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Case report

Metachronous spindle cell sarcoma of the lung with spinal cord infiltration in a patient following oncological treatment: A case report

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ABSTRACT

Primary pulmonary sarcomas are exceptionally rare, and pulmonary spindle cell sarcomas represent an even rarer, highly aggressive subset. Their clinical presentation is often nonspecific, and rapid local invasion may culminate in malignant spinal cord compression (MSCC), posing urgent diagnostic and therapeutic challenges. A 60-year-old man with a history of follicular lymphoma treated with O-CHOP and obinutuzumab maintenance subsequently developed a metachronous undifferentiated spindle cell sarcoma of the right upper lobe with pleural dissemination (FNCLCC grade 2). Despite multimodal therapy including cisplatin/etoposide and thoracic radiotherapy, followed by ifosfamide-imaging in June 2025 demonstrated rapid progression. Right-sided video-assisted thoracoscopic surgery revealed a massive, unresectable tumor infiltrating the chest wall. A subsequent gemcitabine/docetaxel regimen was initiated; shortly thereafter, the patient developed acute urinary retention and rapidly progressive lower-limb sensory symptoms evolving to paraparesis. Computed tomography showed a 127 × 99 × 165 mm right upper-lobe mass with osseous invasion and intraspinal extension at Th4–Th6 and Th11–Th12, with additional liver and bilateral renal metastases. Laboratory testing demonstrated severe anemia, thrombocytopenia, and markedly elevated inflammatory markers. Management included transfusion support, high-dose corticosteroids, antibiotics for MRSE urinary infection, and opioid-based analgesia. Given disseminated disease and ECOG 4 performance status, neurosurgical decompression and further systemic therapy were withheld, and the patient was discharged to home hospice. Abrupt neurological deterioration in aggressive thoracic sarcoma mandates urgent evaluation for MSCC; in terminal disseminated disease, early goal-concordant palliative care is essential.

KEYWORDS

spindle cell sarcoma, malignant spinal cord compression, primary pulmonary sarcoma, metachronous malignancy, palliative care, follicular lymphoma

INTRODUCTION

Primary sarcomas of the lung and thoracic structures constitute an exceptionally rare group of malignant neoplasms originating from mesenchymal cells. Current estimates suggest that primary lung sarcomas account for no more than 0.5% of all pulmonary malignancies [1]. These tumors are characterized by significant challenges in pathomorphological diagnosis and a poor prognosis. The literature reports a significantly higher incidence of pulmonary metastases from extrapulmonary

foci compared to tumors of primary pulmonary origin, of which approximately 300 cases have been described to date [2,3].

Spindle cell sarcomas (SPS) within the lung and thoracic cavity represent an even rarer subgroup of primary sarcomas in this location. They are marked by exceptionally high malignancy, local invasion (including spinal cord infiltration), and a high risk of distant metastases and disease dissemination [4,5]. A critical aspect of diagnosis involves differentiating primary spindle cell sarcomas from sarcomatoid epithelial neoplasms, despite their similar spindle cell morphology [6]. Given the extreme rarity of spindle cell sarcomas primarily located in the lung and thorax, currently available literature is scarce and consists mainly of case reports, highlighting the need for further research and the systematic collection of clinical data [2,4,5].

Lung tumors, alongside prostate and breast cancers, are among the most common malignancies leading to the development of malignant spinal cord compression (MSCC), collectively accounting for 59% of all cases. The thoracic spine is the most frequent site of these lesions, responsible for over two-thirds of cases where the level of compression could be clearly determined [7]. MSCC represents one of the most severe complications of progressive neoplastic disease, occurring in approximately 2.5% of patients with advanced cancer [8]. Among the sequelae of MSCC, the risk of developing significant neurological deficits is of particular concern; these include motor, sensory, and autonomic dysfunction manifesting as paresthesias, limb numbness, paresis, and sphincter dysfunction such as urinary and fecal retention or incontinence [9]. A hallmark of MSCC is spinal pain, which often precedes severe neurological symptoms by several weeks or months [10].

The aim of this study is to present the exceptionally aggressive clinical course of a metachronous undifferentiated spindle cell sarcoma developing within the thoracic cavity of a 60-year-old patient with numerous comorbidities and a prior oncological history. This report highlights the diagnostic difficulties arising from the overlap between the clinical presentation of the primary disease and treatment complications, as well as the rarity and nonspecificity of symptoms associated with this tumor. We discuss the mechanisms of rapid local progression, with particular emphasis on massive infiltration leading to spinal cord compression and its dramatic clinical consequences, underscoring the importance of early recognition of these complications. Furthermore, we address the limitations of available therapeutic options in the disseminated stage of the disease given the unfavorable systemic prognosis, as well as the challenges in palliative care, particularly regarding the management of pain, autonomic dysfunction, and neurological deficits.

