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INDEX

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SCIENTIFIC ARTICLE - CASE REPORT

BALANCED GENERAL ANESTHESIA IN A PATIENT WITH HEMOPHILIC ARTHROPATHY: A CASE REPORT

MATHEUS SILVA DE OLIVEIRA[1], ESTEVAM BORGES LOPES[1], GABRIEL PEIXOTO DO NASCIMENTO[1], DAVYD FONSECA ANDRADE[1], GUSTAVO SIQUEIRA ELMIRO[1], GIULLIANO GARDENGHI[1,2]

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ABSTRACT

Introduction: One of the main complications observed in patients diagnosed with Hemophilia B is hematrosis, a chronic joint condition that causes pain and limited movement, often requiring total arthroplasty of the affected joint. **Case report:** Patient MGS, male, 19 years old, with Hemophilia B, diagnosed with hemophilic arthropathy of the left knee. General anesthesia was chosen for the case, unlike neuraxial blockade due to a higher risk of spinal canal hematoma. The procedure was uneventful. **Discussion:** Hemophilic arthropathy is characterized by a chronic and progressive inflammatory process that leads to stiffness and limited joint movement. Treatment includes conservative management, which may evolve to the need for synovectomy, arthrodesis and, in more severe cases, arthroplasty. However, since hemophilic patients have a higher risk of significant bleeding, some considerations are relevant, such as avoiding unnecessary airway management, avoiding mucosal trauma and avoiding neuraxial blockade due to the risk of causing spinal canal hematoma. Therefore, general anesthesia would be the safest option for such patients. It is also worth mentioning that optimizing coagulation factor levels in the preoperative period can reduce complications, as can the administration of activated recombinant factor VII. Therefore, this article presents a case report of a hemophiliac patient with a high risk of bleeding, requiring specific anesthetic considerations to minimize the chance of major hemorrhagic complications.

Keywords: Hemophilia B, Hemartrhosis, General anesthesia, Arthroplasty, Spinal anesthesia.

INTRODUCTION

Characterized as a coagulation disorder caused by a deficiency of factor IX, Hemophilia B results from an X-linked recessive disorder, with no predilection for geographic area or ethnicity, occurring in approximately 1 in 30,000 male births.[1]

One of the main characteristics of the disease is hemarthrosis, especially in the joints of the knees, elbows, and ankles, causing pain and limited mobility. Intra-articular bleeding leads to synovitis, where proteolytic enzymes cause cartilage degeneration, characterizing hemophilic arthropathy,

which may also present with arthritis, restricted movement, and chronic pain.[1]

From the anesthetic perspective, in many cases that are indicated and eligible for surgical treatment of such complications—such as total knee arthroplasty, a common procedure among patients with hemophilic arthropathy—one would expect the use of neuraxial blockade as the anesthetic technique for this approach, given its low complication rate, shorter hospital stay, and fewer admissions to the intensive care unit (ICU). However, the possibility of bleeding and the formation of a spinal epidural hematoma (SEH) can lead to permanent neurological complications, especially if not diagnosed and treated in a timely manner.[1,2]

Thus, the aim of this case report is to address the particularities of anesthetic management in a patient with Hemophilia B and the challenges involved in formulating an anesthesia plan that ensures greater safety and minimizes risks during the procedures performed.

CASE REPORT

Patient M.G.S., male, 19 years old, 160 cm tall, weighing 62 kg. Diagnosed with Hemophilia B at 8 months of age, with hemophilic arthropathy in the left knee, previously undergoing synovectomy and arthroplasty of that joint (in 2018), and currently presenting with a pathological fracture of the distal femur and proximal tibia.

The patient was evaluated by the hematology team during the pre-anesthetic assessment, with the recommendation to administer factor VIIa at a dose of 6 mg every 4 hours, and every 3 hours on the day of surgery. In addition, the use of tranexamic acid was authorized, with a recommended dose of 500 mg every 6 hours. The use of red blood cell transfusion was also approved if necessary, using leukocyte-filtered and washed units. The use of nonsteroidal anti-inflammatory drugs (NSAIDs) was to be avoided due to their platelet inhibition effect.

The patient was then taken to the operating room, where multiparametric monitoring was established (ECG, SpO_2, Non-Invasive Blood Pressure – NIBP, capnography, Bispectral Index (BIS) cerebral monitor); prior central venous access was established in the right internal jugular vein; pre-oxygenation was performed with 100% O_2. Anesthesia induction was carried out intravenously with 120 mg of propofol, 20 micrograms of sufentanil, 25 mg of ketamine, and 100 mg of rocuronium. Orotracheal intubation (OTI) was performed using a 7.0 cuffed tube without complications, confirmed by capnography on the monitor. Anesthetic maintenance was done with 2% sevoflurane and remifentanil via target-controlled infusion (TCI). The patient was then positioned in the horizontal dorsal decubitus position, with thermal and ocular protection, the tube properly secured, and protection for joint and pressure points. During the procedure, administration of factor VIIa was carried out as recommended by the hematology service, along with 1 g of tranexamic acid and adjunct medications (4 mg dexamethasone, 2 g dipyrone). After approximately 60 minutes of surgery, the surgeon reported severe diffuse bleeding in the surgical field, difficult to control hemostasis, associated with visualization of severe fractures of the distal femur—supracondylar and intercondylar—and proximal tibia, showing signs of marked osteoporosis. Therefore, revision arthroplasty was chosen because surgical reduction with osteosynthesis was not feasible for the condition. The surgeon applied 3 g of Arista® hemostatic powder with good bleeding control. Additionally, prothrombin complex concentrate and two units of leukocyte-filtered packed red blood cells were administered. During the intraoperative period, the patient experienced sustained sinus tachycardia without

hemodynamic compromise, which improved after administration of 2 mg of metoprolol.

The procedure proceeded without major complications. Postoperative pain prevention was provided with 10 mg of morphine, remifentanil was discontinued, neuromuscular blockade was reversed with 200 mg of sugammadex, and the patient awoke and was extubated in the operating room without any adverse events. The patient was then transferred to the ICU for close monitoring and immediate postoperative care.

DISCUSSION

Hemophilic arthropathy is characterized by recurrent intra-articular bleeding, leading to the deposition of hemosiderin in the synovial tissues, which induces hypertrophy, neovascularization, and fibrosis of these tissues. These processes can cause inflammatory reactions and cartilage degeneration, which manifest as frequent pain, joint deformity, stiffness, and limited range of motion.[3]

Initially, treatment is based on conservative management. As the condition progresses, surgical procedures may become necessary, such as synovectomy (previously performed on the patient in this case), arthrodesis, and, finally, arthroplasty, as in the case presented.[3]

Since hemophilic patients have a higher risk of bleeding, it is recommended that their surgical procedures be scheduled for the first slot of the day, as well as at the beginning of the week, to ensure better support from the blood bank if needed. Anesthesiologists managing such cases should preferably have experience in handling hematological disorders.[3,4] Regarding anesthetic management, it is important to recognize that even airway manipulation can cause trauma with significant mucosal bleeding, as can more invasive interventions such as intubation, deep venous access, peripheral nerve blocks, and neuraxial blocks. To minimize these risks, measures are recommended, including lubrication of airway management equipment, the use of videolaryngoscopy to reduce the chance of trauma, avoiding nasotracheal intubation when possible, padding of joints at pressure points, and even avoiding the use of succinylcholine due to the theoretical risk of worsening intramuscular hematomas and hemarthrosis caused by the drug-induced fasciculations.[3,4]

Due to its low complication rate, regional anesthesia is an excellent option for patients requiring orthopedic procedures on the lower limbs, such as knee surgeries. This anesthetic modality reduces hospital stay duration and decreases the number of ICU admissions. However, a feared and potentially catastrophic complication when performing neuraxial anesthesia (subdural or epidural) in patients at increased risk of bleeding is spinal epidural hematoma (SEH), which must be diagnosed and treated within 8 to 12 hours of its formation. If not managed within this timeframe, it can lead to neurological complications due to spinal cord compression.[2] Despite this possible complication, SEH is a rare condition, with increased incidence in elderly patients, females, those using anticoagulants, and in the presence of vertebral canal diseases (such as stenosis, scoliosis, among others).[2]

Thus, some authors recommend general anesthesia for surgical procedures in these patients to reduce the risk of spinal epidural hematoma (SEH).[3] One of the key points in the preoperative management of these patients, aimed at minimizing postoperative complications such as infections and surgical failure, is the optimization of coagulation factor levels: maintaining factor IX levels between 60-80 IU/dL in patients with Hemophilia B undergoing major surgery, gradually

decreasing to approximately 50% until the surgical wound is fully healed. Administration is typically guided by clinical estimates of intraoperative blood loss, preferably kept below 25%.[3,4] These values may vary depending on the literature consulted, as there is no well-defined protocol yet for preoperative coagulation factor levels in this population.[3,4] Therefore, this remains a controversial topic, as demonstrated in a systematic review by Togioka et al., which showed low evidence supporting the need for factor IX levels above 50% for neuraxial anesthesia approaches.[5]

