

Acute kidney injury due to perioperative rhabdomyolysis during meningioma surgery

Ahmed Amine JAOUAHAR ^{1, *}, Achraf MOUSSA ², Hatim BELFQUIH ², Omar MAOUJOUR ¹, Mohammed ASSERRAJI ¹, Ali AKHADDAR ² and Nadir ZEMRAOUI ¹

¹ Department of Nephrology and Hemodialysis, Avicenna Military Hospital, Marrakesh, Morocco.

² Department of Neurosurgery, Avicenna Military Hospital, Marrakesh, Morocco.

World Journal of Advanced Research and Reviews, 2025, 25(02), 2351-2354

Publication history: Received on 15 January 2025; revised on 22 February 2025; accepted on 25 February 2025

Article DOI: <https://doi.org/10.30574/wjarr.2025.25.2.0625>

Abstract

Acute kidney injury (AKI) due to perioperative rhabdomyolysis is a rare but severe complication that can occur during prolonged surgical procedures, especially in patients with predisposing factors such as obesity, diabetes, and metabolic disorders. This case report describes a 40-year-old woman with a history of non-insulin-dependent diabetes and obesity, who developed AKI following a 10-hour meningioma surgery. During the operation, she received low-dose anesthetic agents, including propofol and rocuronium. Postoperatively, she developed oliguric acute kidney injury, accompanied by hyperkalemia and elevated creatine phosphokinase (CPK) and myoglobin levels, confirming rhabdomyolysis. The patient was managed with daily dialysis, resulting in gradual urine clearance, improved renal function, and resolution of muscle damage markers. This case highlights the importance of early diagnosis, prevention, and management of rhabdomyolysis in the perioperative setting, particularly in high-risk patients, to prevent severe complications like AKI.

Keywords: Rhabdomyolysis; Acute Kidney Injury; Hemodialysis; Meningioma

1. Introduction

Rhabdomyolysis is characterized by the breakdown of skeletal muscle fibers, leading to the release of intracellular electrolytes and proteins into the systemic circulation. It is a clinico-biological syndrome that can rapidly become life-threatening due to hyperkalemia in the short term or acute kidney injury (AKI) in the medium term. While the most well-known causes include prolonged muscle ischemia and severe peripheral trauma, postoperative rhabdomyolysis is a recognized entity, particularly in anesthetic settings. In this context, anesthetic agents—especially propofol—and prolonged muscle compression during lengthy surgical procedures are the most frequently implicated factors.

2. Observation

A 40-year-old female with a history of non-insulin-dependent diabetes mellitus (NIDDM) under oral antidiabetic therapy (Metformin 850 mg twice daily), was admitted to the neurosurgery department for the management of a left parietal cerebral meningioma. The patient was overweight (89 kg, height 1.66 m, BMI = 32), but had no history of diabetic complications, including no neuropathy nor micro/macroangiopathy.

Preoperative renal function tests were normal. The surgical procedure lasted 10 hours, during which the patient received 1200 mL of intravenous fluids versus an estimated blood loss of 400 mL. The anesthetic protocol included a relatively low dose of neuromuscular blockade with Rocuronium Bromide (0.8 mg/kg for induction, followed by 0.15

* Corresponding author: Ahmed Amine JAOUAHAR

mg/kg for maintenance) and Propofol (2.2 mg/kg, equivalent to 200 mg for induction, followed by an infusion at approximately 6 mg/kg/h for the 10-hour duration of the procedure).

The patient developed dark reddish, non-clotted urine at the end of the surgery (**Figure 1**), followed by the onset of oligo-anuric acute kidney injury (AKI) with hyperkalemia (6.1 mEq/L). Laboratory findings on day two post-surgery revealed elevated creatine phosphokinase (CPK) levels (15 times the upper normal limit) and myoglobin levels of 1100 IU/L, strongly suggestive of rhabdomyolysis.



Figure 1 Patient's dark brown urine aspect of rhabdomyolysis

The patient underwent daily 3-hour intermittent hemodialysis sessions for 18 days. By the end of the first week the patient was no more oliguric, and we observed a progressive clearing of the urine. Rhabdomyolysis markers showed a significant decline, becoming undetectable by day 16 post-surgery, and creatinine levels normalized a few weeks after.

