of radiotherapy. The histological phenotype of the secondary tumor was distinct from the primary well-differentiated rectal adenocarcinoma. Finally, local recurrence or metastasis of the primary carcinoma were excluded through comprehensive immunohistochemical analysis. The absence of epithelial markers, specifically CK and EMA, ruled out recurrent rectal adenocarcinoma. Furthermore, the negative expression of other lineage-specific markers (including S100, CD34, desmin and MyoD1) helped

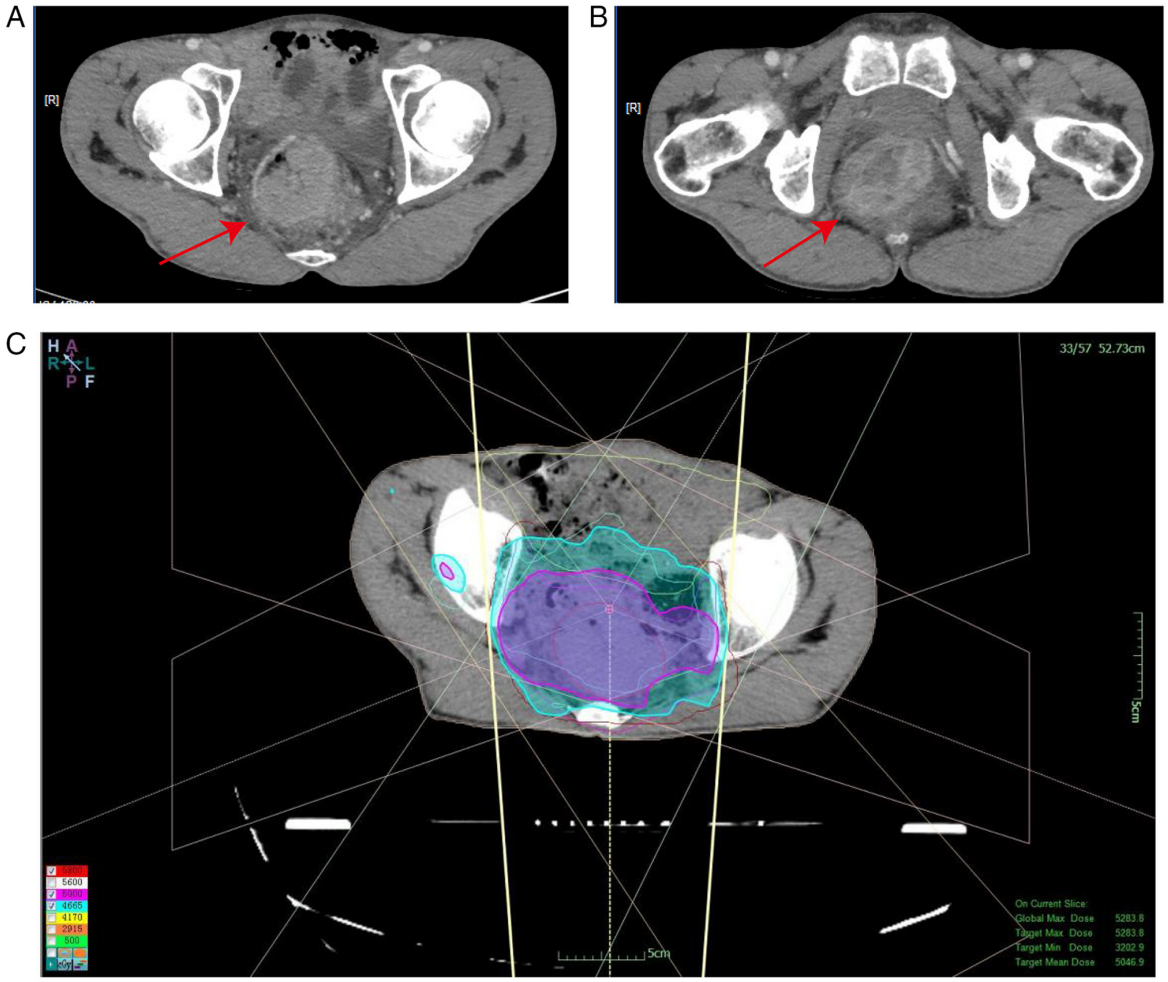

Figure 1. Initial computed tomography imaging in February 2019. Initial detection of rectal mass (red arrow) by CT scan at (A) higher and (B) lower pelvic level. (C) Target volume and dose distribution map of rectal cancer overlapped with the secondary sarcoma area. The colored lines represent various radiation isodose levels

