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Rhabdomyolysis-Associated Gluteal Compartment Syndrome Complicated by Sciatic Neuropathy: A Case Report of Diagnostic and Therapeutic Challenges

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Case Report

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Abstract

Background: Rhabdomyolysis (RM) is a life-threatening emergency resulting from skeletal muscle injury. Gluteal compartment syndrome (GCS) is an uncommon clinical entity, and sciatic neuropathy secondary to RM-associated GCS is even rarer. Because of its nonspecific presentation, this condition is frequently misdiagnosed or diagnosed belatedly. This case report aims to highlight the diagnostic pitfalls and therapeutic decision-making in RM-related GCS complicated by sciatic neuropathy.

Case Presentation: A 38-year-old man presented to the emergency department with a 2-day history of low back pain and gross hematuria. He had slept on the floor and awoke with right-sided lumbar and abdominal distending pain, cola-colored urine, and left lower extremity weakness. Laboratory tests demonstrated markedly elevated creatine kinase (CK; 29,696 U/L), CK-MB (186.1 U/L), and C-reactive protein (93.9 mg/L). He was admitted with a provisional diagnosis of RM. Motor strength of the left lower extremity was graded 0 on the Medical Research Council (MRC) scale. Ultrasound and MRI of the left gluteal region revealed subcutaneous edema and fluid collection, and electromyography (EMG) suggested injury to the left tibial and common peroneal nerves. GCS was diagnosed clinically (intracompartmental pressure was not measured); although surgical decompression was recommended, the patient declined and requested discharge after conservative management. At discharge, CK levels had normalized, but foot drop persisted. At 3 and 6 months after symptom onset, proximal muscle strength had recovered to grade 5, whereas distal strength remained grade 0–1; repeat EMG demonstrated complete injury to the left tibial, common peroneal, sural, and superficial peroneal nerves.

Conclusion: We report a rare case of nontraumatic rhabdomyolysis complicated by GCS and secondary sciatic neuropathy, possibly related to sedative-associated prolonged immobilization. Delayed diagnosis and refusal of surgical decompression likely contributed to persistent foot drop. Diagnosis relies on a high index of suspicion, especially in high-risk populations with prolonged immobilization, substance abuse, or crush injury. Early recognition and timely surgical decompression should be considered to preserve neurological function; conservative management may be an alternative for patients who decline surgery. However, as a single case report, generalizable conclusions cannot be drawn.

Introduction

Rhabdomyolysis (RM) is a life-threatening emergency resulting from skeletal muscle injury. Damaged muscle tissue releases intracellular contents into the systemic circulation, characterized by markedly elevated creatine kinase (CK) levels and myoglobinuria; acute kidney injury (AKI) is a common and serious complication. Gluteal compartment syndrome (GCS) is a rare complication of RM, and sciatic neuropathy secondary to RM-associated GCS is even rarer. Given its rarity and nonspecific early symptoms, this condition is frequently misdiagnosed or subject to delayed diagnosis[1]. We report a case of RM-associated GCS complicated by sciatic neuropathy, describe its clinical presentation, diagnostic workup, treatment course, and prognosis, and discuss the diagnostic and therapeutic challenges encountered in its management.

Case Report

A 38-year-old man presented to our emergency department on September 19 (Day 2) with a 2-day history of low back pain and gross hematuria. On September 17 (Day 0), after sleeping on the floor, he awoke with right-sided lumbar and abdominal distension, cola-colored urine, and left lower extremity weakness that rendered him unable to walk. His symptoms persisted, prompting his presentation to our hospital.