Another possible approach for these patients, both as prophylaxis and treatment for intraoperative bleeding, is the administration of recombinant activated factor VII (rFVIIa), even after the use of factor IX (in the case of Hemophilia B), as was done with the patient in the clinical case, under the guidance of the hematology team. This practice directly activates factor X, leading to thrombin formation independently of factors VIII and IX in hemophilic patients.[6] A review article by Vince and Brandão in 2009 showed that the use of rFVIIa began in the 1980s and started gaining popularity from 1999 onward, with promising results. However, despite many studies demonstrating a reduction in the need for blood transfusions in these patients, the lack of consensus in the literature regarding the ideal dosing regimen and the potential complications associated with rFVIIa use (such as thromboembolic effects) highlight the need for further research on the topic.[6]

Furthermore, Marrone et al.[7] reported a case in which a peripheral nerve block (PNB), specifically an adductor canal block and sacral erector spinae plane block, was performed for a total knee arthroplasty in a hemophilic patient, in order to avoid neuraxial blockade. This approach provided satisfactory analgesia during surgery and up to 72 hours postoperatively, with pain levels up to 4/10, using only periodic acetaminophen. Despite this relevant result, PNBs in patients with a high risk of bleeding should be considered as having a high probability of hemorrhagic complications and may require compression of the puncture site, similar to neuraxial approaches.[2]

CONCLUSION

Because they have a higher risk of hemorrhagic complications during anesthetic and surgical procedures, patients with a history of hemophilia require special attention regarding such adverse events, with important considerations aimed at reducing the risk of difficult-to-control bleeding and its possible catastrophic consequences. In this context, the complexity and diversity of anesthetic management options for these patients pose a significant challenge for the anesthesiologist, who must work closely with the hematology team to ensure the greatest perioperative benefit and safety, as was done in the patient in this clinical case.

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EDITORIAL AND REVIEW

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SCIENTIFIC ARTICLE - CASE REPORT

ANESTHESIA FOR MITRAL VALVE REPLACEMENT IN A PATIENT WITH BETA THALASSEMIA MAJOR : CASE REPORT

MATEUS FERREIRA DE SIQUEIRA E SILVA[1], VICTOR TAVARES TRINDADE[2], STANLEY DE OLIVEIRA LOYOLA[2], GUSTAVO SIQUEIRA ELMIRO[2], ARTUR HENRIQUE DE SOUZA[2], GIULLIANO GARDENGHI[1,2,3]

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ABSTRACT

Post-spinal Thalassemias constitute a heterogeneous group of inherited genetic disorders characterized by deficient synthesis of hemoglobin polypeptide chains, with beta-thalassemia being the most prevalent autosomal recessive hereditary hematologic disease. Caused by mutations in the beta-globin gene on chromosome 11, it leads to anemia with symptoms such as fatigue, dyspnea, weakness, headaches, and hepatosplenomegaly. Peripheral blood smears typically reveal anisocytosis, poikilocytosis, and numerous nucleated erythrocytes. The standard treatment involves lifelong regular blood transfusions and chelation therapy to manage iron overload. This report details the anesthetic and surgical management of a complex patient with beta-thalassemia major undergoing mitral valve replacement. The patient, who also presented with hypertension, diabetes, hypothyroidism, pulmonary hypertension, gastroesophageal reflux disease, and generalized anxiety disorder, received multimodal balanced general anesthesia combined with interfascial blockade of the serratus and deep pectoralis muscles. A minimally invasive biological mitral valve replacement was successfully performed. Postoperatively, the patient exhibited stable hemodynamic and ventilatory parameters and was extubated in the operating room without complications. The discussion emphasizes the importance of regular serum ferritin monitoring in transfusion-dependent patients and the necessity of meticulous perioperative care to prevent complications such as hypoxemia and hypovolemia, particularly given the risk of erythrocyte sickling in this population.

Keywords: Beta-Thalassemia, Thoracic Surgery, Mitral valve, Anemia, Pulmonary hypertension.

INTRODUCTION

Thalassemias comprise a heterogeneous class of inherited genetic conditions defined by a quantitative deficiency in the production of one or more polypeptide chains constituting the hemoglobin molecule.[1,2] Beta-thalassemia is recognized as the most prevalent autosomal recessive hereditary hematologic disorder among various populations and results from mutations in the β-globin gene, located on chromosome. [11] To date, more than 350 distinct mutations responsible

for this condition have been identified, with most representing point mutations situated within functionally significant regions associated with the gene expression of the globin protein.[3]

In the context of β-thalassemia, a reduced synthesis of the beta-globin chain leads to a relative excess of the alpha-globin chain, another essential component of hemoglobin. Consequently, these excess alpha-globin chains aggregate, resulting in damage to red blood cells and subsequent hemolysis. Clinically, individuals with β-thalassemia present with anemia and its associated sequelae, including fatigue, dyspnea, weakness, and headaches, potentially extending to hepatosplenomegaly, growth retardation, cardiac complications, pancreatic alterations, and diabetes.[4,5] The clinical manifestations observed in β-thalassemia major (BTM) are directly influenced by two main sets of factors:

1) the severity of chronic hemolysis, the body's capacity to compensate for anemia, the effects of chronic hypoxia, splenomegaly, and skeletal changes. Consequently, more effective transfusion regimens are associated with a lower risk and reduced severity of these complications;

2) the risks associated with red blood cell transfusions, such as transmission of infectious agents and alloimmunization, but critically, complications arising from iron overload. The latter depends on the efficacy and adherence to iron chelation therapy, aiming to maintain a negative iron balance, mitigating the adverse effects associated with excessive use of chelating agents.[6]

Consequently, the initial suspicion of β-thalassemia minor often arises from a complete blood count indicating microcytic and hypochromic anemia. The characteristic peripheral blood smear in the major form shows anisocytosis, poikilocytosis, and a substantial number of nucleated erythrocytes. Definitive diagnosis requires hemoglobin electrophoresis and genetic analysis (DNA sequencing) of the parents, confirming their mandatory carrier status for a thalassemia gene.[7] Individuals who present with severe anemia in early childhood, requiring regular blood transfusions from that point onward, are classified as carriers of β-thalassemia major (BTM).[8] Consequently, treatment usually involves a lifelong regimen of regular blood transfusions and chelation therapy to control iron overload. Hematopoietic stem cell transplantation from healthy donors represents a potentially curative option, although its application is limited due to the substantial risks associated with the procedure and its high cost.[9] Inadequate treatment and the inherent pathophysiology of BTM are strongly correlated with a higher mortality rate in affected individuals, with specific comorbidities occurring more frequently, including heart disease, infections, liver disease, malignancy, thromboembolism, and severe anemia.[10]

Considering the rarity of the disease along with the specific surgical procedure performed on the patient in this report, the present study aims to document the anesthetic and surgical management employed, as well as to synthesize relevant data from the existing literature regarding the anesthetic management of patients with β-thalassemia major (BTM) in various surgical scenarios.

CASE REPORT

A 43-year-old female patient, measuring 164 cm in height and weighing 79 kg, with a medical history of hypertension, diabetes mellitus, hypothyroidism, pulmonary hypertension, gastroesophageal reflux disease, beta-thalassemia major, and generalized anxiety disorder, underwent mitral valve replacement on October 8, 2024, at Hospital Encore in Aparecida de Goiânia, Brazil. The patient reported a family history of thalassemia trait in both parents and has been receiving blood

transfusions every two weeks since birth. At age 20, she experienced an iron overload crisis that required hospitalization due to pleural effusion.

Regarding the surgery, initial monitoring included electrocardiography, pulse oximetry, and non-invasive blood pressure measurement. Venous access was established with a 16G intravenous catheter in the right upper limb and a 14G intravenous catheter in the left upper limb. After light sedation, the left radial artery was cannulated under local anesthesia using the Seldinger technique. Depth of anesthesia was monitored using a processed electroencephalogram index (CONOX™), and transesophageal echocardiography was employed throughout the procedure.

The anesthetic technique employed was balanced multimodal general anesthesia combined with interfascial plane blocks of the serratus anterior and pectoralis major muscles. The patient underwent biological mitral valve replacement via a minimally invasive technique involving a right anterolateral minithoracotomy. This approach involved two points of cardiac access: one through the right midclavicular line at the fourth or fifth intercostal space (periareolar) and the other through the anterior axillary line. After skin incision and dissection of the subcutaneous tissue, Surgisleeve™ retractors were placed, and pericardiotomy was performed under direct visualization to access the cardiac structures. Subsequently, cardiopulmonary bypass (CPB) was established with full heparinization, cannulating the right femoral artery and vein. A left atriotomy was then performed, followed by dissection and removal of the thickened mitral valve with restricted opening. A long-lasting biological mitral valve prosthesis (No. 31 – Medtronic – Hancock II) was implanted and secured to adjacent structures, followed by closure of the left atrium. The patient was weaned from CPB after a total duration of 102 minutes, with an aortic cross-clamp time of 78 minutes. Right thoracic drainage was performed using a Blake drain – 24Fr, and the procedure was completed with layered closure of the subcutaneous tissue and skin. Postoperative echocardiography demonstrated good function of the implanted prosthesis and normal left ventricular function.

After the completion of the surgery, the patient maintained stable hemodynamic and ventilatory parameters, allowing extubation in the operating room without adverse events. The patient was transferred to the intensive care unit (ICU) breathing spontaneously with supplemental oxygen via nasal cannula at 3 L/min and without the need for vasoactive drugs. The patient remained in the ICU for two days without complications related to the surgical procedure or anesthesia, and was subsequently transferred to a general ward. No blood transfusions were required during the hospital stay. The patient was discharged for outpatient follow-up after a total hospital stay of 3 days, without postoperative complications and with good clinical progress, continuing her usual medications for underlying conditions and with instructions on postoperative care.

