Primary leiomyosarcoma of the lumbar soft tissue treated with multimodal therapy, including anlotinib: A case report

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Abstract. Leiomyosarcoma (LMS) originating in the deep soft tissue of the lumbar region is rare. Notably, data on the treatment response and clinical outcomes of LMS following multimodal therapy, particularly involving the multitarget tyrosine kinase inhibitor anlotinib, remain limited. The present report describes the case of a 48-year-old man with primary LMS of the lumbar deep soft tissue. Pathological evaluation demonstrated tumor infiltration of skeletal muscle, marked cellular atypia and a Ki-67 proliferation index of 70% in the recurrent mass (second resection specimen). The patient underwent three surgical resections combined with adjuvant chemoradiotherapy; following rapid disease progression after the third operation, anlotinib treatment was initiated. Treatment was temporally associated with a complete response, according to Response Evaluation Criteria in Solid Tumors 1.1 criteria, at 2 months, which was accompanied by extensive clinical tumor necrosis and sloughing. Disease progression occurred after ~6 months, consistent with the development of acquired resistance. The patient died 12 months after starting treatment with anlotinib, with an overall survival time of 36 months from the initial diagnosis. The present case illustrates the potential activity of anlotinib in recurrent LMS and highlights the challenge of acquired resistance, emphasizing the need for molecular profiling and combination strategies to achieve more durable disease control.

Introduction

Leiomyosarcoma (LMS) is a malignant soft-tissue sarcoma (STS) originating from smooth muscle cells, which accounts for 10-20% of all STS cases (1). LMS most commonly arises in the uterus, retroperitoneum and soft tissues of the extremities; however, primary LMS occurring in the deep soft tissue of the lumbar region is rare, with only a few isolated case reports in nearby or similar regions documented in the literature (2,3). Due to its rarity, the clinical course, optimal treatment strategies and long-term outcomes for this specific anatomical presentation remain poorly defined.

For localized LMS, wide surgical resection with negative margins is the cornerstone of curative treatment (4). Adjuvant radiotherapy may reduce local recurrence, particularly in high-grade tumors, while adjuvant chemotherapy remains controversial but is often considered for patients with high-risk features, such as tumor size >5 cm, deep fascial involvement and high-grade (grade 3) histology (5). In metastatic or recurrent LMS, systemic therapy options include anthracycline-based regimens, trabectedin and pazopanib (6). Recently, the multitarget tyrosine kinase inhibitor anlotinib has shown promising activity in advanced LMS, with a phase III trial demonstrating a median overall survival (OS) time of 17.45 months in heavily pretreated patients (7). However, acquired resistance inevitably develops, limiting long-term disease control.

The present report describes a case of primary LMS arising in the deep soft tissue of the lumbar region with invasion into the skeletal muscle and a Ki-67 proliferation index of 70%, indicating high proliferative activity. The patient underwent three surgical resections combined with chemoradiotherapy, followed by anlotinib therapy. This case report provides a complete clinical trajectory of a rare, aggressive LMS managed with sequential multimodal therapy, and offers insights into the efficacy and limitations of anlotinib in this challenging clinical scenario.

Case report

In February 2018, a 48-year-old man with a right lumbar mass but no notable pain, with only mild local discomfort when lying on his right side, presented to Guangshan County People's Hospital (Henan, China) and underwent a mass resection 1 week later; the operation consisted of a wide local excision of the right lumbar mass (Fig. 1). Intraoperatively, the tumor was found to be adherent to the deep fascia and

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involved the superficial layer of the erector spinae aponeurosis, but there was no gross invasion of the underlying muscle bellies. Subsequently, the patient was referred to The General Hospital of the Central Theater Command, (China) for further management. The pathological slides from the initial resection were subsequently reviewed and confirmed at Central Theater Command General Hospital of the People's Liberation Army (Wuhan, China). Postoperative pathological examination confirmed a diagnosis of right lumbar LMS. Microscopically, the tumor consisted of intersecting fascicles of spindle cells with moderate atypia, a mitotic count of 10 per 10 high-power fields (HPFs) and no necrosis; the resection margin was microscopically positive (R1) (Fig. S1A). The final diagnosis was LMS, French Federation of Cancer Centers Sarcoma Group (FNCLCC) grade 2 (8). The immunohistochemistry results were as follows: Vimentin(+), desmin(-), smooth muscle actin (SMA)(+), h-Caldesmon(+), pan-cytokeratin (PCK)(-), CD34(-), S-100(-), CD68(-), anaplastic lymphoma kinase 1(-), E3 ubiquitin-protein ligase Mdm2 (MDM2)(-), cyclin-dependent kinase 4 (CDK4)(-), β-catenin(-), Bcl-2(-), integrase interactor 1(+) and Ki-67(+; 15%) (Fig. S2). Although desmin expression was negative, the diffuse strong SMA positivity, the fascicular growth pattern and the lack of other lineage markers detected by immunohistochemistry supported this diagnosis after exclusion of other spindle cell malignancies. The patient received six cycles of intravenous chemotherapy with ifosfamide (IFO) at 2 g/m^2 per day on days 1-3 and pirarubicin (THP) at 50 mg/m^2 on day 1, repeated every 21 days. The chemotherapy was generally well tolerated; the main toxicities were grade 2 nausea, grade 2 alopecia and grade 1 fatigue, without febrile neutropenia or cardiotoxicity as per the National Cancer Institute Common Terminology Criteria for Adverse Events version 5.0 (9). Subsequently, local radiotherapy was administered to the right lumbar region using a clinical target volume (CTV) of 40 Gy in 20 fractions, followed by a CTV boost of 20 Gy in eight fractions. A total of 3 months after radiotherapy, computed tomography (CT) showed no evidence of recurrence (Fig. S1B).