His past medical history was unremarkable, with no history of hypertension, diabetes mellitus, or coronary artery disease. He denied any history of crayfish consumption (a known cause of rhabdomyolysis in China), strenuous physical exertion, alcohol abuse, statin use, or seizure. On physical examination, his height was 178 cm, weight 90 kg (BMI 28.4 kg/m²). Percussion tenderness was noted over the lumbar spine. Motor strength in the left lower extremity was grade 0 (MRC scale), while strength in all other limbs was normal. Emergency laboratory tests demonstrated markedly elevated creatine kinase (CK, 29,696 U/L), CK-MB (186.1 U/L), and C-reactive protein (CRP, 93.9 mg/L); the white blood cell count was 17.75 × 10⁹/L. Serum creatinine was 75 μmol/L, blood urea nitrogen was6.01mmol/L, and serum potassium was 2.90 mmol/L. Urinalysis showeddark tea-colored urine, positive occult blood (BLD), positive protein (PRO, +□+++), and no significant red blood cells on microscopy. MRI of the brain (September 19) showed scattered white matter hyperintensities at the corticomedullary junction of the bilateral frontoparietal lobes, and CT of the lumbar spine (September 19) revealed degenerative changes. These studies were performed to exclude central causes of lower extremity weakness. The patient was admitted with a provisional diagnosis of rhabdomyolysis and lower limb paralysis of unknown etiology. Supportive management was initiated, including fluid resuscitation, hepatoprotective therapy, dehydrating therapy, and neurotrophic therapy. Three days later (September 22), the patient's left lower extremity strength had improved, with proximal strength of approximately grade 3-4 and distal strength of grade 0 (Video 1). His CK level had decreased to 6072 U/L. Ultrasound of the left gluteal region on September 23 showed subcutaneous soft tissue swelling. MRI of the lumbar spine and hip regions on the same day revealed lumbar degeneration, edematous signal changes in the right paraspinal muscles, left gluteal subcutaneous edema with fluid collection, and muscle edema with altered signal intensity involving the tissue spaces, piriformis muscle, and gluteus minimus muscle (Fig 1,2). Electromyography performed on the same day suggested injury to the left tibial nerve and left common peroneal nerve (Table 1).

Given the patient's clinical symptoms and imaging findings, gluteal compartment syndrome was diagnosed clinically (intracompartmental pressure was not measured). The patient was informed of the poor prognosis associated with delayed decompression, and surgical decompression was recommended, but he declined. Therefore, anti-edema and neurotrophic therapies were continued. The patient requested discharge on September 30 (Day 13). At discharge, CK levels had normalized ([180] U/L); however, distal weakness of the left lower limb (foot drop) persisted. Symptomatic pharmacotherapy (including Fursultiamine and Mecobalamin) was continued after discharge.

At 3 months after symptom onset, proximal muscle strength of the left lower limb was grade 5, and distal muscle strength was grade 0-1 (Video 2). Repeat electromyography demonstrated complete injury to the left tibial, common peroneal, sural, and superficial peroneal nerves(Table 1). At 6 months after symptom onset, proximal muscle strength remained grade 5, and distal muscle strength remained grade 0-1. The patient was referred to the Department of Hand Surgery at Shanghai Huashan Hospital, where continued rehabilitation was recommended and elective tendon transfer surgery was advised to improve foot dorsiflexion if necessary.

Discussion

RM is an acute emergency that classically presents with the triad of myalgia, muscle weakness, and cola-colored urine. Severe cases may lead to life-threatening complications, including acute kidney injury (AKI), hyperkalemia, and compartment syndrome[2]. Based on its underlying etiology, RM is classified into four subtypes: traumatic, nontraumatic, hereditary, and miscellaneous[2]. Because RM has heterogeneous etiologies, a meticulous history is essential for making a clinical diagnosis. In our case, the patient denied any history of trauma, strenuous physical exertion, excessive alcohol consumption, or statin use, all of which are classic triggers of RM. He also denied seizure, infection, or known metabolic myopathy. He was obese (BMI 28.4 kg/m²), had an irregular lifestyle, and had a history of bedtime oral estazolam use. He fell to the ground during the night and was found lying on the floor in the early morning, presumably still under the sedative effect of estazolam. We hypothesize that estazolam-induced deep sleep led to sustained passive compression of the left gluteal region and lower extremity for several hours, resulting in muscle ischemia and hypoxia, and ultimately leading to RM. However, serum estazolam concentration was not measured, and this causal relationship remains speculative. This prolonged immobilization likely also contributed to the development of gluteal compartment syndrome, which is discussed below.