DISCUSSION

Placental The existing body of literature contains a limited number of reports detailing anesthetic management strategies across various surgical procedures. Furthermore, the occurrence of complications and the need for perioperative blood transfusions are infrequently documented in research, thus representing a considerable challenge for anesthesiologists. Olivieri et al., in their study involving patients with β-thalassemia major (BTM), demonstrated that for an individual patient, a prolonged period during which ferritin levels remain below 2,500 ng/ml correlates with a lower likelihood of developing cardiac disease and a higher overall survival rate (90% at 15 years)[11]. Consequently, periodic assessment (every two to three months) of serum ferritin levels

is considered essential for the longitudinal monitoring of patients undergoing regular red blood cell transfusions.[11] The investigation by Angulo et al. emphasizes the importance of quantifying hepatic and myocardial iron deposition through magnetic resonance imaging, and correlating this methodological approach with serum ferritin levels to stratify the degree of iron overload, thus guiding subsequent therapeutic interventions based on these findings.[12]

In the present clinical case, the patient underwent a comprehensive panel of laboratory investigations, including measurement of ferritin levels, transthoracic and transesophageal echocardiography, and received specialized consultations from a nephrologist and a hematologist. Given the patient's multiple pre-existing comorbidities, a standardized anesthetic protocol was selected and implemented by the surgical team. The intraoperative phase proceeded uneventfully, with the patient requiring no blood transfusions and showing no clinical or laboratory evidence of hemolysis. Throughout the surgical procedure, meticulous attention was directed toward the prevention of hypoxia, along with vigilant monitoring for potential cardiac complications in the postoperative period. Recent findings reported by Borgna-Pignatti et al. indicated improved survival outcomes in Italian patients with β-thalassemia major, potentially attributable to more effective assessments of iron overload, including myocardial evaluation using T2* magnetic resonance imaging, and the use of oral iron chelators, particularly combination therapy involving deferoxamine and deferiprone. This therapeutic regimen appears to offer greater cardioprotection, resulting in a lower incidence of cardiac complications and reduced mortality.[13]

According to Baş and Özlü, the administration of general anesthesia and the physiological stress associated with surgical trauma may increase the risk of complications due to alterations in core body temperature, blood pH, oxygen tension, and intravascular fluid volume. Consequently, diligent perioperative care and postoperative monitoring are crucial to prevent hypoxemia, hypovolemia, hypothermia, acidosis, and the need for increased blood transfusions, particularly considering the propensity for erythrocyte sickling in this patient population.[14]

CONCLUSION

Patients with β-thalassemia major requiring surgical intervention demand meticulous and individualized care, beginning with a comprehensive pre-anesthetic evaluation and extending through to patient discharge. Tailoring the management strategy to address comorbidities beyond thalassemia is essential for the safe and successful conduct of any surgical procedure.

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SCIENTIFIC ARTICLE - CASE REPORT

FATAL ACUTE SUBDURAL HEMATOMA AFTER SPINAL ANESTHESIA - CASE REPORT

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ABSTRACT

Post-spinal anesthesia headache is a complication with a prevalence that varies between 1/500,000 and 1/1,000,000 and its occurrence points to a pressure gradient between the intracranial subarachnoid and spinal spaces due to the presence of a dural injury caused by the puncture. The persistence of the barometric gradient, however, can determine greater separation of the cerebral surface from the dura mater, with rupture of the bridging veins and formation of acute subdural hematoma (aSDH), a condition associated with high morbidity and mortality. aSDH after spinal anesthesia is a rare and potentially fatal complication. In the literature, it is observed that the majority of aSDHs after dural puncture did not result in exuberant mass effects or major clinical changes, meaning that urgent surgical treatment was not necessary, with the institution of clinical treatment for subsequent surgical surgery in a hematoma already showing signs of urgency chronification. In this reported case, it is evident that a patient had a head CT upon admission within the normal age range, however, she developed neurological changes and a new imaging examination showed a large aSDH with great clinical repercussions, and in the evaluation by neurosurgery, she presented signs of brain death without the possibility of neurosurgical treatment due to the impossibility of reversing the condition or providing any benefit to the patient.

Keywords: Brain death, Post-spinal anesthesia headache, Acute subdural hematoma, Headache disorders, Secondary, Brain edema.

INTRODUCTION

Post-spinal anesthesia headache is a complication with a prevalence that varies between 1/500,000 and 1/1,000,0001 and its occurrence points to a pressure gradient between the intracranial subarachnoid and spinal spaces caused by the puncture. The persistence of the barometric gradient, however, can determine greater separation of the cerebral surface from the dura mater, with rupture of the bridging veins and formation of acute subdural hematoma (aSDH) between the dura

mater and the subarachnoid space[2], a condition associated with high morbidity and mortality. The following are risk factors for the occurrence of hematoma after lumbar puncture: excessive drainage of cerebrospinal fluid (>250 ml); use of a traumatic needle or needle of inadequate diameter; cerebral atrophy and use of anticoagulants. In elderly patients, the subdural space is wider due to senescent cerebral atrophy, which results in a greater propensity to hemorrhage due to rupture of the bridging veins. aSDH mimics the occurrence of post-dural puncture headache, except for the fact that it presents with non-postural pain.[3] It is therefore considered an important differential diagnosis in the presence of unfavorable neurological outcomes in patients undergoing spinal anesthesia. We describe the case of a young patient who underwent orthopedic surgery on the lower limb under spinal anesthesia, who developed spontaneous aSDH. This is a rare complication, but avoidable with an adequate syndromic diagnosis. The literature on this topic is scarce and, to date, few cases have been reported with a fatal outcome.

CASE REPORT

In Female patient, 44 years old, previously healthy, was admitted to Goiás Emergency Hospital (HUGO) in July 2023, at night, with a history of multiple trauma due to a motorcycle accident (collision with a stopped car) 30 minutes ago. Reports she falling to the right side. Denies ejection or loss of helmet at the time. She only reports pain in the upper and lower limbs on the right. She denied the use of continuous medications, illicit substances and also did not use anticoagulants or antiplatelet agents and had no history of related comorbidities. On physical examination, a score of 15 on the Glasgow Coma Scale (ECG), isochoric and photoreactive pupils and no apparent neurological deficits. She presented a 10 cm blunt injury on the anterolateral surface of the right foot with tendon exposure, with limitations in dorsiflexion movement and immobilization of the right upper limb. She underwent complementary exams, being diagnosed with a fracture of the right proximal humerus and a distal fracture of the ipsilateral exposed tibia, being approached by the orthopedics team with successful. The procedure was performed using spinal anesthesia with bupivacaine with a 22Gx3 spinal needle at the level of L3-L4 without complications. The following day, the patient began to experience symptoms of postural headache and episodes of vomiting, with a normal physical examination, and was referred for a head tomography (figure 1), which showed no changes. The patient progressed the following day with worsening nausea and vomiting, as well as a non-postural holocranial headache, and presented with re-entrant seizures without improvement with anti-seizure drugs, requiring the provision of a definitive airway, mechanical ventilation and sedoanalgesia. New investigation exams were requested, including computed tomography angiography (CTA) of the skull with contrast, with evidence of aSDH, left fronto- temporo-parietal with a deviation of approximately 1.41 cm from the midline and cerebral edema more pronounced on the left (figure 2). During the inspection of the scalp, no signs of local hematoma were seen resulting from a fall from a bed, for example. On neurological examination, she presented bilateral fixed mydriasis and absence of brainstem reflexes, such as: oculocephalic, corneal-palpebral and cough reflexes. Sedation is discontinued for subsequent neurological assessment and laboratory tests are requested. She had a hydroelectrolyte disorder with kalaemia of 3.1 mmol/L, natremia of 159 mmol/L, without acid-base disorder with arterial pH within normal limits, however, with arterial lactate of 17.5 mg/dL. She was admitted to an intensive care unit for correction of metabolic changes and neurological surveillance. Comatose patient without sedation for 18 hours, absence of brainstem reflexes during clinical

consultations, developed hemodynamic instability requiring vasoactive amine (norepinephrine) and invasive blood pressure monitoring. The brain death protocol was initiated at 10 pm on 11/07, fulfilling clinical criteria: core temperature 36.1 ° C, blood pressure 130 x 90 mmHg, SatO2 99%, PaCo2 37 mmHg. She followed with hemodynamic stress in the following days, with norepinephrine at 2.8 mcg/kg/min and vasopressin at 0.04 IU/min, on mechanical ventilation in volume-control mode with FiO2 100%, tidal volume of 380 ml and PEEP 7 , presenting arterial pH with metabolic acidosis and hypokalemia. The patient was compensated from a hydroelectrolytic and acid-base point of view, and an apnea test was performed uneventfully. However, in the evaluation with daily transcranial Doppler, showed residual arterial blood flow. Nine days after admission, brain death was confirmed with transcranial Doppler confirming the absence of cerebral blood perfusion and family members were informed about the death, who chose not to donate the organs.