CASE REPORT

General data and medical history

A 60-year-old male with a significant oncological history was admitted to the Department of Internal Medicine on an emergency basis from the Emergency Department due to general weakness, severe anemia, and progressive neurological symptoms manifesting as lower limb paresis and sensory disturbances.

The patient's oncological history was complex. Between 2022 and 2023, the patient was treated for follicular lymphoma (8 cycles of O-CHOP, maintenance therapy with Gazyvaro). In January 2025, a second, independent proliferative process was diagnosed: undifferentiated spindle cell sarcoma within the right upper lung lobe with pleural dissemination (FNCLCC G2).

The medical history also included chronic hepatitis B (suspected past HBV infection, entecavir prophylaxis), hypertension, chronic kidney disease (CKD G2), and a status post bilateral inguinal hernia repair.

Physical examination at admission

On physical examination (performed on August 4, 2025), the patient was in fair general condition, conscious, and fully responsive. Pallor of the skin and mucous membranes was noted. Vital signs were stable, with a regular heart rate of approximately 80 bpm. Neurological examination revealed signs of spinal cord damage presenting as bilateral lower limb paresis (paraparesis, grade 2 on the Lovett scale), sensory deficits below the Th7 level, absent ankle jerks with diminished knee reflexes, and a bilateral positive Babinski sign.

History of present illness

The clinical course of the sarcoma was characterized by rapid progression and resistance to multi-line treatment. Key events in the disease history are as follows. On July 28, 2022, follicular lymphoma was diagnosed, initiating a multi-stage oncological process. In 2024, preliminary treatment was implemented, comprising four cycles of cisplatin and etoposide chemotherapy, completed in October, and radiotherapy with a dose of 45 Gy in 30 fractions, administered in September. Another pivotal moment occurred on February 12, 2025, when histopathological examination confirmed a second, independent neoplastic process: grade 2 spindle cell sarcoma. In response to the new diagnosis, two cycles of ifosfamide were administered between February and April 2025. Despite these measures, imaging studies in June 2025 confirmed rapid disease dissemination. On June 13, 2025, a right-sided video-assisted thoracoscopic surgery (VATS) was performed at a center in Warsaw; however, the intraoperative finding of a massive tumor infiltrating the chest wall forced the abandonment of resection.

In July 2025, following confirmed progression, a decision was made to implement a subsequent line of chemotherapy using a gemcitabine and docetaxel regimen, preceded by vascular port placement on July 22. By July 28, after gemcitabine administration, further tumor progression was noted, and two days later an episode of urinary retention occurred; the patient reported to the urological emergency room, where he was catheterized due to a neurogenic bladder. The culmination of symptoms occurred at the turn of July and August 2025, when, over just a few days prior to hospitalization, paresthesias of the lower limbs appeared and rapidly intensified, quickly progressing to mobility difficulties and paresis, indicating progressive compression of the spinal canal structures.