3. Discussion

The term rhabdomyolysis refers to the breakdown of striated skeletal muscle fibers, leading to cellular lysis and the release of intracellular contents into the bloodstream. This muscle cell injury can result from an intrinsic structural or hereditary defect, but it most commonly occurs secondary to trauma, infection, intoxication, or other external factors [1,2]. This phenomenon is typically associated with myalgias, cramps, and abnormal urine discoloration (tea-colored urine), primarily due to myoglobinuria. Rhabdomyolysis is classically defined by an elevated serum creatine phosphokinase (CPK) level, along with increased urinary excretion of myoglobin, known as myoglobinuria [3]. In plasma, myoglobin is typically bound to proteins, which limits its tissue diffusion, particularly at the renal level. Rhabdomyolysis occurring in a surgical context is a rare complication.

Several severe complications associated with rhabdomyolysis can be life-threatening. The most critical is hyperkalemia, which can lead to cardiac arrhythmias, as well as tubular obstruction, potentially resulting in oliguric renal failure. Other potential complications include fever, tubular necrosis due to myoglobin deposition, hypovolemia, leukocytosis, metabolic acidosis caused by the release of intracellular sulfate and phosphate, initial hypocalcemia due to calcium carbonate precipitation in damaged tissues (which can exacerbate hyperkalemia), hyperphosphatemia, and disseminated intravascular coagulation. Certain medical history factors (such as diabetes, obesity, smoking, and alcoholism), either associated with or independent of hereditary muscular fragility (e.g., myopathies) or acquired conditions (such as those induced by HMG-CoA reductase inhibitors), are the most common risk factors for postoperative rhabdomyolysis. These events have primarily been reported in relation to certain intraoperative positions, including gynecological positioning, lateral decubitus, and dorsal decubitus during abdominal aortic or digestive surgery, particularly in obese patients [4-8].

Propofol infusion syndrome (PRIS) is a rare clinico-biological entity whose mechanisms are still not fully understood. It is characterized by cardiac arrhythmias, metabolic acidosis, hyperlipidemia, muscular damage with rhabdomyolysis, and myoglobinuria. PRIS is typically reported in intensive care units following prolonged administration of propofol (lasting more than 48 hours) at high doses (greater than 4 mg/kg/h) [9]. However, several cases of PRIS have been reported following a shorter infusion duration and at lower dosages [10,11].

In our case, PRIS wasn't the sole implicated factor in the acute episode of rhabdomyolysis, considering the relatively short exposure duration to the drug compared to what is reported in the literature. However, the combination of multiple risk factors present in our patient, including the long duration of surgery, obesity, and diabetes, may explain the rhabdomyolysis. The diagnosis is typically made based on the presence of one or more causes of rhabdomyolysis, and is usually supported by clinical symptoms, which can vary, and the nearly constant biological markers of muscle injury. The elevation of CPK, which is a good indicator of the volume of muscle affected, is the only sign consistently observed. However, it does not reliably predict the onset of acute renal failure, which is often influenced by the combination of several etiological factors.

In certain challenging contexts (e.g., poor clinical examination, localized lesions), imaging techniques (magnetic resonance imaging, CT scan, ultrasound) can help guide the diagnosis. Scintigraphy methods are mainly used to assess the extent of the lesions. Due to its sensitivity, MRI is the most effective examination. It allows for the identification of very suggestive signs of rhabdomyolysis, such as hyperintensities on T2-weighted images in the subcutaneous fat, superficial and deep muscular fasciae [12]. MRI can also assist in identifying muscle groups that, due to significant fluid accumulation, require immediate decompression [13]. CT scan and ultrasound are much less specific, and their contribution remains limited in diagnosing rhabdomyolysis [14,15]. In our case, given the strongly suggestive context and the presence of several clinical and biological elements supporting this complication, no imaging was requested to confirm the diagnosis.

Preventive measures are particularly important during prolonged surgeries in patients with risk factors and include: discontinuing HMG-CoA reductase inhibitors (statins) 5 to 7 days before major surgery, careful patient positioning to avoid compression and minimize thigh flexion over the pelvis and dorsiflexion, regular monitoring of cutaneous, neural, and vascular territories, and maintaining stable perioperative hemodynamics, with an emphasis on adequate extracellular volume expansion. This improves renal blood flow, reduces vasoconstrictive stimuli, and may potentially limit exposure to nephrotoxic agents by increasing urinary output