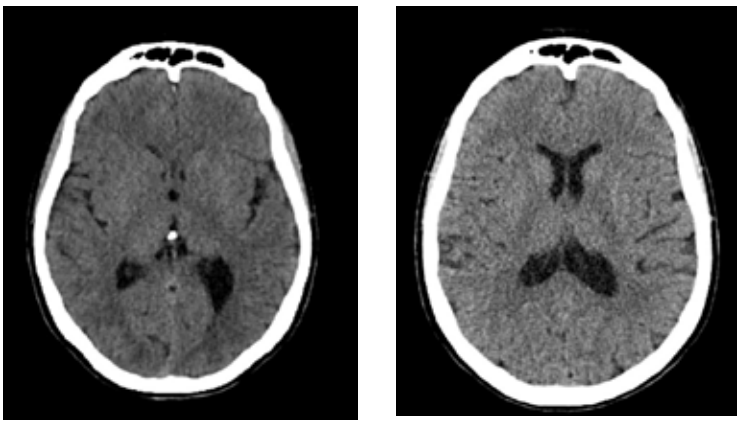

Figure 1. Cranial tomography on admission (07/07/2023) and on 08/07/2023 without changes.

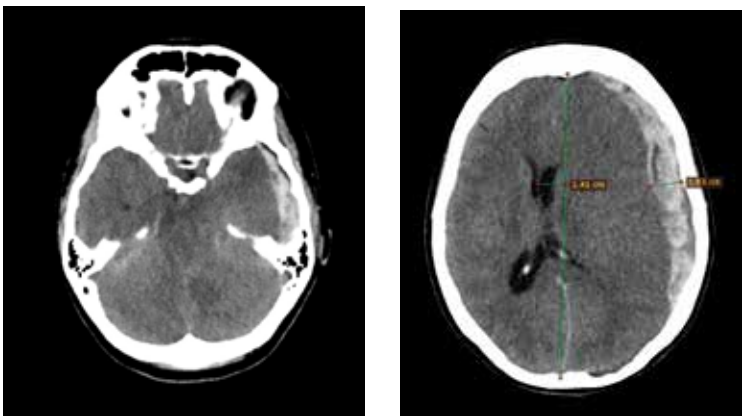

Figure 2. Head CT on the second day after admission showing acute left fronto-temporo-parietal subdural hematoma with midline shift of 1.41 cm, diffuse cerebral edema with compression of the cisterns and effacement of cerebral grooves and fissures.

DISCUSSION

The Intracranial subdural hematoma post-spinal anesthesia is a complication considered rare and potentially fatal after a dural puncture procedure, thus, there are not many cases reported in the medical literature.[4] Therefore, this article aims to contribute with an unusual and fatal report, being useful to highlight the importance of clinical suspicion and early diagnosis of this complication to which patients are subject to avoid possible catastrophic outcomes.[5]

In most of the articles analyzed, it was observed that the majority of acute subdural hematomas after spinal anesthesia did not result in exuberant mass effects and little deviation from the midline, therefore, with symptoms that were not significant from a neurological point of view, such as: headache, nausea , vomiting, dizziness, diplopia, not requiring urgent surgical treatment, clinical treatment and neurological surveillance for subsequent surgical drainage of an already chronic hematoma were instituted. In this reported case, we observe a young patient, victim of polytrauma with mild cranial trauma with head tomography on admission with no evidence of changes, but she developed a convulsive status, fixed mydriasis with a new head CT indicating aSDH with large deviation from the midline and erasure of cerebral grooves and fissures. A situation in which urgent decompressive craniectomy would be the only treatment, however, the patient already presented with an absence of brainstem reflexes, progressing to brain death 6 days later. This demonstrates the severity of this complication, which can lead to clear signs of intracranial hypertension and the need for urgent surgery.

The aspect of temporality draws attention, as in most cases patients presented aSDH 48 hours[6] after spinal anesthesia and in other cases there was no well-defined pattern between the onset of symptoms and the identification of bleeding. Furthermore, the importance of early diagnosis of postural headache and initiation of appropriate treatment is highlighted, from rest, intravenous hydration and analgesia to blood patch treatment to treat symptoms of intracranial hypotension.[7] A change in headache patterns to non-postural, without improvement with the supine position, associated with intense nausea and vomiting, change in the level of consciousness, visual blurring, contralateral hemiparesis and anisocoria[2,3,5], constitute alarm signals that require assessment and imaging quickly to exclude intracranial pathologies. On head CT, the aSDH appears as a hyperdense crescent-shaped lamina across the cerebral convexity, not respecting the limits of the cranial sutures.

From a pathophysiological point of view, one of the most accepted theories is based on the reduction in intracranial pressure caused by dural puncture associated with hypovolemia of cerebral blood flow, which leads to distension and consequent rupture of the bridging veins located in the subdural space.[8] In a previously healthy patient like the one in the case presented and without neurological changes, with a head CT examination on admission within normal limits, post-spinal anesthesia headache must be suspected as the most common change or even more serious cases, such as pathologies intracranial. From this pathophysiological concept, it is understood that the catastrophic evolution presented by this patient is precisely due to the changes in cerebral hydrodynamics caused by spinal anesthesia, which in a certain way affected the mechanisms for regulating intracranial pressure and cerebral blood flow, culminating in the formation of an acute hematoma in the subdural space quickly and aggressively, resulting in significant mass effect and compression of vital structures, such as the brain stem.

The treatment of aSDH can range from conservative treatment to surgical drainage of the hematoma through decompressive craniectomy, which is indicated in cases of unilateral or bilateral cerebral edema, more specifically when it falls into the Marhsall III or IV classifications.[9]

CONCLUSION

Placental Post-spinal anesthesia headache is a common complication, with incidence rates that can reach 70% of cases, however, the occurrence of aSDH is a rare and potentially fatal outcome that involves early diagnosis and treatment for greater patient survival.

Most of the cases reported were pregnant patients undergoing spinal anesthesia for obstetric procedures, with aSDH occurring in other types of surgery being much less common, as in the case described above in a patient with orthopedic pathology. Also noteworthy, is the evolution of the clinical condition to death, which was not observed in most studies published in the literature. Therefore, this report serves as an alert to differentiate cases of post-dural puncture headache with a benign course from other cases with severe neurological symptoms due to spontaneous aSDH. New studies are needed to identify the incidence of this complication, risk factors and the best therapeutic approach.

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PULMONARY LOBECTOMY AND THE POTENTIAL OCCURRENCE OF POSTOPERATIVE DYSAUTONOMIA

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ABSTRACT

Despite advances, pulmonary lobectomy can lead to postoperative dysautonomia, a dysfunction of the autonomic nervous system. This occurs due to possible vagus nerve injury during surgery, causing chronic cough or gastrointestinal issues. Additionally, surgical stress alters autonomic activity, manifesting as hemodynamic instability and orthostatic intolerance, which may include Postural Orthostatic Tachycardia Syndrome (POTS). Further symptoms include fatigue and cognitive problems. Although not common, early recognition of these signs is vital for proper patient management and to improve their quality of life.

Keywords: Lobectomy, Dysautonomia, Vagus nerve, Postoperative period, Postural orthostatic tachycardia syndrome.

INTRODUCTION

Pulmonary lobectomy is a surgical procedure widely used in the treatment of various pulmonary conditions, notably lung cancer. Despite advances in surgical techniques, such as video-assisted thoracoscopic surgery, the procedure is not without complications.[1] The autonomic nervous system (ANS), which controls involuntary bodily functions such as heart rate, blood pressure, digestion, and respiration, may be susceptible to dysfunction due to surgical events.[2] Dysautonomia, characterized by an imbalance in ANS regulation, emerges as a potential—though less explored—complication in the postoperative context of pulmonary lobectomy.

A narrative literature review was conducted using the PubMed database to identify articles published from 2015 onward. The search was performed using a combination of the following descriptors: Lobectomy, Dysautonomia, Vagus Nerve, Postoperative Period, and Postural Orthostatic Tachycardia Syndrome. The objective was to compile and synthesize relevant information regarding the relationship between lobectomy, autonomic dysfunctions involving the vagus nerve in the postoperative period, and the occurrence of Postural Orthostatic Tachycardia Syndrome.

Potential Mechanisms of Dysautonomia Following Pulmonary Lobectomy

The occurrence of dysautonomia after pulmonary lobectomy can be explained by multiple interconnected mechanisms:

Vagus Nerve Injury

The vagus nerve (cranial nerve X) is the main component of the parasympathetic nervous system, with extensive thoracic branches that innervate vital organs, including the lungs and heart.[3] Figure 1 illustrates the course of the vagus nerve in the thorax.[4] During lobectomy, the dissection and manipulation of anatomical structures adjacent to the vagus nerve and its branches—such as the bronchi, pulmonary vessels, and mediastinal lymph nodes—may result in direct injury, stretching, or neural disruption.[5]

The vagus nerve (cranial nerve X) emerges as the principal component of the parasympathetic nervous system, exhibiting extensive thoracic branches that innervate vital organs, including the lungs and heart.[3] This widespread innervation plays a crucial role in regulating essential autonomic functions such as heart rate, bronchoconstriction, and gastrointestinal motility. As illustrated in Figure 1, the course of the vagus nerve through the thorax is complex and intricate, with its detailed anatomy and multiple branching points well established in the medical literature.[4] A precise understanding of its topography is essential for thoracic surgeons, given its vulnerability during surgical interventions.