Notably, at the 1-year follow-up after the first surgery, magnetic resonance imaging (MRI) revealed a new nodule in the subcutaneous soft tissue of the right waist, measuring 34 mm at the largest diameter, which was suggestive of tumor recurrence. An ultrasound-guided biopsy of the right lumbar mass was immediately performed and pathology confirmed recurrent LMS (data not shown). The recurrent tumor was found to involve the full thickness of the abdominal wall musculature, including the external oblique, internal oblique and transversus abdominis, with the deep aspect extending to the extraperitoneal fat. A fusiform incision measuring ~15x6 cm was made along the long axis of the tumor. The 12th rib and a portion of the diaphragm adherent to the tumor were resected en bloc to ensure clear margins, and the tumor together with the surrounding soft tissues was completely excised. The resulting full-thickness abdominal wall defect was repaired using a 15x15-cm synthetic mesh placed in the preperitoneal space and secured to the posterior rectus sheath. The abdominal wall incision was closed by full-thickness suturing of the skin, subcutaneous tissue and residual muscle layers (Fig. 2). The tumor exhibited a mitotic count of 22/10 HPFs, 30% necrosis and was classified as FNCLCC grade 3. The surgical margins were positive (R1). The immunohistochemistry results of the second specimen were as follows: Vimentin(+), desmin(-), SMA(+), h-Caldesmon(-), MyoD1(-), PCK(+), epithelial membrane antigen (EMA)(-), CD31(-), CD34(-), factor VIII (FVIII)(+), S-100(-), HMB45(-), CD68(-), CDK4(-), MDM2(±) and Ki-67(+; 70%) (Fig. S3 and S4). The equivocal MDM2 staining, in combination with negative CDK4 staining, indicated that dedifferentiated liposarcoma was unlikely; however, MDM2 amplification testing was not performed. Despite negative staining for h-Caldesmon and desmin, the diagnosis of LMS was favored based on diffuse SMA positivity, characteristic fascicular architecture and the exclusion of other types of sarcoma (including a malignant peripheral nerve sheath tumor, dedifferentiated liposarcoma and synovial sarcoma) by a dedicated sarcoma pathologist.

A total of 4 months after the second surgery, CT revealed small nodules on the right lumbar back, indicating a second recurrence. The third operation consisted of abdominal wall tumor resection combined with skin flap surgery. The tumor extensively involved the erector spinae, iliocostalis and longissimus muscles. A resection margin of ~3 cm from the tumor border was delineated, and the dissection proceeded layer by layer through the skin, subcutaneous tissue and affected muscles down to the extraperitoneal fat. The tumor and the involved segments of the right lumbar back muscles were completely removed. The resultant soft tissue defect was reconstructed using a skin flap based on the Limberg technique: A rhomboid flap was designed, elevated and mobilized over the right lumbar back defect, and the flap was sutured in place. Pathological evaluation again confirmed recurrent LMS involving skeletal muscle, without skin involvement (Fig. 3). The resection was R1, with a mitotic count of 25/10 HPFs and 30% necrosis, consistent with FNCLCC grade 3. The immunohistochemistry results of the specimen were as follows: Vimentin(+), desmin(+), SMA(+), h-Caldesmon(+), MyoD1(-), PCK(+), EMA(-), CD31(-), CD34(-), FVIII(-), S-100(-), HMB45(-), CD68(-), MDM2(-), CDK4(-) and Ki-67(+; 60%) (Fig. S5). Postoperatively, a second course of radiotherapy was administered to the right lumbar recurrent region; a total dose of 30 Gy was delivered in 10 fractions of 3 Gy over 2 weeks.

A total of 6 months after the third surgery, a right lumbar mass was detected that was progressively enlarging. By 8 months, the mass surface had ulcerated and exhibited bleeding. MRI confirmed tumor recurrence in the right lumbar back soft tissues (Figs. 4 and 5). Contrast-enhanced CT with three-dimensional reconstruction showed multiple irregular mass-like and nodular lesions with mild heterogeneous enhancement and ill-defined borders. The largest lesion was a cauliflower-shaped mass protruding into the retroperitoneum, measuring ~11.4x7.4x10.2 cm, with invasion of the right iliocostalis, longissimus and erector spinae muscles. New pulmonary nodules were also detected. Following a multidisciplinary sarcoma tumor board discussion, surgery, radiotherapy and chemotherapy were deemed inappropriate. The tumor was rapidly growing, making resection no longer feasible. Genetic testing was recommended but was declined by the family of the patient due to financial constraints.