Compartment syndrome is a limb-threatening orthopedic emergency caused by elevated intracompartmental pressure. The forearm and leg are the most commonly affected sites; however, GCS is relatively uncommon. Despite incomplete osseous boundaries, the gluteal musculature is encased within thick deep fascia that partitions the buttock into distinct fascial compartments. The gluteal region is divided into three distinct compartments: the gluteus maximus compartment, the combined gluteus medius–minimus compartment, and the piriformis compartment[3]. Since Evanski and Waugh[4] first described GCS in 1977, numerous case reports have been published. A 2022 systematic review[5] identified postoperative complications (41%) and prolonged immobilization due to drug overdose or altered mental status (35%) as the leading causes of GCS, with traumatic causes accounting for only 19%. Ghanem et al. [6] found that healthy young and middle-aged men with a history of polysubstance abuse represent the predominant population affected by GCS, with onset largely attributed to prolonged immobilization following loss of consciousness or iatrogenic injuries. In our patient, prolonged immobilization due to sedative use likely contributed to both RM and GCS.

GCS and lower extremity compartment syndrome differ in their pathogenesis, clinical presentation, neurological involvement, and systemic manifestations. GCS typically presents with nonspecific symptoms, most notably gluteal pain and sciatic nerve dysfunction, and is frequently associated with rhabdomyolysis[1]. Prolonged compression of the affected region causes ischemic necrosis and interstitial edema of skeletal muscle, manifesting as localized pain. Along with electrolyte imbalance (predominantly hyperkalemia), these factors contribute to limb weakness. The sciatic nerve courses through the deep gluteal space and lies adjacent to the fascial compartments[7, 8]. This anatomical relationship renders the nerve vulnerable to compressive ischemic injury, which accounts for the majority of residual neurological dysfunction[7, 8]. Three mechanisms have been proposed to explain such nerve injuries [5, 7, 8]: (1) mechanical nerve trauma from prolonged immobilization; (2) compressive neuropathy secondary to elevated intracompartmental pressure; and (3) muscle breakdown eliciting regional perineural inflammation, leading to secondary ischemic nerve damage. These mechanisms may act synergistically. In our patient, this anatomical vulnerability likely resulted in compressive ischemic injury, which accounts for the persistent foot drop despite normalization of CK levels.

A bidirectional relationship likely exists between GCS and RM[5, 9]. In our patient, the two conditions appeared to exacerbate each other: muscle breakdown from prolonged compression contributed to elevated CK levels, while elevated intracompartmental pressure further compromised the sciatic nerve and aggravated muscle injury. In 2022, Adib et al.[5] performed a systematic review and meta-analysis of 139 patients with GCS, reporting rhabdomyolysis in 94% and sciatic nerve injury in 74%. Seok et al.[9] identified 28 patients with rhabdomyolysis attributable to prolonged immobilization, of whom 7 had concomitant lower limb peripheral nerve lesions involving the sciatic nerve and lumbosacral plexus. Nath et al. [10] reported that secondary foot drop occurred in 0.4% of patients with rhabdomyolysis. Although hemodialysis effectively manages acute kidney injury and electrolyte imbalances in patients with rhabdomyolysis, foot drop and other irreversible neurological sequelae often persist. These findings highlight the importance of early diagnosis and timely surgical decompression.

Acute compartment syndrome can lead to lower extremity neurovascular injury and requires urgent fasciotomy. However, unlike conventional lower extremity compartment syndrome, the management of GCS requires comprehensive assessment of renal function, neurological deficit severity, and muscle enzyme levels to develop individualized treatment strategies[5], because GCS is frequently complicated by rhabdomyolysis and acute kidney injury. Based on their systematic review, Adib et al.[5] developed a treatment algorithm in which fasciotomy indications are determined by the patient's neurological status at initial assessment. For patients with early-confirmed GCS who present with neurological deficits at initial assessment, urgent surgical decompression is recommended. Patients without neurological impairment should first undergo conservative management, while urgent surgical decompression is indicated if new neurological deficits develop during conservative therapy. However, clinicians face considerable challenges in diagnosing GCS, and no consensus has been reached on the management of delayed or missed GCS[11]. This is particularly relevant to our case, in which the diagnosis was delayed and the patient declined surgery.