In thoracic surgical procedures such as lobectomy, the anatomical proximity of the vagus nerve and its branches to essential structures—notably the bronchi, pulmonary vessels, and mediastinal lymph nodes—makes it particularly susceptible to iatrogenic injury. The meticulous dissection and manipulation required in these regions may inadvertently result in various types of vagal trauma, ranging from excessive stretching to complete neural disruption.[5] Such injuries can lead to a range of postoperative complications, including hoarseness, dysphagia, bradycardia, or gastrointestinal motility disorders, all of which directly affect patient recovery and quality of life. Therefore, identification and preservation of the vagus nerve are considered critical priorities to reduce the risk of morbidity and optimize surgical outcomes in thoracic procedures.

Figure 1. Path of the Vagus Nerve. The image shows the path of the vagus nerve (and a section of the facial nerve)[4]

Clinical Implications: Vagal injury can disrupt autonomic homeostasis, clinically manifesting as chronic cough due to its role in the cough reflex arc.[5] Additionally, gastrointestinal dysfunctions—such as gastroparesis (delayed gastric emptying), constipation, or diarrhea—may occur, given the vagus nerve's influence on digestive tract motility.[6]

Alterations in Sympathetic and Parasympathetic Activity

Surgical stress, the systemic inflammatory response, and postoperative pain are factors that can induce significant modulations in autonomic nervous system (ANS) activity. Major surgeries, including thoracic procedures, have been shown to precipitate postoperative autonomic dysfunction.[7] This dysfunction may present with the following characteristics:

• Hemodynamic Instability: Fluctuations in blood pressure (hypotension or hypertension) and cardiac arrhythmias (tachycardia or bradycardia) are manifestations of autonomic dysregulation.[7]

• Orthostatic Intolerance: The inability to maintain blood pressure homeostasis upon transitioning to an upright position can lead to dizziness, vertigo, or syncope. Postural Orthostatic Tachycardia Syndrome (POTS), a form of dysautonomia frequently triggered by surgical events, exemplifies this mechanism.[8]

• Fatigue and Cognitive Dysfunction: Persistent fatigue, disproportionate to physical effort, and cognitive deficits such as difficulty concentrating and "brain fog" may reflect both autonomic and cerebral dysfunction.[1]

• Sudomotor and Thermoregulatory Dysfunctions: Alterations in sweating (hypo- or hyperhidrosis) and in the regulation of body temperature are indicative of autonomic dysregulation.[2]

• Sleep Disturbances: Postoperative disruption of sleep patterns may also be associated with autonomic dysfunction.[1]

Inflammatory Response and Oxidative Stress

Surgical trauma and the subsequent inflammatory response can release mediators that directly affect the integrity or function of autonomic nerves. Additionally, postoperative oxidative stress may contribute to neural injury.[7]

Clinical Manifestations of Dysautonomia After Pulmonary Lobectomy

The symptoms of dysautonomia are diverse and may be nonspecific, making diagnosis challenging. In the postoperative context of pulmonary lobectomy, manifestations may include:

- Dizziness, vertigo, presyncope, or syncope, particularly with postural changes.[8]
- Palpitations or tachycardia at rest or with minimal exertion.[8]
- Severe fatigue and exercise intolerance.[1]
- Gastrointestinal symptoms such as nausea, vomiting, constipation, or diarrhea.[6]
- Unexplained dyspnea at rest.[1]
- Difficulty concentrating, memory problems, or "brain fog".[1]
- Abnormalities in thermoregulation and sweating patterns.[2]
- Persistent chronic cough.[5]

Diagnosis of Dysautonomia / POTS

The diagnosis of dysautonomia, and specifically of POTS, is challenging due to the wide

variety of symptoms and the absence of a single definitive diagnostic test. It requires a comprehensive approach that includes a detailed clinical history, physical examination, and specific tests to assess autonomic function.[9] The diagnostic criteria for POTS include chronic symptoms of orthostatic intolerance—such as dizziness, presyncope, palpitations, weakness, fatigue, nausea, and "brain fog"—that worsen upon standing and improve when lying down.[9] Additional criteria include increases in heart rate: a sustained increase of at least 30 beats per minute (bpm) or a heart rate of 120 bpm or more within the first 10 minutes of transitioning from a supine to a standing position (in adults). In adolescents, the increase must be at least 40 bpm[9]. There must be an absence of classical orthostatic hypotension, defined as a drop in systolic blood pressure not exceeding 20 mmHg and in diastolic pressure not exceeding 10 mmHg within the first three minutes of standing. The presence of significant orthostatic hypotension suggests other forms of dysautonomia.[9]

Care should also focus on ruling out other conditions that may cause similar symptoms, such as dehydration, anemia, thyroid disorders, pheochromocytoma, structural heart disease, or primary arrhythmias.[9]

Common Diagnostic Tests

• Tilt Table Test: This is the primary test for diagnosing POTS. The patient is placed on a table that is then tilted to an upright position (typically 70 degrees), while heart rate and blood pressure are continuously monitored. A sustained increase in heart rate that meets diagnostic criteria, without significant hypotension, confirms the diagnosis of POTS.[9]

• Quantitative Autonomic Function Tests: These may include deep breathing tests (to assess parasympathetic function), the Valsalva maneuver (to assess both sympathetic and parasympathetic function), quantitative sudomotor axon reflex test (QSART), and pupillary function tests, all of which help identify specific patterns of autonomic dysfunction.[10, 11]

• Ambulatory Blood Pressure Monitoring (ABPM) and 24-Hour Holter Monitoring: These can be useful for detecting blood pressure patterns and arrhythmias not evident during routine office visits.

Potential Non-Pharmacological Treatments for Dysautonomia
POTS: Autonomic Rehabilitation

The treatment of dysautonomia and POTS is multifaceted and aims to relieve symptoms, improve quality of life, and, when possible, address the underlying cause. Increasing emphasis has been placed on autonomic rehabilitation, which integrates non-pharmacological strategies with progressive exercise programs.[12]

Non-Pharmacological Measures and Lifestyle Modifications

• Increased Fluid and Salt Intake: Helps increase blood volume, which can mitigate orthostatic hypotension and tachycardia. It is recommended to consume 2–3 liters of fluids per day and 10–12 grams of salt, if there are no contraindications.[9]

• Compression Stockings: May help reduce blood pooling in the legs and abdomen, improving venous return and decreasing tachycardia when standing.[9]

• Head-of-Bed Elevation: Sleeping with the head of the bed elevated by 10 cm can reduce nighttime diuresis and help adjust morning plasma volume.

• Smaller, More Frequent Meals: Avoid large meals rich in carbohydrates, which can divert blood flow to the gastrointestinal tract and exacerbate orthostatic symptoms.

• Avoiding Triggers: Identify and avoid factors that worsen symptoms, such as excessive heat, dehydration, alcohol, and prolonged standing.[12]

Autonomic Rehabilitation and Physical Exercise

Autonomic rehabilitation is a crucial component of treatment, especially for POTS, aiming to improve functional capacity and exercise tolerance. Adapted and progressive physical exercise can enhance cardiovascular conditioning and autonomic regulation.[12]

• Starting with Supine or Seated Exercises: For patients with severe orthostatic intolerance, exercises should begin in positions that minimize the effects of gravity, such as using a recumbent bike, swimming, or rowing.[12]

• Gradual Progression: The program should slowly increase in intensity and duration, with a gradual transition to upright exercises such as walking. Supervision by a physical therapist experienced in dysautonomia is essential to ensure safety and effectiveness.[13]

• Strength Training: Including muscle-strengthening exercises, especially for the legs and core, can support venous return and help stabilize blood pressure.[13]

• Heart Rate Monitoring: Patients should be advised to monitor their heart rate and symptoms to avoid overexertion, which can worsen symptoms and lead to demotivation.

Pharmacological Treatment

Medications may be used to control specific symptoms that do not respond to non-pharmacological measures.[9] Options include:

• Fludrocortisone: A mineralocorticoid that increases blood volume.

• Midodrine: An alpha-1 agonist that causes vasoconstriction and raises blood pressure.

• Low-dose beta-blockers (e.g., propranolol): May reduce tachycardia, especially in patients with sympathetic hyperactivity.

• Ivabradine: A medication that reduces heart rate without affecting blood pressure, useful for refractory tachycardia.

• Pyridostigmine: An acetylcholinesterase inhibitor that may enhance cholinergic transmission and alleviate some dysautonomic symptoms.

The choice of treatment should be individualized, taking into account the severity of symptoms, the presence of comorbidities, and the patient's response.

CONCLUSION

Although dysautonomia is not classified as a common complication in most patients undergoing pulmonary lobectomy, its occurrence is plausible and clinically relevant. The complexity of thoracic anatomy and the surgical manipulation near vital neural structures justify attention to this possibility. Early recognition of autonomic symptoms is crucial for proper management and for improving patients' postoperative quality of life. A thorough investigation is recommended in patients who

develop symptoms consistent with dysautonomia after pulmonary lobectomy, aiming for an accurate diagnosis and the implementation of effective therapeutic strategies.