The patient was subsequently administered 12 mg anlotinib orally once daily on a 2-weeks-on, 1-week-off schedule (21-day cycles). The treatment was well tolerated, with the only

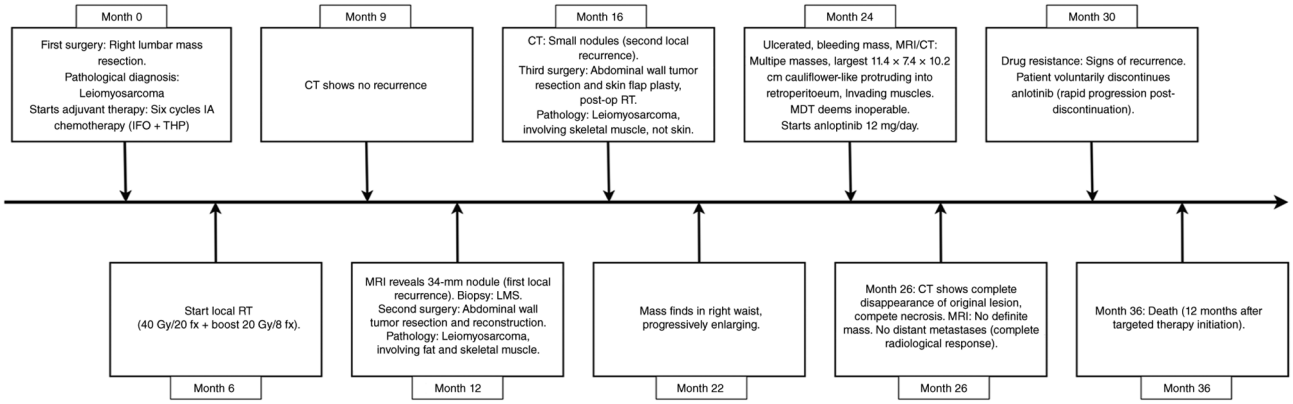

Figure 1. A detailed timeline for each event: First surgery, chemotherapy, radiotherapy, recurrences, subsequent surgeries, initiation of anlotinib treatment, key imaging assessments, progression and death. IFO, ifosfamide; THP, pirarubicin; RT, radiation therapy; CT, computed tomography; LMS, leiomyosarcoma; MRI, magnetic resonance imaging; fx, fractions; MDT, multidisciplinary team.