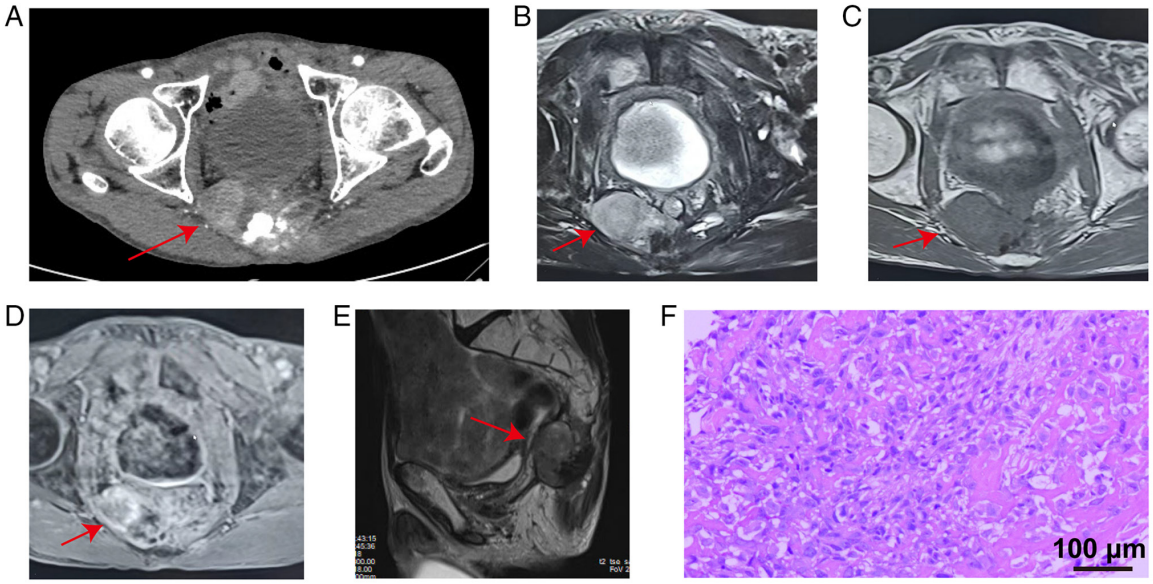

Figure 2. Imaging data for the patient diagnosis of radiation-induced sarcoma. (A) Computed tomography scan demonstrated a mixed-density soft tissue mass in the sacrococcygeal region and its surroundings, with invasion of the sacrococcygeal bones (red arrow). (B) Axial T2-weighted MRI showing an ill-defined mass-like lesion (red arrow) with heterogeneous hyperintense signals anterior to the sacrococcygeal vertebrae. (C) Axial T1-weighted MRI showing the mass (red arrow) with patchy isointense signals. (D) Contrast-enhanced axial MRI demonstrating marked heterogeneous enhancement of the lesion (red arrow) and surrounding soft tissue. (E) Sagittal MRI view revealing the mass (red arrow) and adjacent coccygeal bone destruction. (F) Pathological findings of sacrococcygeal mass puncture biopsy (hematoxylin and eosin staining; magnification, x100). Scale bar, 100 μm.

exclude melanoma, gastrointestinal stromal tumors and other differentiated soft tissue sarcoma, leading to the definitive diagnosis of RIS. While RIS has been well-documented following breast or head/neck irradiation (18-21), its occurrence following pelvic radiotherapy for rectal cancer is rare, with only isolated cases, such as a perianal leiomyosarcoma, reported in the literature (14). Pelvic secondary sarcomas in clinical practice typically manifest as angiosarcoma or leiomyosarcoma (10). Here, it presented as high-grade undifferentiated pleomorphic sarcoma. The mass exhibited invasive osteolytic destruction of the sacrococcygeal bone with a large area of central necrosis.