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SCIENTIFIC ARTICLE - CASE REPORT

ANESTHETIC MANAGEMENT IN A PATIENT WITH SUSAC SYNDROME: A CASE REPORT

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ABSTRACT

Susac's Syndrome (SS) is defined as an occlusive microangiopathy of autoimmune etiology, characterized by the involvement of arterioles in the cerebral, retinal, and cochlear vascular beds. The classic clinical triad manifests as encephalopathy, visual deficits, and hearing loss. Given its relative rarity and variable clinical presentation, SS frequently poses a diagnostic challenge, resulting in underdiagnosis or misdiagnosis. Currently, the predominant therapy for SS involves immunomodulation with corticotherapy. This case report describes the clinical course of a 36-year-old female patient who underwent inhalational general anesthesia for elective laparoscopic cholecystectomy. Although invasive intracranial pressure monitoring was not implemented, the anesthetic management proved effective for the patient in question, highlighting the importance of a detailed clinical and neurological evaluation in the perioperative period, even in procedures considered to be of low neurological risk.

Keywords: Anesthesia general, Anesthesia intravenous, Susac syndrome, Cholecystectomy laparoscopic, Primary immunodeficiency diseases, Anesthesia.

INTRODUCTION

Thalassemias Susac Syndrome (SS) is defined as an autoimmune endotheliopathy characterized by a clinical triad of encephalopathy (with or without focal neurological signs), retinal artery occlusions, and hearing loss[1]. The pathogenesis of SS involves a microangiopathy affecting the precapillary arterioles of the retina, inner ear, and brain parenchyma.[2]

This disease predominantly affects young adults, with an average age of symptom onset around 18 years and a female-to-male ratio of 3.5:1.[3] SS can present with a wide spectrum of symptoms, including headache, mental confusion, personality and behavioral changes, ataxia, loss of balance, and dysarthria. In some cases, isolated vision loss or hearing loss may be the initial symptom.[4]

Due to its relative rarity and heterogeneous clinical presentation, the syndrome is often underdiagnosed or misdiagnosed, usually being identified only after the exclusion of other neurological, psychiatric, ophthalmological, and auditory conditions.[5] Characteristic findings on magnetic resonance imaging and audiometry contribute significantly to establishing the diagnosis.[4]

Current therapeutic recommendations for SS are based on expert-developed guidelines,

longitudinal follow-up of various patient cohorts, and recommendations extrapolated from other severe autoimmune diseases with similar immunopathogenesis. In this context, immunosuppression constitutes the cornerstone of treatment, with therapy intensity being adjusted primarily according to the severity of central nervous system involvement.[6]

Due to its rarity and the diagnostic challenge it still presents, there is a lack of studies in the medical literature addressing the management of this disease in the context of anesthesiology. In light of this, the present scientific article aims to report the anesthetic management of a patient with SS who underwent general anesthesia.

CASE REPORT

A 36-year-old female patient with a diagnosis of SS underwent elective videolaparoscopic cholecystectomy. On pre-anesthetic clinical evaluation, she presented with hearing impairment, did not respond to verbal commands, was hyperactive, and minimally cooperative. Additionally, she was overweight and classified as Mallampati class III.

Anesthetic induction was performed with 250 micrograms (mcg) of fentanyl, 150 milligrams (mg) of propofol, and 80 mg of succinylcholine administered intravenously, followed by direct laryngoscopy, which revealed a Cormack-Lehane grade 2A view. Orotracheal intubation was performed using a 7.0 mm cuffed endotracheal tube. Additional neuromuscular relaxation was achieved with 30 mg of rocuronium. General inhalational anesthesia was maintained with 2% sevoflurane. Additionally, adjuvant medications included 8 mg of ondansetron, 10 mg of dexamethasone, 2 grams of dipyrone, and 40 mg of parecoxib.

The surgical procedure proceeded without complications and lasted 60 minutes. Monitoring included pulse oximetry, cardiac monitoring, non-invasive blood pressure, and temperature measurement. Intracranial pressure was not assessed before, during, or after anesthesia.

At the end of the procedure, neuromuscular blockade was reversed with 200 mg of sugammadex; however, the patient experienced difficulty resuming spontaneous ventilation, resulting in carbon dioxide (CO_2) retention. Arterial blood gas analysis was consistent with severe respiratory acidosis. Consequently, the first extubation attempt failed, and mechanical ventilation had to be prolonged to correct the respiratory-origin acid–base imbalance.

Approximately 30 minutes after the first extubation attempt, the patient was successfully extubated without complications. Her recovery took place in the post-anesthesia care unit within the surgical center, where she remained for two hours before being transferred to the ward.

DISCUSSION

Patients with neurological manifestations of SS are predisposed to elevated intracranial pressure (ICP)[7]. In this context, the use of non-invasive techniques—such as ultrasonographic assessment of the optic nerve sheath diameter—has shown diagnostic accuracy for detecting increased ICP in the intraoperative setting. However, this method was not employed in the anesthetic management of the patient in the present case.

Furthermore, the choice of anesthetic technique should be based on the presence or absence of intracranial hypertension[1]. In this regard, it is recommended to minimize the patient's exposure to stimuli that could increase ICP, such as sympathetic stimulation. Accordingly, direct laryngoscopy was performed only under an adequate anesthetic plane and after the

administration of fentanyl at a dose of 4 to 5 mcg/kg.

Pneumoperitoneum is known to induce reflex vasodilation, which can lead to ICP[8]. To mitigate this effect, peritoneal insufflation was performed gradually rather than abruptly, maintaining intra-abdominal pressure below 12 mmHg. Although the use of lower insufflation pressures may be considered, the most effective strategy for minimizing the impact on ICP would be the use of abdominal wall lift systems for performing videolaparoscopy[9].

Another measure aimed at preventing increased ICP was the reduction of arterial partial pressure of carbon dioxide ($PaCO_2$) during the period the patient remained under controlled mechanical ventilation, through hyperventilation. The decrease in $PaCO_2$ induces alkalosis, and the resulting increase in blood pH has a direct effect on cerebral arterioles, promoting vasoconstriction, reducing intracranial blood volume, and consequently lowering ICP[10]. However, at the time of extubation, the patient presented with carbon dioxide retention secondary to apnea, progressing to severe respiratory acidosis. Thus, the effect of hyperventilation on ICP may not have been beneficial during extubation.

Regarding the selection of anesthetic agents in cases of intracranial hypertension, intravenous propofol, used in total intravenous anesthesia, is known to induce cerebral vasoconstriction and reduce ICP[11], compared to inhalational agents. On the other hand, in the absence of intracranial hypertension, the use of propofol may not be advisable, as it may exacerbate cerebral, retinal, and cochlear hypoperfusion in the context of preexisting microangiopathy in SS[1].

Other interventions for ICP control, such as the administration of hypertonic saline and diuretics, were not implemented during the anesthetic management of this case. However, elevation of the head of the bed while in the supine position was performed, as it also represents an appropriate surgical positioning for the proposed procedure.

In conclusion, the anesthetic management described in this case proved to be effective in the anesthetic care of a patient with SS undergoing elective videolaparoscopic cholecystectomy. Although invasive ICP monitoring was not performed, measures were implemented with the aim of preventing its elevation, as detailed above.

However, there is a significant lack of studies addressing anesthetic management in patients with this syndrome, highlighting the need for future research on the topic.

Other interventions for ICP control, such as the administration of hypertonic saline and diuretics, were not implemented during the anesthetic management of this case. However, head elevation in the supine position was performed, as it also represents an appropriate surgical positioning for the proposed procedure.

CONCLUSION

The anesthetic management described in this case proved to be effective in the anesthetic care of a patient with Susac's syndrome undergoing elective laparoscopic cholecystectomy. Although invasive ICP monitoring was not performed, measures were implemented to prevent its elevation, as detailed. Nevertheless, there is a significant lack of studies addressing anesthetic management in patients with this syndrome, highlighting the need for further research on the subject.

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SCIENTIFIC ARTICLE - NARRATIVE REVIEW

MAGNETIC RESONANCE IMAGING IN THE DIAGNOSIS OF ADENOMYOSIS: A NARRATIVE REVIEW

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ABSTRACT

Adenomyosis is a gynecological condition characterized by the ectopic presence of endometrial tissue within the uterine myometrium, and it can present in either focal or diffuse forms. This narrative review aims to evaluate the role of magnetic resonance imaging (MRI) in the diagnosis and clinical management of adenomyosis. Studies published between 2005 and 2025 were analyzed from databases including PubMed, Scopus, Google Scholar, and Lilacs, focusing on the application of MRI in identifying adenomyosis. MRI stands out as a non-invasive imaging modality with high sensitivity and specificity, surpassing transvaginal ultrasound (TVUS), particularly in inconclusive cases. The main diagnostic feature is a thickened junctional zone (>12 mm), although additional findings—such as small cysts, low-signal-intensity striations on T2-weighted images, and hemorrhagic foci on T1—are also relevant. MRI allows for differentiation between disease subtypes, including focal adenomyosis, diffuse adenomyosis, adenomyoma, and cystic adenomyosis, and provides comprehensive evaluation of adjacent pelvic structures. It also assists in ruling out differential diagnoses, such as leiomyomas and endometrial tumors. Despite its higher cost, MRI is recommended as a second-line imaging modality according to the 2017 guidelines of the European Society of Urogenital Radiology (ESUR), especially in the assessment of endometriosis and complex pelvic pathologies. In conclusion, MRI is an essential tool for accurate diagnosis and therapeutic planning in adenomyosis.

Keywords: Adenomyosis, Magnetic resonance imaging, Diagnostic imaging, Junctional zone, Gynecology.