Fasciotomy is generally recommended within 6 hours of symptom onset in patients with compartment syndrome[12]. Surgical decompression performed more than 12 hours after symptom onset is associated with substantially poorer clinical outcomes[12]. Earlier studies demonstrated that fasciotomy for compartment syndrome secondary to severe crush injuries is associated with major adverse events, including surgical site infection, sepsis, and risk of amputation[11]. Finkelstein et al.[13] defined delayed fasciotomy as surgical decompression performed ≥ 35 hours after symptom onset. They proposed that surgical decompression may remain preferable to conservative management even beyond this threshold if patients exhibit progressive neurological deterioration or persistently elevated muscle enzyme levels despite conservative treatment. In contrast, Lawrence et al.[11] reported a case of delayed surgical decompression performed more than 56 hours after GCS symptom onset. The patient exhibited immediate and remarkable neurological improvement and achieved complete functional recovery at the 8-week follow-up. However, Panagiotopoulos et al.[14] reported a case of GCS resulting from prolonged immobilization secondary to substance abuse. Despite fasciotomy performed 12 hours after symptom onset, residual sciatic nerve palsy persisted at the 1-year follow-up. Adib et al.[5] reported that only 60% (75/125) of enrolled patients received a definitive clinical diagnosis of GCS. Of the 80% (111/138) of patients who underwent surgical decompression, 41% had persistent, irreversible neurological deficits. These findings underscore the diagnostic and therapeutic challenges encountered in our case.

Given the substantial etiological heterogeneity of GCS—with triggers including crush injuries, substance abuse, and prolonged immobilization—and the lack of a standardized early warning scoring system, clinicians may not maintain adequate vigilance when encountering these patients. Delayed diagnosis of GCS precludes timely surgical decompression, a major contributor to poor long-term functional outcomes. Surgeons often face a therapeutic dilemma when managing patients with delayed presentation of GCS. Although delayed fasciotomy may still confer neurological recovery, it is associated with significantly increased risks of surgical site infection and wound complications, especially in patients with crush injury-related GCS. Delayed presentation (2 days after symptom onset) and diagnostic delay (2–3 days after initial presentation) both complicated treatment decisions and likely affected the clinical prognosis in our case. Our patient declined surgical decompression, reflecting a common clinical dilemma in GCS management. Despite normalization of creatine kinase levels, our patient had persistent distal weakness at 3 and 6 months, consistent with the observation that foot drop is associated with a poor prognosis in GCS-associated sciatic neuropathy.

This case report has several limitations. First, intracompartmental pressure was not measured; therefore, the diagnosis of GCS was based on clinical and imaging findings rather than direct pressure measurement. Second, serum sedative concentration was not measured, so the causal relationship between sedative-associated prolonged immobilization and rhabdomyolysis was inferred solely from clinical history. Third, the patient declined surgical decompression, which precluded evaluation of the potential therapeutic benefit of early surgery in this case. Finally, as a single case report, the findings cannot be generalized and should be interpreted with caution.

Despite these limitations, this case provides important insights into the diagnosis and management of GCS. We report a rare case of nontraumatic RM complicated by GCS and subsequent sciatic neuropathy, possibly associated with estazolam-related prolonged immobilization. The patient's nonspecific initial presentation, delayed diagnosis, and refusal of surgical decompression likely contributed to persistent foot drop at the 6-month follow-up. The management of GCS may differ from that of lower extremity compartment syndrome and requires individualized consideration. The diagnosis of GCS requires a high index of suspicion, particularly in high-risk populations, including those with prolonged immobilization, substance abuse, or crush injury. Early recognition and timely surgical decompression should be considered to preserve neurological function. Future studies should investigate the optimal timing of surgical decompression in patients with delayed-presentation GCS.

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Committee of Hangzhou Hospital of Traditional Chinese Medicine. All procedures performed were in accordance with the Declaration of Helsinki and relevant national laws and regulations. Written informed consent was obtained from the patient to report and publish individual patient data.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

Yake Meng and Hao Pan conceived and designed the case report. Yake Meng drafted the manuscript. Fenggui Zhu and Si Wang contributed to the diagnosis and management of rhabdomyolysis and acute kidney injury. Rongxue Shao and Jiaming Liang contributed to the orthopedic evaluation and clinical decision-making regarding gluteal compartment syndrome. Wei Fang performed and interpreted the electromyographic studies. Huiguo Chen contributed to the collection and interpretation of imaging data.