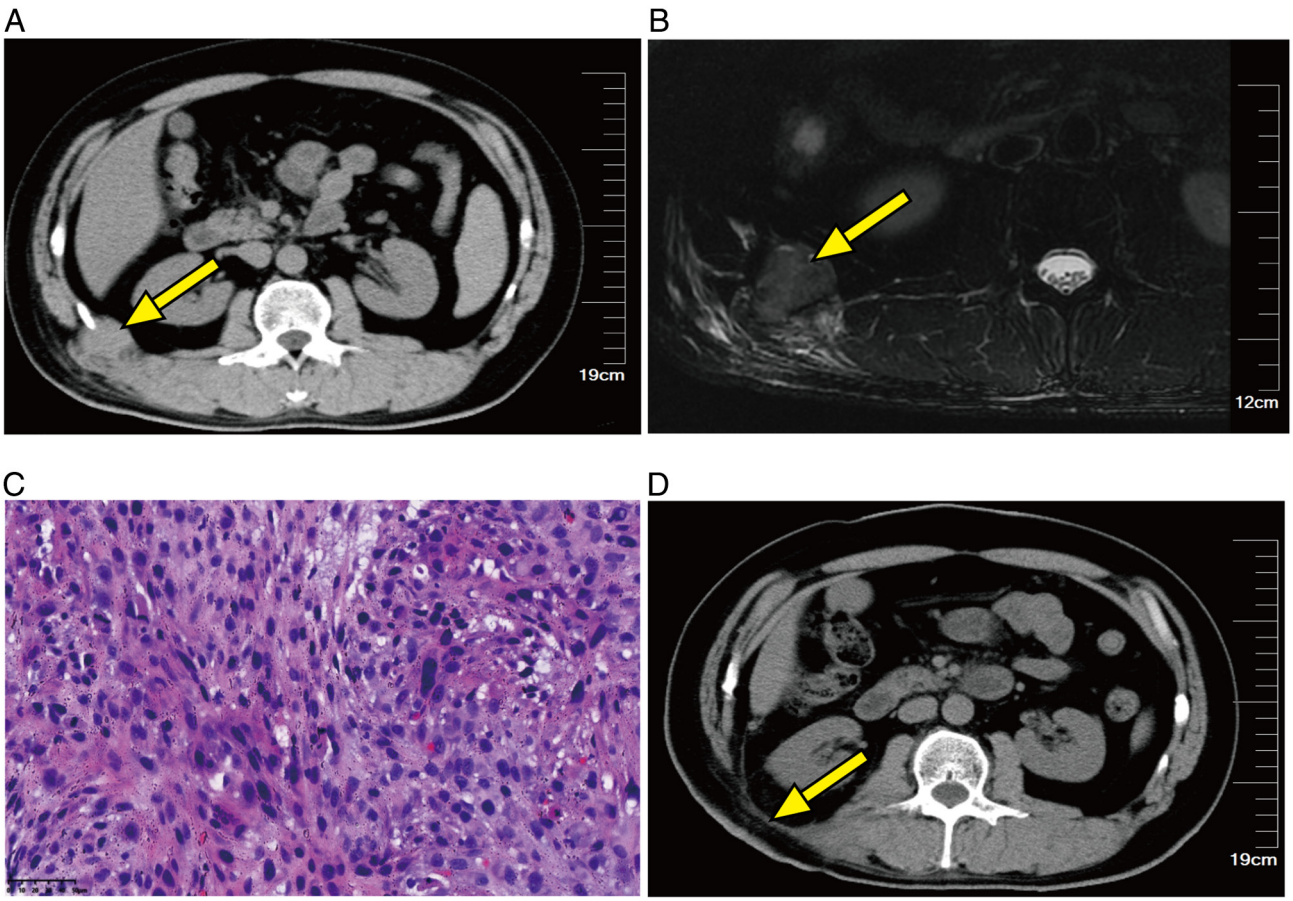

Figure 2. Abdominal wall malignant tumor resection and abdominal wall reconstruction were performed 1 year after the first surgery. (A) Preoperative CT; (B) Preoperative magnetic resonance imaging; (C) Postoperative pathology (hematoxylin and eosin staining; original magnification, x400); and (D) Postoperative CT re-examination. CT, computed tomography.

adverse events being grade 1 hand-foot syndrome and medically controlled hypertension. Tumor response to anlotinib was assessed according to the Response Evaluation Criteria in Solid Tumors (RECIST) version 1.1 (10), supplemented by serial clinical images documenting progressive tumor necrosis and sloughing (Fig. 6). At 8 weeks after anlotinib initiation, the primary tumor was no longer clinically detectable, no enlarged lymph nodes were observed in the retroperitoneum or pelvic wall, and no distant metastases were detected. Radiographic evaluation at ~2 months demonstrated complete disappearance of the primary target lesion (Fig. 7), consistent with a complete response (CR) according to the RECIST 1.1 criteria, and no new lesions were identified at that time. After 6 months of targeted therapy, the patient developed drug resistance, with signs of recurrence at the primary tumor site; thus, the patient voluntarily discontinued anlotinib. Following drug