4. Conclusion

The incidence of perioperative rhabdomyolysis is low, which often leads to a delayed diagnosis. A good understanding of the contributing factors helps to suspect the condition, identify it through muscle enzyme measurements, implement careful preventive measures, and initiate early, appropriate treatment to prevent renal failure. The coexistence of certain risk factors and prolonged surgery (duration > 5 hours), as seen in our case of meningioma surgery, should always raise suspicion. The prognosis of renal function and, in some cases, even the patient's survival, depends on the timeliness of the diagnosis. Curative treatment is primarily based on fluid management and forced alkaline diuresis. It is supplemented by renal replacement therapy, especially in refractory acidosis and severe hyperkalemia.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

References

- [1] Knochel JP. Mechanisms of rhabdomyolysis. *Curr Opin Rheumatol* 1993;5:725–31. <https://doi.org/10.1097/00002281-199305060-00006>.
- [2] Warren JD, Blumbergs PC, Thompson PD. Rhabdomyolysis: A review. *Muscle & Nerve* 2002;25:332–47. <https://doi.org/10.1002/mus.10053>.

- [3] David WS. Myoglobinuria. *Neurol Clin* 2000;18:215–43. [https://doi.org/10.1016/s0733-8619\(05\)70187-0](https://doi.org/10.1016/s0733-8619(05)70187-0).
- [4] Torres-Villalobos G, Kimura E, Mosqueda JL, García-García E, Domínguez-Cherit. G, Herrera MF. Pressure-induced Rhabdomyolysis after Bariatric Surgery. *OBES SURG* 2003;13:297–301. <https://doi.org/10.1381/096089203764467252>.
- [5] Osamura N, Takahashi K, Endo M, Kurumaya H, Shima I. Lumbar Paraspinal Myonecrosis After Abdominal Vascular Surgery: A Case Report. *Spine* 2000;25:1852–1854.
- [6] Ferreira J, Galle C, Aminian A, Michel P, Guyot S, De Wilde J-P, et al. Lumbar paraspinal rhabdomyolysis and compartment syndrome after abdominal aortic aneurysm repair. *Journal of Vascular Surgery* 2003;37:198–201. <https://doi.org/10.1067/mva.2003.108>.
- [7] Filis D, Daskalakis M, Askoxylakis I, Metaxari M, Melissas J. Rhabdomyolysis Following Laparoscopic Gastric Bypass. *OBES SURG* 2005;15:1496–500. <https://doi.org/10.1381/096089205774859317>.
- [8] Stroh C, Hohmann U, Remmler K, Urban H, Meyer F, Lippert H, et al. Rhabdomyolysis after Biliopancreatic Diversion with Duodenal Switch. *OBES SURG* 2005;15:1347–51. <https://doi.org/10.1381/096089205774512672>.
- [9] Masson E. Le syndrome de perfusion du propofol. *EM-Consulte* n.d. <https://www.em-consulte.com/article/253671/le-syndrome-de-perfusion-du-propofol> (accessed September 19, 2021).
- [10] Koch M, Backer DD, Vincent J-L. Lactic acidosis: An early marker of propofol infusion syndrome? *Intensive Care Med* 2004;30:522–522. <https://doi.org/10.1007/s00134-003-2130-3>.
- [11] Withington DE, Decell MK, Ayed TA. A case of propofol toxicity: further evidence for a causal mechanism. *Pediatric Anesthesia* 2004;14:505–8. <https://doi.org/10.1111/j.1460-9592.2004.01299.x>.
- [12] Kakuda W, Naritomi H, Miyashita K, Kinugawa H. Rhabdomyolysis Lesions Showing Magnetic Resonance Contrast Enhancement. *Journal of Neuroimaging* 1999;9:182–4. <https://doi.org/10.1111/jon199993182>.
- [13] Shintani S, Shiigai T. Repeat MRI in acute rhabdomyolysis: correlation with clinicopathological findings. *J Comput Assist Tomogr* 1993; 17:786–91. <https://doi.org/10.1097/00004728-199309000-00023>.
- [14] Russ PD, Dillingham M. Demonstration of CT hyperdensity in patients with acute renal failure associated with rhabdomyolysis. *J Comput Assist Tomogr* 1991; 15:458–63. <https://doi.org/10.1097/00004728-199105000-00021>.
- [15] Steeds RP, Alexander PJ, Muthusamy R, Bradley M. Sonography in the diagnosis of rhabdomyolysis. *Journal of Clinical Ultrasound* 1999;27:531–3. [https://doi.org/10.1002/\(SICI\)1097-0096\(199911/12\)27:9<531::AID-JCU7>3.0.CO;2-8](https://doi.org/10.1002/(SICI)1097-0096(199911/12)27:9<531::AID-JCU7>3.0.CO;2-8).