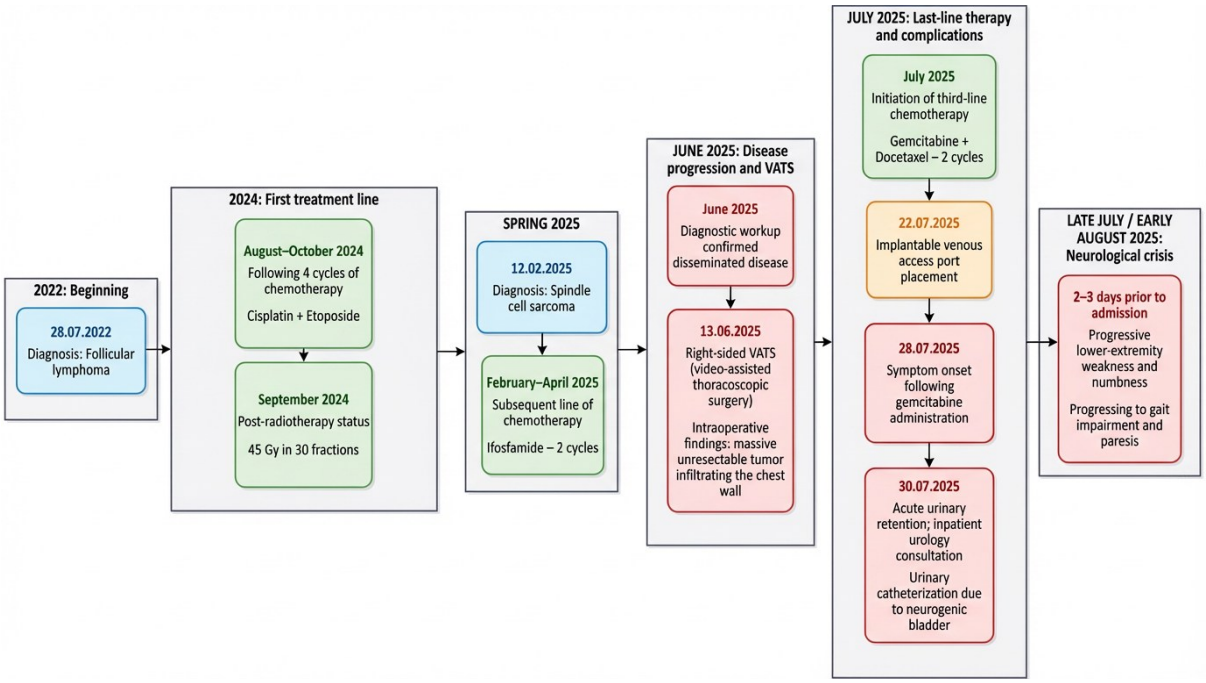

Fig. 1. The patient's clinical course

Diagnostic work-up

Diagnostic imaging, including computed tomography (CT) of the chest and spine, was performed to investigate the underlying cause of the neurological deterioration. The imaging revealed tumor progression in the right upper lung lobe (measuring 127 × 99 × 165 mm), with infiltration of osseous structures and intraspinal extension of tumor masses at the Th4–Th6 (Figure 2) and Th11–Th12 levels (Figure 3). Additionally, metastatic lesions were identified in the liver and both kidneys.

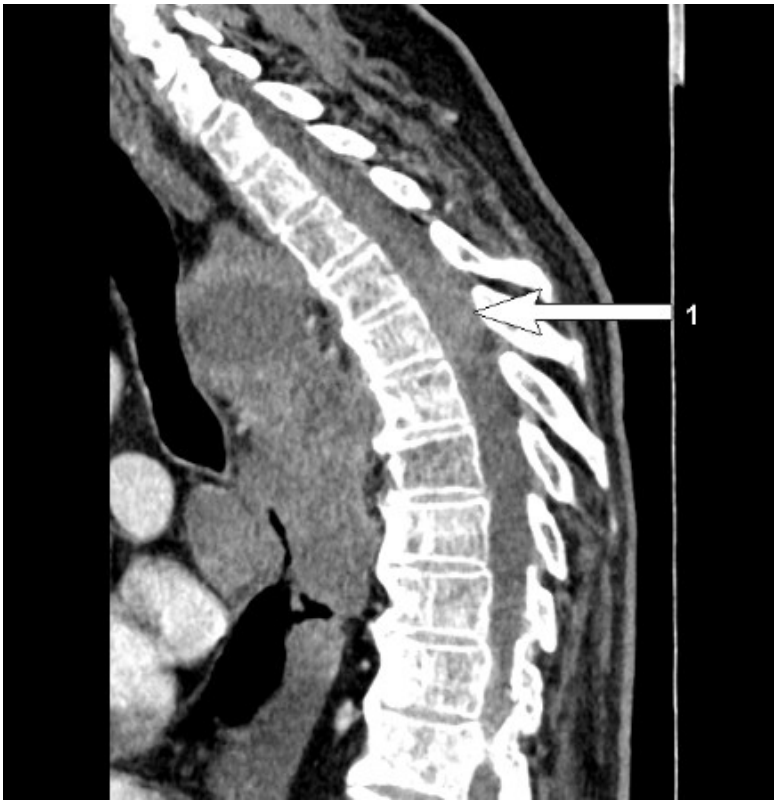

Fig. 2. The infiltration of osseous structures and intraspinal extension of tumor masses at the Th4–Th6 levels (1)