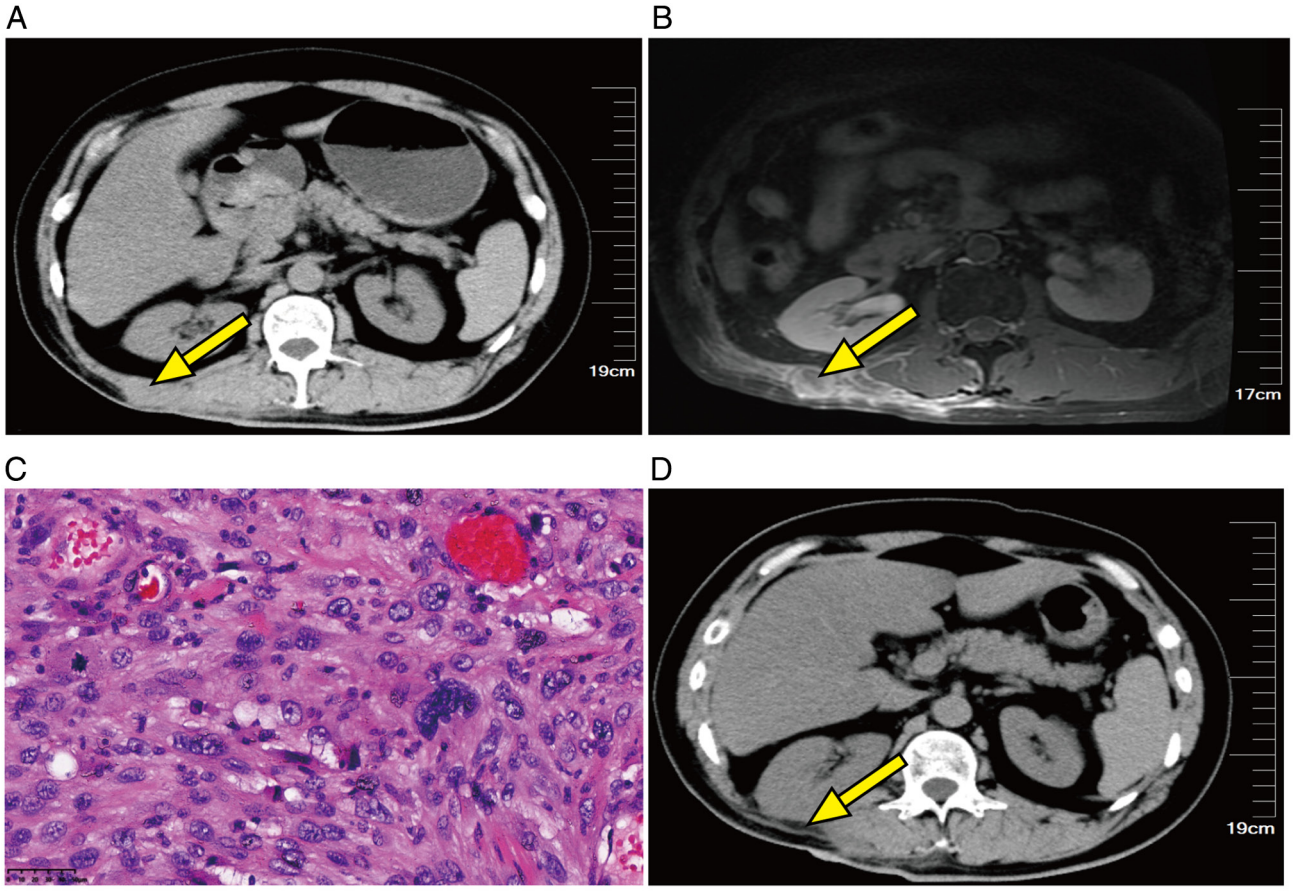

Figure 3. Tumor recurrence occurred 4 months after the second operation, and abdominal wall tumor resection with skin flap surgery was performed. (A) CT before reoperation; (B) Magnetic resonance imaging before reoperation; (C) Postoperative pathology (hematoxylin and eosin staining; original magnification, x400); and (D) Postoperative CT reexamination. CT, computed tomography.

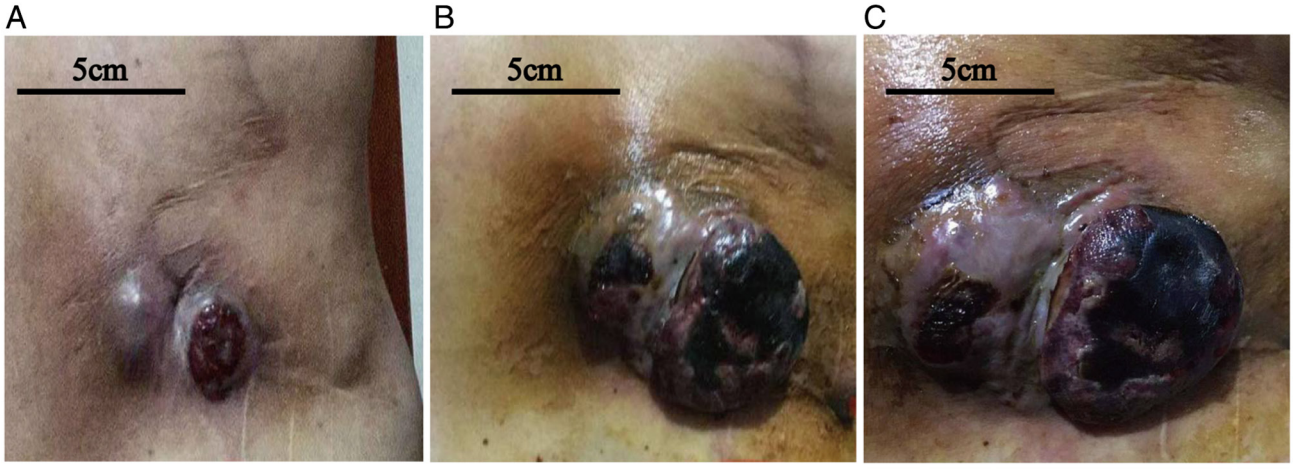

Figure 4. Clinical images of the recurrent lesion at (A) 6, (B) 7 and (C) 8 months after the third operation, showing progressive enlargement.

withdrawal, the disease progressed rapidly, with a marked increase in tumor volume (Fig. 8). The patient succumbed 12 months after initiation of the targeted therapy.

Discussion

STS encompasses a heterogeneous group of >60 histological subtypes, together accounting for <1% of all adult malignancies. LMS is a subtype defined by smooth muscle differentiation and represents 10-20% of all STS cases (1). While LMS most commonly arises in the uterus, retroperitoneum or extremities, primary occurrence in the deep soft tissue of the lumbar region is rare (6,11), with detailed descriptions of LMS confined to the lumbar paraspinal or deep lumbar soft tissue remaining scarce. A 2025 report documented the case of a 49-year-old woman with primary