The non-specific clinical and radiological presentation of RIS typically mimics local recurrence, underscoring the key role of confirmatory biopsy to prevent misdiagnosis (11). This highlights the need for heightened clinical vigilance and structured long-term surveillance protocols to enable the early detection of secondary malignancy in rectal cancer survivors.

The pathogenesis of RIS involves complex interactions between radiation-induced DNA damage, genetic susceptibility and alterations in the tumor microenvironment (22,23). Molecular studies reveal that RIS, compared with primary sarcomas, frequently harbors TP53 mutations and exhibits higher tumor mutational burden and microsatellite instability, indicative of genomic instability (23,24). Notably, immune cell infiltration is often more abundant in RIS, suggesting that immune evasion serves a key role in carcinogenesis, providing a rationale for exploring immune checkpoint inhibitors (24).

To date, no standardized treatment exists for RIS. Management requires an individualized strategy based on the prior radiation history and overall health status of the patient and tumor-specific factors. As RIS is typically resistant to re-radiotherapy and chemotherapy, surgical resection is the cornerstone of curative-intent therapy (25). For patients who have previously received radiotherapy in the pelvic region, reoperation faces challenges due to severe local tissue adhesion, difficulty in ensuring adequate surgical margins and a relatively high incidence of post-operative complications (26). In the present case, as the tumor had completely invaded part of the sacrococcygeal bone, complete resection with adequate margins was technically challenging. For inoperable cases, anthracycline-based chemotherapy is the first-line systemic option, although responses are typically suboptimal and toxicity is common (27). The generally poor prognosis associated with RIS, worse than that of primary sarcoma, stems from diagnostic delay, aggressive tumor biology and intrinsic treatment resistance (27,28).

Emerging combination strategies present promising avenues. The anti-angiogenic agent, anlotinib, which remodels the tumor microenvironment, has demonstrated synergy with programmed cell death protein 1 inhibitors (29). Sintilimab + Anlotinib in patients with advanced sarcomas trial reported an objective response rate (ORR) of 30.9% and a disease control rate of 76.2% with anlotinib + sintilimab in advanced soft tissue sarcoma (30). Similarly, the histone deacetylase inhibitor chidamide combined with toripalimab achieved an ORR of 30.4% and a median progression-free survival of 7.1 months (31). Furthermore, novel B7-H3-targeted antibody-drug conjugates such as HS-20093 have shown encouraging activity in sarcoma (32). However, the aforementioned data originate from studies on primary sarcomas. Due to the unique and highly aggressive tumor biological characteristics of RIS and its genomic instability, caution is required when applying these treatment regimens. The actual efficacy and safety of these drugs in RIS have not been clinically confirmed and further clinical trials are warranted for verification.

One limitation of the present report is the lack of molecular and genetic characterization, such as TP53 mutation analysis or MYC amplification, which are frequently implicated in radiation-driven sarcomagenesis (22). Such molecular data provide direct evidence to guide personalized targeted therapy or immune checkpoint blockade. Due to the rapidly progressive disease and financial considerations of the present patient, these advanced diagnostics were not pursued. In addition, as an individual case observation, the clinical manifestations and treatment responses may not be fully representative of the broader RIS patient population. More studies are required to better define the optimal management strategies for this rare and aggressive complication.

In conclusion, RIS is a rare, severe late effect of pelvic radiotherapy. High clinical suspicion and mandatory biopsy are key to distinguish RIS from recurrence. Long-term surveillance is required. Although prognosis is poor, novel immunotherapeutic combinations require further study.

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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

YY and YL collected the clinical data and interpreted the findings. YY drafted the manuscript. YL revised the manuscript. YY and YL confirm the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

The present study was approved by the Ethics Committee of the Central Hospital of Shaoyang, Shaoyang, China; approval no. KY-2025-42). Written informed consent was acquired from the patient for participation and publication of data. The whole research process followed the Declaration of Helsinki.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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