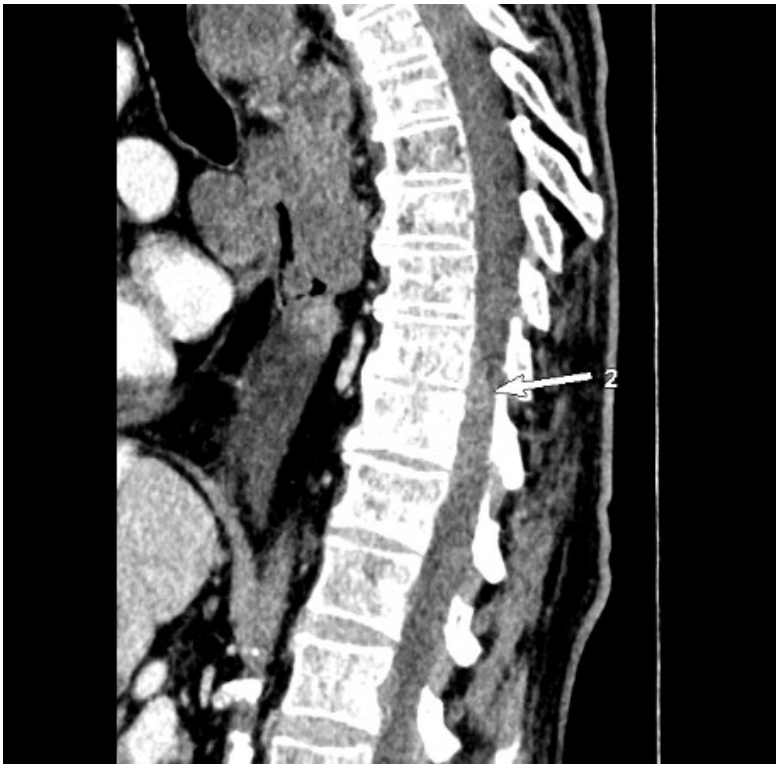

Fig. 3. The infiltration of osseous structures and intraspinal extension of tumor masses at the Th11–Th12 levels (2)

Laboratory findings: Admission laboratory tests were notable for severe anemia (hemoglobin 7.8 g/dl and thrombocytopenia (platelet count 116 × 10^3/µl). Inflammatory markers were significantly elevated (CRP 188 mg/l, PCT 0.51 ng/ml; Table I).

Table I. Selected laboratory findings during the patient's hospitalization

Parameter	Value at admission (02.08.25)	Follow-up value (date)	Laboratory reference range
Hgb	7.80 g/dl	9.80 g/dl (04.08 post transfusion)	13.7–17.5
PLT	$116 \times 10^3/\mu l$	$55 \times 10^3/\mu l$ (05.08)	150–400
CRP	188 mg/l	167 mg/l	< 5.0
PCT	0.51 ng/ml	0.46 ng/ml (04.08)	< 0.5
Crt	1.13 mg/dl	0.94 mg/dl (06.08)	0.67–1.17
Alb	24 g/l	–	35–52
GGT	553 U/I	973 U/I (04.08)	<60
ALP	590 U/I	587 U/I (04.08)	40–129
ALT	117 U/I	264 U/I (08.08)	10–50

Hgb – hemoglobin; PLT – platelets; CRP – C-reactive protein; PCT – procalcitonin; Crt – creatinine; Alb – albumin; GGT – gamma-glutamyl transferase; ALP – alkaline phosphatase; ALT – alanine aminotransferase.

Therapeutic interventions and hospital course

Given the patient's poor general condition upon admission, immediate measures were taken to stabilize vital signs. Priority was given to correcting hematological abnormalities. In response to severe, symptomatic anemia (baseline hemoglobin 7.7 g/dl) and concurrent thrombocytopenia, aggressive transfusion therapy was initiated, consisting of 3 units of packed red blood cells (PRBCs) and 1 unit of fresh frozen plasma (FFP). This resulted in the desired improvement in red blood cell indices, with hemoglobin levels rising to 9.9 g/dl. Concurrently, dexamethasone therapy was introduced to reduce progressive spinal cord edema and alleviate neurological symptoms of

compression. Furthermore, due to elevated inflammatory markers (PCT, CRP) and the isolation of methicillin-resistant Staphylococcus epidermidis (MRSE) in urine cultures, while blood cultures remained sterile, empirical antibiotic therapy with ceftriaxone was administered. Additionally, analgesic treatment (oxycodone/naloxone) was implemented in accordance with the WHO analgesic ladder, effectively reducing pain symptoms.

Specialist consultations and final decisions

Following a multidisciplinary case review, a decision was made to withhold further causal treatment and surgical interventions, deeming the patient's condition terminal in light of the rapid progression of the spindle cell sarcoma. Despite radiological evidence of multilevel spinal cord compression (Th4–Th6 and Th11–Th12), neurosurgical consultation ruled out decompressive spinal surgery. This decision was justified by the presence of generalized metastatic spread to parenchymal organs and the chest wall, as well as the patient's extremely poor performance status (ECOG 4), which entailed high perioperative risk with no guarantee of motor function recovery.