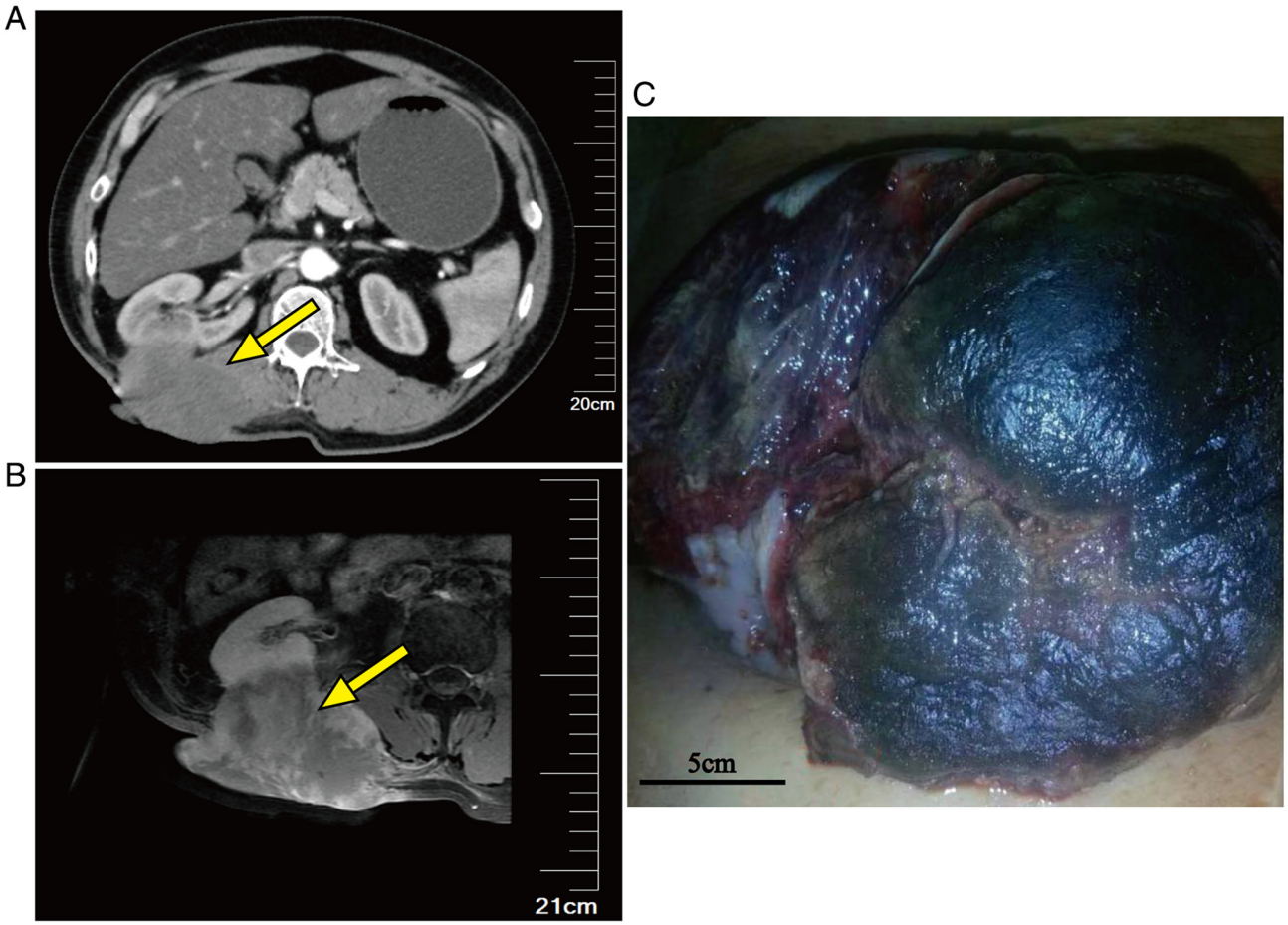

Figure 5. Imaging examination 8 months after the third operation. (A) Computed tomography examination; (B) Magnetic resonance imaging examination; and (C) Clinical image of the visible lesion.

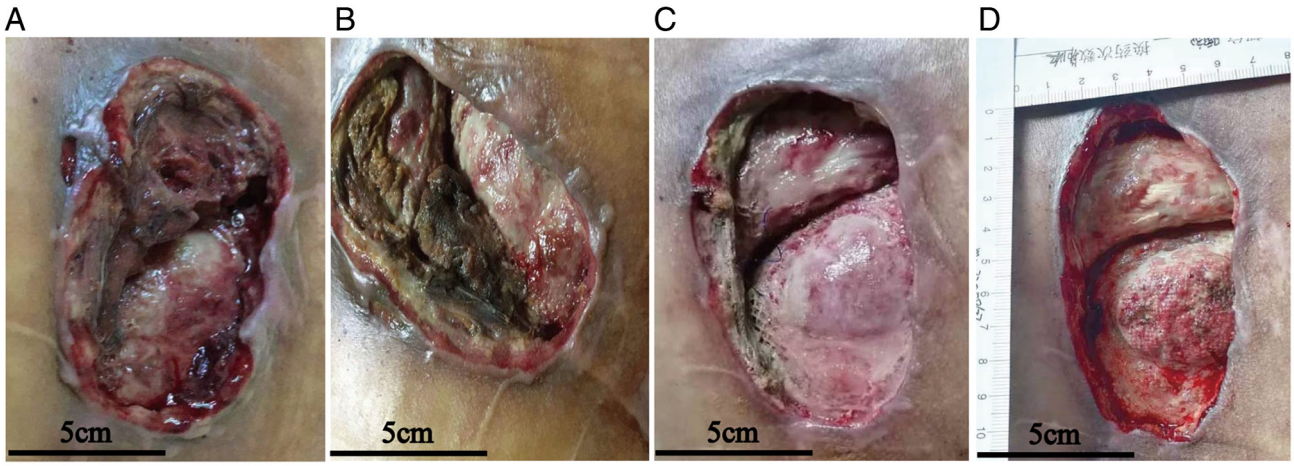

Figure 6. Sequential clinical images of the tumor at (A) 2, (B) 4, (C) 6 and (D) 8 weeks after anlotinib initiation, demonstrating progressive shrinkage, necrosis and sloughing.

paraspinal LMS who presented with lower back pain and lower extremity weakness, and was successfully managed with complete surgical excision; however, the patient was lost to follow-up after the second visit to the clinic (3). An earlier report described the case of a patient with unresectable mismatch repair-deficient LMS harboring biallelic PTEN loss, in whom the combination of antiangiogenic agents and pembrolizumab rendered the tumor resectable, and CR was achieved after surgery (2).