INTRODUCTION

Adenomyosis is a condition that occurs in the body of the uterus, characterized by the presence of endometrial tissue, with ectopic endometrial glands and stroma within the myometrium. It can present in two forms: focal, in which the foci are located in a limited area of the myometrium, and diffuse, in which the foci are distributed throughout the myometrium. Magnetic resonance

imaging (MRI) is an excellent exam, more specific than ultrasonography, for detecting adenomyosis, as it highlights the zonal uterine anatomy, allowing visualization of the uterus's three layers: the endometrium, the junctional zone, and the myometrium. It is important to remember that the junctional zone is part of the myometrium, but due to being less hydrated, it appears hypointense on MRI. The assessment of uterine size and junctional zone thickening is essential for confirming the diagnosis of adenomyosis.[1,2]

METHODS

This narrative review aims to evaluate the role of magnetic resonance imaging (MRI) in the diagnosis and clinical management of adenomyosis. For the selection of studies, articles published between 2005 and 2025 in English, Portuguese, and Spanish were included, provided they discussed the application of MRI in the evaluation of adenomyosis. The literature search was conducted in the PubMed, Scopus, Google Scholar, and Lilacs databases, using keywords such as "adenomyosis," "magnetic resonance imaging," "diagnostic imaging," among other related terms. Only studies that directly addressed the use of MRI in the identification of adenomyosis were considered for this review, including clinical trials, systematic reviews, and guidelines. Articles that focused on alternative diagnostic methods, other gynecological conditions, or studies conducted in non-human populations were excluded.

Magnetic resonance imaging

First described in 1946, nuclear magnetic resonance is defined as the ability of the nuclei of certain chemical elements to emit radiofrequency signals when exposed to a strong magnetic field. These signals can be detected and transformed into images. Initially developed and used for the analysis of chemical and physical molecules, magnetic resonance imaging (MRI) gradually shifted from the scientific to the clinical context—especially after the 1980s. With excellent spatial resolution, high tissue differentiation capability, and the possibility of multiplanar and three-dimensional reconstructions, MRI has become one of the main non-invasive techniques for anatomical visualization and the diagnosis of various diseases.[3-5]

The particles responsible for the emitted radiofrequency signal are hydrogen protons and water molecules in the tissue. The signal and contrast are determined by differences in proton density and in the signal decay properties of different tissues.[5] The magnetically excited nucleus returns to its initial state by releasing energy in the form of electromagnetic waves. This process is called relaxation and is defined by two time constants: the energy release time or longitudinal relaxation time, T1, and the oscillation time of the protons during the process, or transverse relaxation time, T2. These constants are recorded as spatially localized signals by a coil installed in the MRI machine and are processed by computerized algorithms to produce anatomical images. Factors such as the individual characteristics of each molecule and tissue composition affect the generation of different proton relaxation times, and therefore, images can be appropriately weighted in T1 or T2, depending on the features of interest to be analyzed.[3]

Magnetic resonance imaging in the diagnosis of adenomyosis

In 1970, American physician Raymond Damadian conducted an experiment on rats and observed different responses to magnetic excitation between normal and tumorous tissues. These signals

varied in their contrast characteristics due to differences in tissue composition and relaxation times, allowing the identification of changes in the analyzed tissues. From this perspective, magnetic resonance imaging (MRI) stands out as an excellent diagnostic method for the involvement of organs and tissues by various diseases, including adenomyosis.[4,6] For decades, the diagnosis of adenomyosis relied exclusively on histopathological examination, which involves the identification of endometrial glands within the myometrium, at least 2.5 mm from the junctional zone, through the collection and analysis of myometrial tissue. Advances in gynecological techniques introduced imaging criteria into the process and facilitated diagnosis, making it less invasive. Currently, transvaginal ultrasonography (TVUS) and magnetic resonance imaging (MRI) are the main methods used for the diagnosis of adenomyosis. Due to its wide availability and relatively low cost, TVUS is the first-choice examination to identify adenomyosis. However, ultrasonography is operator-dependent, which may result in variability between different exams.[6] Thus, in the case of inconclusive ultrasound findings, MRI is the recommended imaging method. Unlike ultrasonography, MRI is less available, more expensive, and therefore less accessible, being considered a second-line examination in the investigation of conditions such as adenomyosis. On the other hand, MRI offers greater accuracy in detecting adenomyosis due to its higher sensitivity and specificity, and reduced operator dependency. Additionally, MRI demonstrates a high capacity for soft tissue differentiation and is capable of identifying different subtypes of adenomyosis. It also enables the evaluation of surrounding anatomical structures and the detection of other pathological conditions that may be present simultaneously in the pelvic region, such as fibroids and endometriosis.[6] The MRI protocol used for detecting adenomyotic disease may vary depending on the clinic or the type of equipment used. In 2017, the European Society of Urogenital Radiology (ESUR) developed a set of standard guidelines for performing MRI in the investigation of endometriosis, which should also be applied when analyzing adenomyosis. It is recommended that patients follow a low-fiber diet for two to three days prior to the exam, fast for two to three hours, and take an antiperistaltic agent immediately before the MRI. Patients should also be instructed not to urinate for at least one hour before the procedure, in order to maintain moderate bladder filling. This avoids detrusor muscle contraction due to a full bladder and prevents poor visualization of the ureters caused by an empty bladder.[6] The MRI imaging protocol recommended by ESUR should include high-resolution, fat-unsaturated T2-weighted sequences in sagittal and axial planes (or oblique planes, if necessary) and axial T1-weighted sequences with and without fat saturation. T1-weighted sequences with contrast and fat saturation are not required for diagnosis but are recommended in cases of atypical features seen on T2-weighted images. In this way, T1- and T2-weighted images complement each other, providing information that supports pathological definition.[6] The most frequently used characteristic in the diagnosis of adenomyosis is a junctional zone thickness greater than 12 mm. However, this thickening may occur physiologically due to hormonal changes during the menstrual cycle; therefore, the exam is preferably performed in the postmenstrual period. T2-weighted sequences are essential for the diagnosis of adenomyosis, as they highlight the anatomy of the junctional zone. Increased thickness may appear on T2 as a poorly defined area with low signal intensity, characterizing smooth muscle hyperplasia adjacent to ectopic endometrial tissue. T1-weighted imaging also contributes to diagnosis, as it reveals foci of high signal intensity representing the presence of methemoglobin, indicating hemorrhage — a highly specific predictive sign of adenomyotic disease.[2,7]

MRI findings in adenomyosis

On magnetic resonance imaging, adenomyosis presents as an irregular enlargement of the uterus and thickening of the junctional zone greater than 12 mm. When the junctional zone measures less than 8 mm, the possibility of adenomyosis can be excluded. It is important to note that recent literature has emphasized the need to include additional indirect criteria for diagnosing adenomyosis, rather than relying solely on junctional zone thickness, due to potential hormonal influences. The thickening may appear regular or irregular, homogeneous or heterogeneous, and may also present small cysts within it. Small hemorrhagic foci may be identified as hyperintense foci on fat-saturated T1-weighted images. In addition, changes in the pattern of the adjacent myometrium are common, appearing either diffusely or with striated features. These characteristics on MRI help confirm the diagnosis of adenomyosis (Figure 1).[8,9] To perform MRI for adenomyosis detection, it is recommended to use a primary high-resolution sagittal T2-weighted sequence with a high matrix, oriented along the longitudinal axis of the uterus. Additionally, a second T2-weighted sequence in the coronal plane, and a T1-weighted fat-saturated sequence to identify hemorrhagic foci, should be obtained—this last sequence can be performed in either the sagittal or coronal plane. It is important to note that motion artifacts are quite common and can cause visual "noise" in the exam. To minimize respiratory motion artifacts, a compression band may be applied to the anterior abdominal wall of the patient. To reduce artifacts caused by peristalsis, some healthcare services administer injectable antispasmodics, such as Buscopan. The use of contrast is not necessary for the detection of adenomyosis.[1,6] When investigating adenomyosis through MRI, it is crucial to consider that physiological factors, such as hormonal variations during the menstrual phase, can cause thickening of the junctional zone. Therefore, it is recommended to perform the exam during the late proliferative phase. Additionally, transient uterine contractions, which appear as hypointense bands, and conditions such as postmenopause (Figure 2) or hormonal contraception, in which the junctional layer may not be measurable, can also produce findings that do not reflect pathological changes. It is also essential to recognize that adenomyosis may appear on MRI as a pseudo-widening (Figure 3) of the endometrium, visualized as hyperintense linear striations on T2-weighted images radiating from the endometrium toward the myometrium, similar to myometrial invasion seen in carcinoma. This highlights the need for a detailed analysis of MRI results to confirm the diagnosis of adenomyosis.[1,2,10]

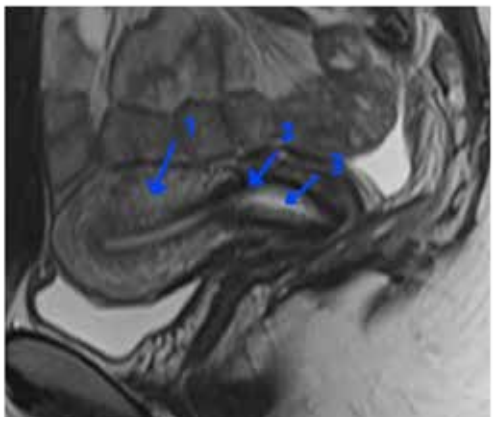

FIGURE 1. Sagittal T2-weighted image of a normal postpubertal uterus: (1) myometrium, (2) junctional zone, and (3) endometrium.[10]