Simultaneously, during the oncological consultation on August 5, 2025, the initiation of a third line of systemic treatment was considered futile. Disqualification was based on the lack of clinical response to prior ifosfamide- and gemcitabine/docetaxel-based regimens, as well as the development of critical organ complications, including bone marrow failure (CTCAE grade 3/4 anemia and thrombocytopenia), hypoalbuminemia, and progressive cachexia. Consequently, therapeutic goals were redefined to prioritize quality of life optimization through intensive symptomatic management, including pain control protocols and anti-edema corticosteroid therapy. Ultimately, respecting the patient's wishes and aiming to avoid futile medical intervention, the patient was discharged in a hemodynamically stable but fully dependent state to the care of a Home Hospice.

DISCUSSION

The diagnosis and management of lung sarcomas constitute one of the greatest challenges in contemporary thoracic oncology, primarily due to the significant rarity of these neoplasms and their nonspecific clinical presentation. As indicated by Collini et al. [4], primary lung sarcomas account for merely 0.001% to 0.28% of all malignancies of this organ, making them a classic "diagnosis of exclusion." In patients with a significant oncological history, such as the discussed case of a patient with a history of small cell lymphoma, this process becomes particularly insidious. Symptoms such as cough, dyspnea, or chest pain may be misinterpreted as a recurrence of the primary disease or infectious complications, delaying the diagnostic process and the establishment of the correct histopathological diagnosis [4,11].

Precise definition of the tumor type based on cellular morphology is insufficient in these cases and requires the application of advanced molecular techniques. The discussed case should be interpreted in light of the updated 2020 WHO classification of soft tissue tumors. Sbaraglia et al. [12] emphasize that the category of "undifferentiated sarcomas" has been significantly narrowed due to the identification of new, genetically defined subtypes, such as spindle cell neoplasms with NTRK gene rearrangement. These tumors, morphologically mimicking classic fibrosarcomas, constitute a significant diagnostic pitfall, and their detection opens the path to highly effective targeted therapies. The fact that our patient was ultimately diagnosed with "Undifferentiated Spindle Cell Sarcoma" indicates that this tumor proved refractory even to the latest methods of molecular stratification and exhibited no actionable therapeutic targets, confirming its classification within a group of neoplasms with the most aggressive course and one of the poorest prognoses.

It must be emphasized that the microscopic appearance of spindle cell tumors within the thoracic cavity can be extremely misleading. In his analysis of the differential diagnosis of mediastinal and lung tumors, Suster [13] points to a broad spectrum of entities that must be excluded before making a final diagnosis of primary sarcoma. This requires the pathologist to systematically rule out spindle cell variants of epithelial neoplasms (e.g., sarcomatoid carcinomas, thymomas), as well as vascular tumors such as angiosarcoma or epithelioid hemangioendothelioma, which may exhibit deceptive histological mimicry. In the presented case, the lack of expression of markers specific to epithelial, vascular, or lymphoid lineages confirmed the mesenchymal nature of the neoplasm, while simultaneously indicating its low degree of differentiation, which drastically limited available therapeutic options.

The patient’s remote oncological history warrants particular attention in the analysis of this case. The occurrence of soft tissue sarcomas (STS) as secondary malignancies in individuals cured of non-Hodgkin lymphomas (NHL) is a rare but documented phenomenon, falling into the category of therapy-related neoplasms. As indicated by epidemiological analyses, the cumulative risk of developing a secondary sarcoma in patients following lymphoma treatment is significantly higher than in the general population, and the latency period may range from several years to as many as 20 years [14]. A key role in the pathomechanism of this phenomenon is played by the late effects of cytostatic treatment, particularly alkylating agents (e.g., cyclophosphamide), which are commonly used in CHOP-like regimens. These drugs, by inducing DNA double-strand breaks and forming interstrand cross-links, can lead to genomic instability in mesenchymal stromal cells [15]. Although a full molecular profile of the past tumors is not available in this case, the sequence of events suggests that the current spindle cell sarcoma may represent a somatic manifestation of

remote genetic toxicity from prior therapy, further complicating the clinical picture through the potential drug resistance of secondary clones [16]. While the sequence of clinical events initially suggests the possibility of a therapy-related neoplasm, it is critical to acknowledge that the latency period between the administration of the O-CHOP regimen with obinutuzumab (2022–2023) and the diagnosis of the spindle cell sarcoma (January 2025) is exceptionally short. Therapy-related soft tissue sarcomas typically present with a latency of 5 to 20 years following exposure to alkylating agents [17].