Unlike cutaneous or subcutaneous LMS, which generally carries a lower metastatic potential, deep-seated LMS is associated with a substantially higher risk of local recurrence (40-60%) and distant metastasis (20-60%) (12). In the current patient, the tumor invaded the skeletal muscle but spared the

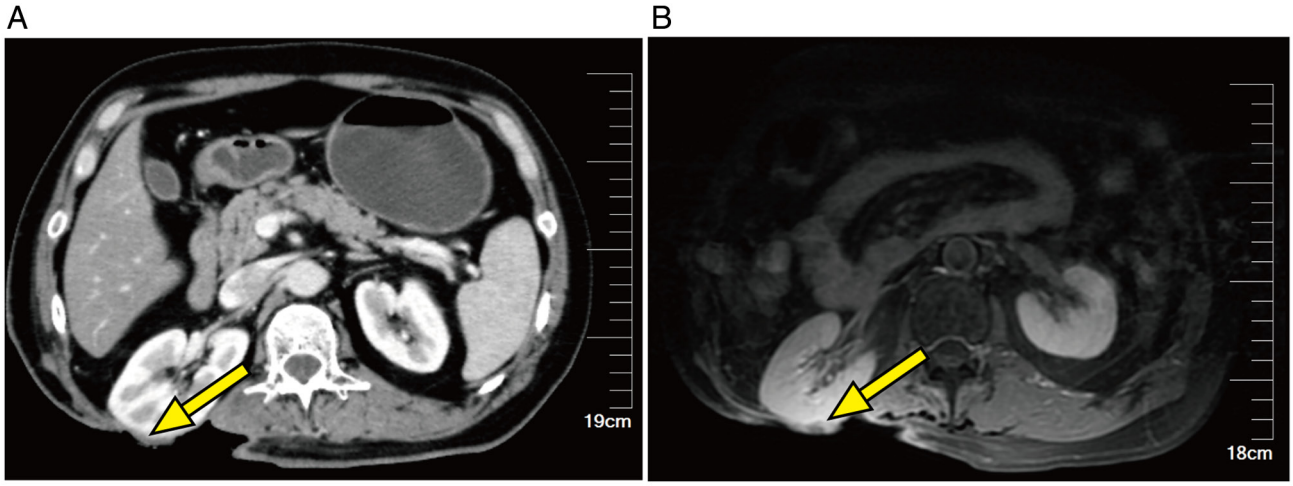

Figure 7. Imaging follow-up after anlotinib initiation. (A) Magnetic resonance imaging at 2 months after anlotinib showed no identifiable residual tumor mass at the primary site. (B) Computed tomography at 2 months after anlotinib confirmed sustained tumor regression.

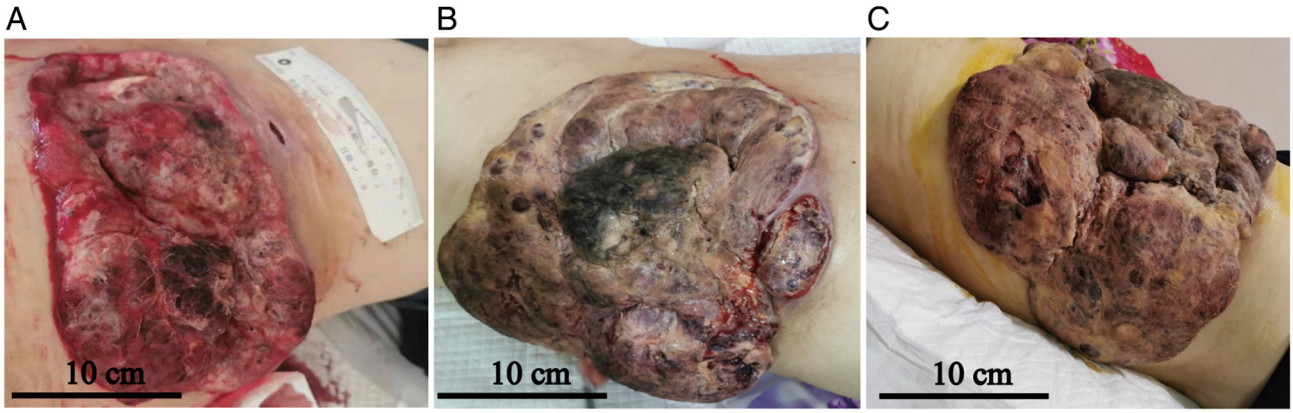

Figure 8. Clinical images showing tumor progression at (A) 6, (B) 9 and (C) 12 months after anlotinib initiation. The corresponding drug discontinuation times were 0, 3 and 6 months, respectively. Tumor progression was first detected 6 months after initiation of anlotinib.

skin and bone, consistent with a deep soft-tissue origin. This anatomical location likely contributed to the aggressive clinical course, which was characterized by multiple local recurrences despite repeated resections. Pathological evaluation showed a spindle cell neoplasm with marked cellular atypia, and immunohistochemistry revealed a Ki-67 proliferation index of 70%, which is considerably higher than the typical range of 20-40% reported for conventional LMS (13,14). Such an elevated proliferative index is associated with aggressive tumor behavior and has been related to shortened recurrence-free survival times (15). This pathological feature provides a biological explanation for the rapid recurrences observed in the current patient and may also have facilitated the emergence of resistant subclones under therapeutic pressure.