Hao Pan critically revised the manuscript for important intellectual content and supervised the study. All authors read and approved the final manuscript.

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Availability of data and materials

Data availability

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Figures

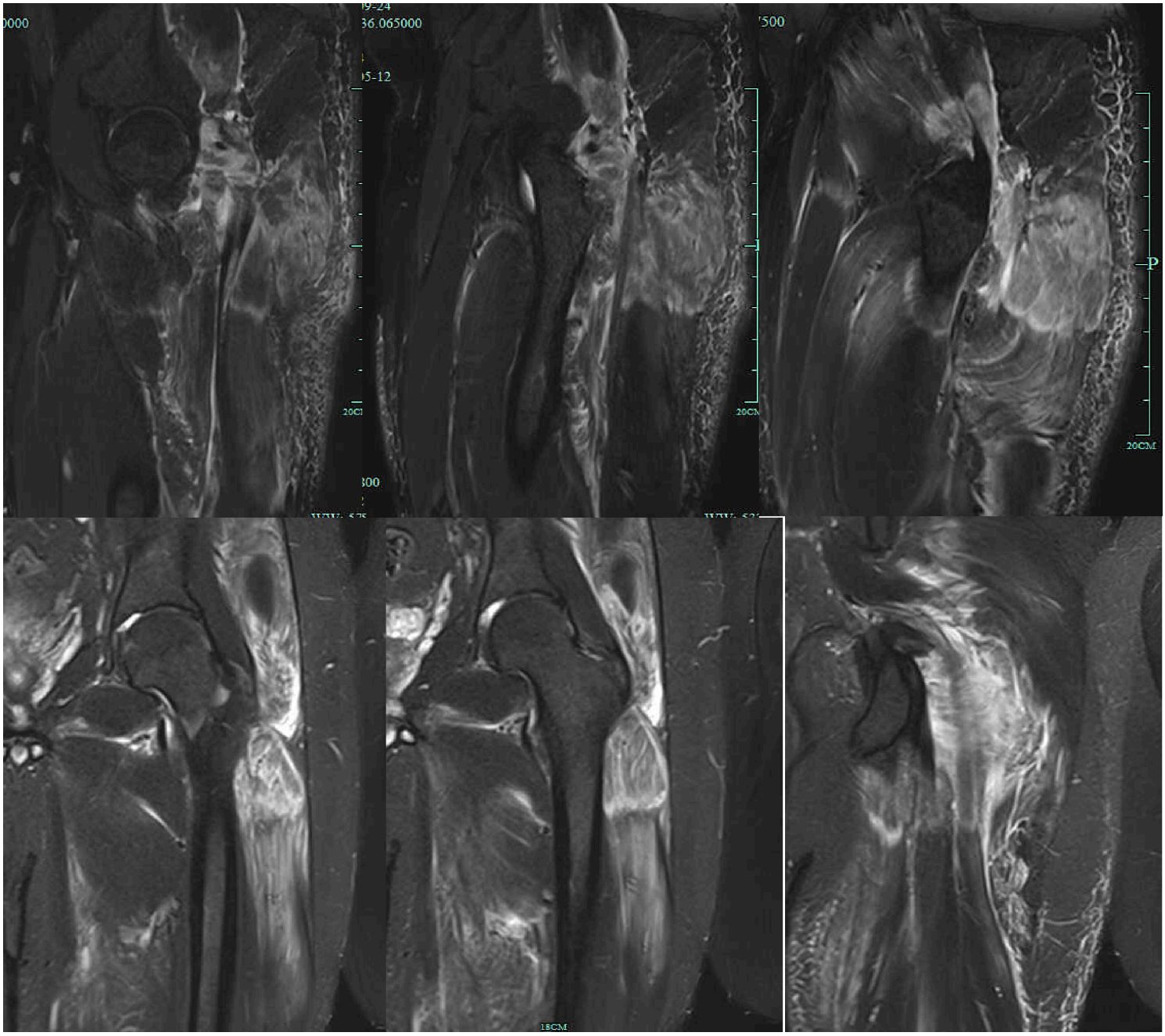


Figure 1

Sagittal and coronal MRI of the hip showed edema and swelling of the left gluteus minimus, gluteus medius, and gluteus maximus muscles.