Therefore, while a secondary malignancy cannot be definitively excluded, a multifactorial etiology and shared risk factors are highly probable in this clinical scenario. Both follicular lymphoma and primary pulmonary sarcomas share several broad predisposing factors, primarily advanced age and age-related immunosenescence. Furthermore, the profound and prolonged state of immunosuppression-induced inherently by the indolent lymphoma itself and significantly exacerbated by B-cell depleting maintenance therapy (obinutuzumab) and cytotoxic agents may have facilitated the rapid growth and immune evasion of a pre-existing, subclinical sarcoma clone [18].

Additionally, unidentified underlying genetic predispositions, such as somatic or germline mutations affecting tumor suppressor pathways (e.g., *TP53* or *RB1* mutations), combined with potential unrecorded environmental or occupational exposures, could have created a favorable biological landscape for the rapid, metachronous development of both independent malignancies [19]. Consequently, the coexistence of these two rare entities is most likely the result of a complex interplay between an altered immune microenvironment, systemic vulnerability, and intrinsic genetic susceptibility, rather than being solely a direct consequence of the recent systemic therapy.

The onset of symptoms of metastatic spinal cord compression (MSCC) constitutes one of the most dramatic moments in the course of neoplastic disease, requiring immediate decisions based on the "Time is Spine" paradigm. As indicated by Singer et al. [20] in the latest review of guidelines from 2025, the patient's functional prognosis is strictly correlated with their neurological status at the time treatment is initiated. Back pain, often referred to as the "sentinel symptom," may precede motor deficits by weeks; however, from the moment paresis appears until total paraplegia occurs, the therapeutic window closes rapidly. In the discussed case, the dynamics of the process were fulminant, the patient reported a deterioration in mobility just 2–3 days prior to hospitalization, arriving at the ward with established paralysis and a neurogenic bladder. As recommended by Singer et al. [20], in patients with such advanced deficits who have lost the ability to walk (non-ambulatory), the chances of reversing the changes are minimal, and the priority

becomes the pharmacological reduction of vasogenic edema through the immediate administration of high-dose corticosteroids, as was done in the presented case.

A key ethical and clinical challenge in such a situation is delineating the fine line between providing aid and exposing the patient to the iatrogenic risk associated with "futile therapy." Lawton et al. [21], in their multidisciplinary analysis, emphasize that although surgical spinal decompression combined with radiotherapy offers statistically better results than radiotherapy alone, this benefit applies exclusively to a narrow group of patients with a predicted survival exceeding 3 months. The decision to withhold surgery in our patient finds strong justification within the NOMS (Neurologic, Oncologic, Mechanical, Systemic) decision-making framework, where the systemic factor, understood as general disease advancement, plays a paramount role. The presence of dissemination to parenchymal organs (liver, kidneys) and the patient's poor general condition constituted, in light of the data reported by Lawton et al. [21] , an absolute contraindication to major neurosurgical intervention, which would have exposed the patient to perioperative suffering without a real chance for quality of life improvement.

The validity of this therapeutic prudence is confirmed by the studies of Lo and Yang [22], who demonstrated a strict correlation between scores in prognostic scales (such as the Tomita or Tokuhashi scales) and overall survival. Patients with an aggressive primary tumor type and visceral metastases, as in the discussed case, achieve scores qualifying them for the worst prognosis group (median survival approx. 4 months), in which palliative decompression yields no benefit. Furthermore, these authors prove that the failure to regain walking function after surgery is an independent predictor of rapid death, rendering the operation of patients with established paraplegia medically unjustified.

Reference must also be made to the decision to withhold palliative radiotherapy to the spine. Rades et al. [23] developed a predictive tool for patients with MSCC near the end of life, identifying factors indicating a lack of benefit from irradiation. Negative predictors include, among others, the presence of visceral metastases, a rapid onset of motor deficits (under 14 days), and non-ambulatory status prior to the initiation of therapy. Our patient met all these criteria for a poor prognosis. In such a clinical constellation, as indicated by Rades et al. [23] research, the implementation of radiotherapy does not improve motor function nor extend survival, instead generating only additional stress associated with transport and hospitalization. Thus, referring the patient to hospice care and implementing exclusively symptomatic management was the optimal decision, protecting the patient from futile medical procedures in the terminal phase of the disease.