For localized STS, the cornerstone of management is wide surgical resection, frequently combined with perioperative radiotherapy and chemotherapy, an approach that has improved outcomes (16-18). The 5-year survival rate for localized disease can reach 70-80%, whereas for advanced and metastatic sarcoma, it remains at <20% (19). Over a 36-month disease course, the patient described in the present study underwent three surgical resections combined with adjuvant IFO-THP chemotherapy and radiotherapy. Despite the high proliferative index and repeated recurrences, the patient achieved an OS time of 36 months. This prolonged survival may reflect the cumulative benefit of aggressive local control through repeated surgeries and sequential systemic therapies, consistent with current guidelines that underscore the importance of multidisciplinary management for localized LMS, even in cases of recurrence (4,5).

Anlotinib is an oral multitarget tyrosine kinase inhibitor that targets VEGF receptors 1-3, platelet-derived growth factor receptor-α, c-Kit and fibroblast growth factor receptors 1-3, thereby inhibiting tumor angiogenesis and proliferation (20-22). Anlotinib is approved in China for the treatment of advanced non-small cell lung cancer, small cell lung cancer, medullary thyroid carcinoma and STS. Anlotinib has been proven effective in a variety of STSs, such as liposarcoma and synovial sarcoma (23,24). In the present patient, the early clinical course following anlotinib initiation was characterized by marked radiographic tumor regression, accompanied by clinically evident necrosis and tumoral sloughing. Follow-up imaging performed 2 months after the start of anlotinib treatment demonstrated complete disappearance of the

primary target lesion according to RECIST 1.1 criteria, with no residual tumor mass identifiable at the original site. Mild, non-specific enhancement in the right psoas muscle and adjacent subcutaneous tissue was interpreted as post-treatment change, rather than evidence of active disease. However, disease progression developed after ~6 months of therapy and the patient survived for 12 months from the initiation of anlotinib. Notably, the marked necrosis and sloughing observed at the initiation of anlotinib treatment may reflect not only drug effect but also contributions from tumor biology, ulceration, vascular compromise, infection, prior treatment effects, or a combination of these factors. Nevertheless, the temporal association between anlotinib exposure and tumor regression, followed by subsequent progression consistent with acquired resistance, suggests a potential therapeutic effect during the initial treatment period.

In the context of published data, a recent phase III trial evaluating anlotinib in advanced LMS reported a median progression-free survival time of 3.42 months (7). In the present patient, the progression-free survival interval of ~6 months appears clinically meaningful when viewed alongside this finding, although no definitive comparative conclusion can be drawn from a single uncontrolled observation. The development of progression after 6 months highlights the challenge of acquired resistance to tyrosine kinase inhibitors in sarcoma and emphasizes the need for combination strategies that may prolong durable disease control. Recent studies have investigated the addition of anlotinib to chemotherapy in the neoadjuvant conversion setting for unresectable STS (25,26). In a study of 28 patients, the combination of doxorubicin, IFO and anlotinib markedly improved tumor regression, surgical conversion rates and R0 resection rates, particularly in synovial sarcoma and liposarcoma, although with an expected increase in manageable toxicity (27). The marked initial response observed in the present case suggests that anlotinib may offer benefit in selected patients with advanced LMS and provides a clinical rationale for investigating anlotinib-based combination regimens in this specific subtype.

Several limitations must be acknowledged when interpreting the present case. First, as a single case report, the findings cannot be generalized to broader populations. Second, since post-progression tissue was not available for molecular analysis, the exact mechanisms underlying the acquired resistance to anlotinib remain undetermined; notably, this limitation constrains any discussion of resistance pathways to the level of hypothesis generation. Potential mechanisms, while speculative, may include clonal selection of pre-existing resistant subclones, activation of bypass signaling pathways such as MET or AXL, and alterations in the tumor microenvironment (28-30). In the current patient, the notably high Ki-67 index (70%) may have accelerated the expansion of resistant clones once selective pressure was applied. Future cases would benefit from paired pre- and post-progression biopsies and genomic profiling to elucidate resistance mechanisms.

In summary, the present case illustrates that a sequential multimodal treatment approach, incorporating repeated surgical resections, chemoradiotherapy and subsequent targeted therapy, was able to achieve prolonged OS time in a patient with a rare, highly aggressive primary LMS of the deep soft tissue in the lumbar region. The rapid and profound response to anlotinib, followed by the emergence of resistance, underscores the potential activity of this agent in recurrent LMS, and highlights the critical need for molecular profiling and the development of rational combination strategies to delay or overcome drug resistance.

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Availability of data and materials

The data generated in the present study are included in the figures and/or tables of this article.

Authors' contributions

JHL and TF confirm the authenticity of all the raw data. JHL and TF performed case data collection and manuscript drafting, and conceived the study. DXL, ZH and DDM were in charge of the literature search and review, acquiring the patient's pathological images and interpreting the reports. TF, ZYZ and WDJ revised the manuscript and advised on patient treatment. In addition, all authors agreed on the journal to which the article has been submitted and agree to be accountable for all aspects of the work. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

The patient described in this case report is deceased. Written informed consent for the publication of this case report and all accompanying clinical images, including identifiable images, was obtained from the patient's son, who is the legal next of kin.

Competing interests

The authors declare that they have no competing interests.

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