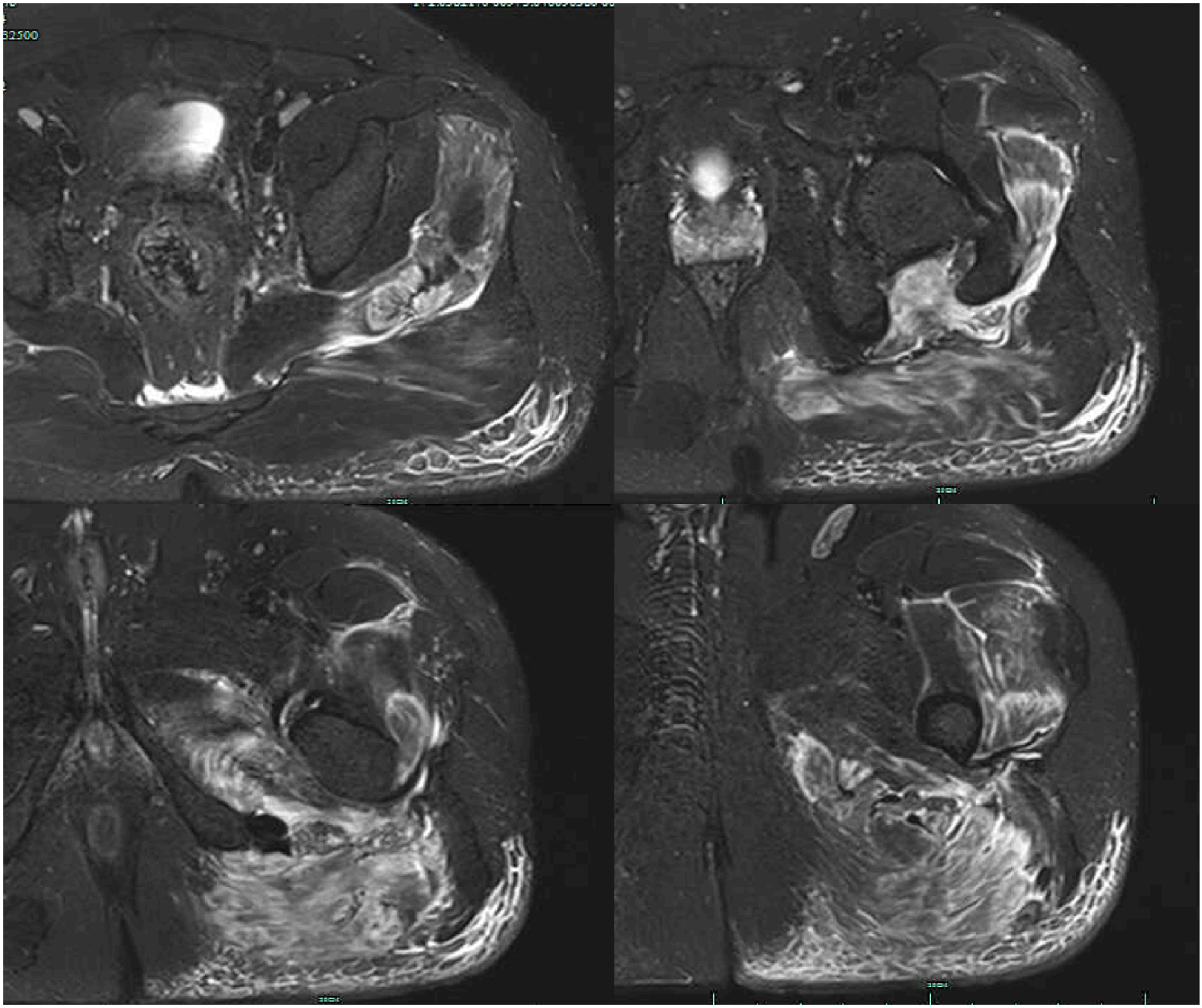


Figure 2

Axial MRI of the hip showed edema and swelling of the left gluteus minimus, gluteus medius, and gluteus maximus muscles.

Supplementary Files

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