One of the most challenging aspects of palliative care in the presented case was the management of severe gastrointestinal dysfunction resulting from a "double hit" mechanism: the superimposition of neurogenic and pharmacological factors. According to the latest classification presented by Magnuson et al. [24] and Hakim et al. [25], spinal cord injury at the thoracic level (supraconal) results in the development of an upper motor neuron (UMN) neurogenic bowel. This condition is characterized by anal sphincter hypertonia and spastic stool retention with preserved but uncoordinated spinal reflexes. The clinical picture was dramatically complicated by the necessity of using high doses of opioids, which caused Opioid-Induced Bowel Dysfunction (OIBD) to overlay the existing neurogenic sphincter dyssynergia. To break this mechanism, a modern pharmacological strategy based on peripherally acting mu-opioid receptor antagonists (PAMORA) was applied. In accordance with Blair's [26] recommendations, the introduction of naldemedine (Rizmoic) and a fixed-dose combination of oxycodone/naloxone (Targin) aimed to restore propulsive peristalsis by blocking μ-receptors in the gut without attenuating the central analgesic effect. The validity of this approach is confirmed by the latest results of a randomized multicenter study published by Hamano et al. [27]. These authors demonstrated that naldemedine significantly increases the frequency of spontaneous bowel movements in cancer patients without negatively affecting analgesic efficacy.

Despite the implementation of this aggressive and targeted pharmacotherapy and the standard conservative protocol according to Cotterill's et al. [28] guidelines (osmotic laxatives, enemas), the patient required manual fecal evacuation. This observation confirms Emmanuel's [29] thesis that in nearly half of patients with neurogenic bowel dysfunction, conservative treatment is ineffective. In the case of complete nerve pathway damage, the spastic component may dominate over the metabolic one, rendering even advanced PAMORA-class pharmacotherapy insufficient without mechanical support. However, this procedure finds full justification in the updated NBD treatment algorithm published by Magnuson et al. [24]. The authors indicate that in the case of spastic bowel (UMN), anal electrostimulation and manual extraction are not evidence of therapeutic failure, but constitute an integral element of the standard of care, necessary to overcome the resistance of the spastic sphincter.

Effective pain control in a patient with malignant spinal cord compression (MSCC) required an aggressive pharmacological approach, accounting for a complex, mixed pain etiology. As Ye et al. [30] point out in a neurobiological analysis, damage to spinal cord tissue induces a cascade of glial reactions, activation of microglia and astrocytes – leading to central sensitization and the entrenchment of neuropathic pain refractory to standard non-opioid analgesics. This necessitated the use of high doses of potent opioids (oxycodone), which, however, was associated with a direct

risk of severe gastrointestinal complications. Liu and Brenner [31] specify that in patients with existing neurological dysfunction, the introduction of opioids leads to the phenomenon of "Opioid-Exacerbated Constipation" (OEC), where pharmacological paralysis of peristalsis superimposes upon existing pelvic floor pathology, creating a vicious cycle mechanism.

To resolve this clinical dilemma and allow for the escalation of analgesic doses, a modern targeted therapy with a peripherally acting mu-opioid receptor antagonist (PAMORA) – naldemedine (Rizmoic). Naldemedine, a high molecular weight derivative of naltrexone, blocks opioid binding to μ-receptors in the enteric plexus without crossing the blood–brain barrier, thereby restoring transit without antagonizing central analgesia, a safety profile confirmed in a multicenter RCT by Hamano et al. [27], which unequivocally demonstrated that naldemedine improves the quality of life of cancer patients by reducing constipation, while maintaining stable pain control and causing no withdrawal symptoms. The use of combined pain management (Targin) together with targeted constipation prophylaxis (Rizmoic) in our patient thus reflected the highest standards of palliative care, aiming to interrupt the OEC pathomechanism while simultaneously ensuring analgesic comfort.

The final stage of the patient's treatment illustrates one of the most difficult dilemmas in modern palliative oncology: risk management amidst the collapse of organismal homeostasis. The onset of severe thrombocytopenia (CTCAE grade 3/4) resulting from chemotherapy-induced myelotoxicity necessitated the decision to discontinue low-molecular-weight heparin (LMWH), despite the high risk of venous thromboembolism (VTE). This decision represented an acceptance of the "lesser of two evils" paradigm. In the face of a massive, vascularized tumor infiltrating spinal cord structures, the risk of catastrophic intratumoral hemorrhage or gastrointestinal bleeding was deemed an immediate threat to life and a potential source of suffering that outweighed the risk of embolic death. This aligns with the guidelines of the ISTH (International Society on Thrombosis and Haemostasis), which recommend withholding anticoagulation in patients with a platelet count <25–50 G/L, even in the presence of active malignancy [32]. Discontinuing antithrombotic prophylaxis in the terminal phase of the disease constitutes an element of the transition from preventive medicine to comfort care. As noted by palliative care experts, in patients in an agonal state, a so-called "silent" pulmonary embolism may paradoxically be a mechanism of death that is less burdensome than massive hemorrhage, which ethically justifies the decision not to escalate treatment in the setting of thrombocytopenia [32].

Given the exhaustion of causal treatment options due to disease progression despite second-line chemotherapy, and disqualification from radiotherapy and surgical decompressive intervention, the key aspect during this period became not the extension of life, but the maximization of its

quality in the face of rapidly increasing neurological disability. The implementation of invasive treatment in a state of such advanced neoplastic wasting and multi-organ failure would have borne the hallmarks of futile therapy. According to the NOMS decision-making model, the systemic factor (Systemic illness) in a patient with visceral dissemination represents an absolute contraindication to spinal surgery, as the median survival in this group of patients rarely exceeds 3 months [21]. The decision to discontinue disease-directed therapy in favor of hospice care and exclusively symptomatic management is a critical aspect of this case. In modern oncology, the transition from active anticancer treatment to best supportive care represents a fundamental component of high-quality, patient-centered care rather than therapeutic failure. This decision is typically guided by the absence of meaningful clinical benefit from further systemic therapy, rapid disease progression despite multiple treatment lines, declining performance status (ECOG ≥3), and the increasing burden of treatment-related toxicity. Evidence indicates that continuation of anticancer therapy near the end of life is frequently associated with worsened quality of life, increased hospitalizations, and no survival advantage [33,34], while early integration of palliative care improves symptom control, patient satisfaction, and even survival in some populations [33,35]. Moreover, avoiding non-beneficial interventions aligns with the principles of proportionality and non-maleficence, reducing the risk of so-called “diagnostic and therapeutic aggressiveness” at the end of life [36]. International guidelines, including those from ASCO and ESMO, emphasize that discontinuation of anticancer therapy should be considered when there is no reasonable expectation of tumor response or clinical stabilization, particularly in patients with poor functional status and widespread metastatic disease [36,37]. In the present case, the combination of rapid tumor progression, lack of response to multiple chemotherapy regimens, severe organ dysfunction, and ECOG 4 status fulfilled widely accepted criteria for medical futility [38,39]. Referring the patient to hospice care allowed for the implementation of optimized symptomatic pharmacotherapy, including a combination of opioids and corticosteroids, which permitted effective control of pain and dyspnea, constituting the "gold standard" of palliative management in inoperable spinal cord compression syndrome [30]. Transitioning to hospice care therefore represented an ethically and clinically appropriate decision, prioritizing quality of life, dignity, and symptom relief over non-beneficial life-prolonging interventions.

CONCLUSIONS

The presented case of metachronous spindle cell sarcoma in a patient following lymphoma treatment illustrates the complexity of modern oncology, where therapeutic success in one disease may be associated with the risk of late complications, including secondary therapy-related neoplasms. The clinical picture, dominated by nonspecific symptoms and rapid progression to

malignant spinal cord compression (MSCC), underscores the necessity of maintaining high oncological vigilance in patients with a complex medical history, in whom new symptoms may be misinterpreted as a recurrence of the primary disease or degenerative changes.

An analysis of the management in the face of advanced neurological deficit confirms the utility of the NOMS decision-making framework in qualifying patients for neurosurgical treatment. As demonstrated, in a patient with a disseminated neoplastic process and poor performance status, withholding risky surgical decompression in favor of conservative treatment is a decision that is both medically and ethically justified, protecting the patient from futile therapy.

The described case also highlights the challenges of modern palliative care, particularly regarding the management of iatrogenic complications. The use of peripherally acting mu-opioid receptor antagonists (PAMORA) allowed for the effective disruption of the "double hit" mechanism within the gastrointestinal tract (combined neurogenic and opioid-induced bowel), enabling simultaneous aggressive pain control and improvement in quality of life. Ultimately, the patient's clinical history serves as evidence that in the terminal phase of neoplastic disease, when causal treatment options have been exhausted, the paramount goal becomes quaternary prevention. Thanks to hospice care, the patient can spend their final moments in peace, avoiding the suffering associated with excessive hospital interventions.

Authors' contribution

Study design – A. Stanek, M. Zych

Data collection – J. Mikołajczyk, A. Joniec, E. Mażul-Kulesza, J. Gonscz, T. Fajferek

Manuscript preparation – J. Mikołajczyk, A. Joniec, T. Fajferek

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Final approval of the version to be published – J. Mikołajczyk, A. Joniec, E. Mażul-Kulesza, J. Gonscz, T. Fajferek, M. Zych, A. Stanek

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