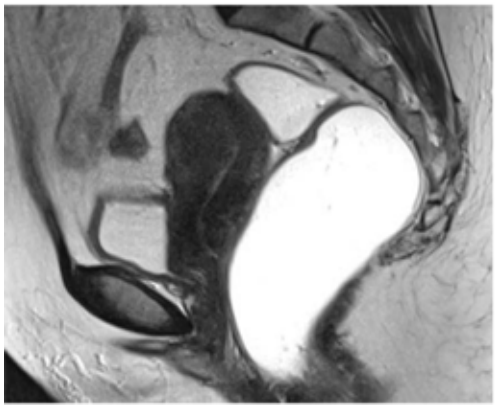

FIGURE 2. Postmenopausal uterus: sagittal T2-weighted images of a postmenopausal uterus in which the junctional zone is not measurable.[10]

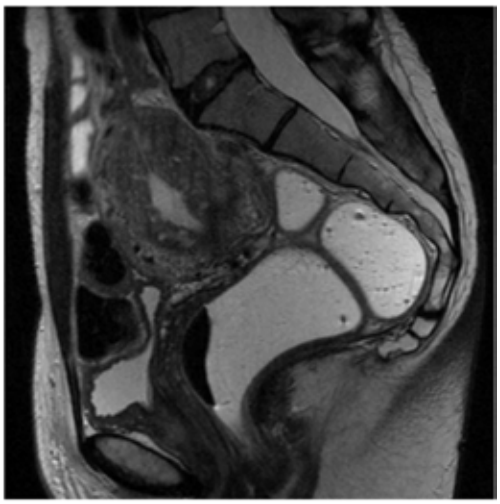

FIGURE 3. Pseudo-widening of the endometrium: sagittal T2-weighted images showing a thickened junctional zone with striated high-signal areas radiating from the endometrium toward the myometrium, resembling the appearance of endometrial carcinoma invasion.[10]

Focal adenomyosis

Focal adenomyosis (Figure 4) is characterized by small intramyometrial cysts, which may or may not be associated with edema of the junctional zone. These cysts can be distributed as single or multiple foci within the myometrium. It is important to note that, unlike diffuse adenomyosis, focal adenomyosis is generally not associated with significant changes in uterine thickness or myometrial texture. In many cases, focal adenomyosis is asymptomatic and may be diagnosed incidentally during imaging exams performed for other reasons. However, in other cases, the condition may be associated with symptoms such as pelvic pain and dysmenorrhea.[1,8]

Diffuse adenomyosis

Diffuse adenomyosis (Figure 5) is characterized by the presence of small diffuse cysts in the inner myometrium, along with thickening of the junctional zone (JZ). Both symmetric and asymmetric distributions can be observed in this type of adenomyosis. The distribution may be symmetric, affecting the anterior and posterior uterine walls equally, or asymmetric, when it predominantly affects only one of the walls.[2,6]

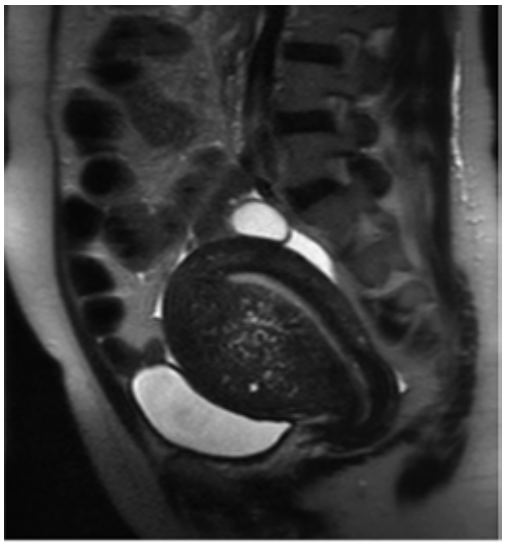

FIGURE 4. Focal adenomyosis. Sagittal T2-weighted FSE image (1100/123 repetition time/echo time). Focal diffuse thickening of the junctional zone along the anterior surface of the uterine body with associated pinpoint foci of increased T2 signal.

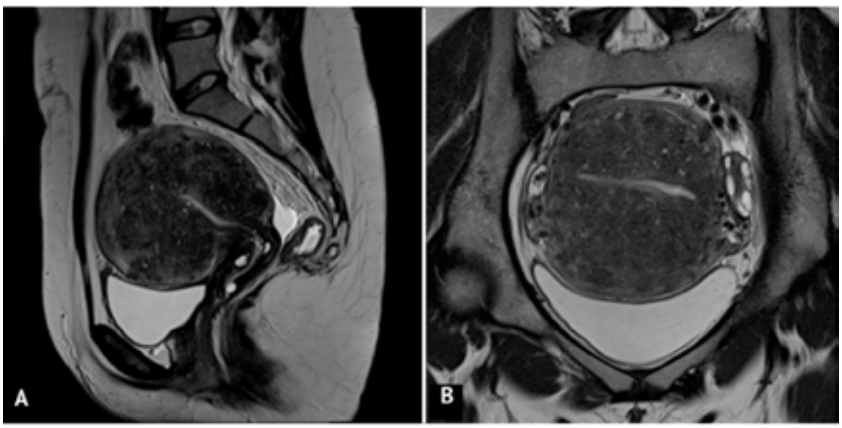

FIGURE 5. Diffuse adenomyosis: A. Sagittal and B. Coronal T2-weighted images showing thickening of the junctional zone forming a poorly defined low-signal area, with punctate foci of high intensity in the myometrium.[10]

UNCOMMON MRI FINDINGS IN ADENOMYOSIS

Adenomyoma and adenomyotic polyp

Adenomyoma (Figure 6) is a form of adenomyosis that consists of a confluence of adenomyotic glands resembling a mass. This type of adenomyosis may appear as an intramyometrial mass,

primarily located in the uterine body. In some cases, the adenomyoma may deform the endometrium, characterizing a submucosal adenomyoma. Another possibility is that it projects into the endometrial cavity, growing as a polypoid mass, thus forming a polypoid adenomyoma (Figure 7).[9]

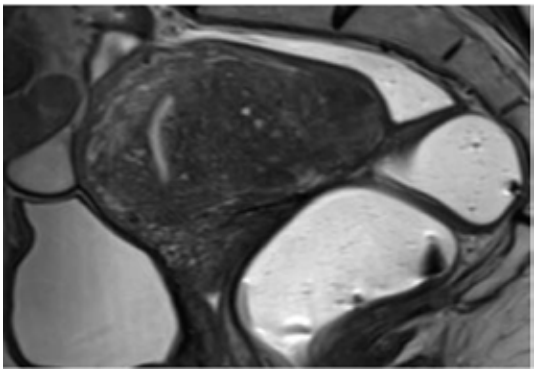

FIGURE 6. Adenomyoma on MRI: sagittal T2-weighted image showing a circumscribed hypointense intramyometrial mass with ill-defined margins, minimal mass effect, and foci of high signal intensity.[10]

Swiss cheese appearance

Diffuse adenomyosis may be identified on magnetic resonance imaging by a "Swiss cheese" appearance (Figure 8), characterized by myometrial cysts and nodules on contrast-enhanced and T2-weighted sequences. This appearance is caused by the dilation of endometrial glands within the myometrium. In addition, the junctional zone appears thickened and poorly defined, along with the presence of linear striations.[2,8]

Cystic adenomyosis

Cystic adenomyoma (Figure 9) is usually asymptomatic, but in some cases, it may cause pelvic pain and abnormal bleeding. Cystic adenomyoma is a lesion characterized by a large hemorrhagic cyst, resulting from extensive menstrual bleeding within ectopic endometrial tissue. This condition may be located within the myometrium, submucosal, or subserosal. On magnetic resonance imaging, the surrounding adenomyotic tissue can be identified by a high signal on T1 and low signal on T2. This finding is important for distinguishing cystic adenomyoma from other uterine masses, such as fibroids or carcinomas.[2]

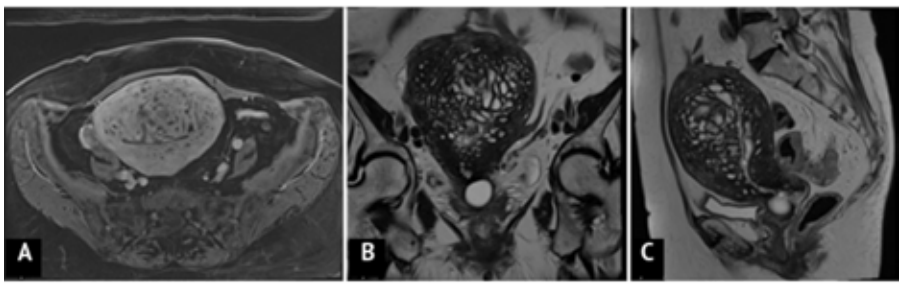

FIGURE 7. Swiss cheese appearance in adenomyosis: A. Axial T1 3D fat-saturated image, B. Coronal T2, and C. Sagittal T2-weighted images showing poor definition of the endometrial junctional zone with prominent glandular myometrial cysts, myometrial nodules, and linear striations.[10]

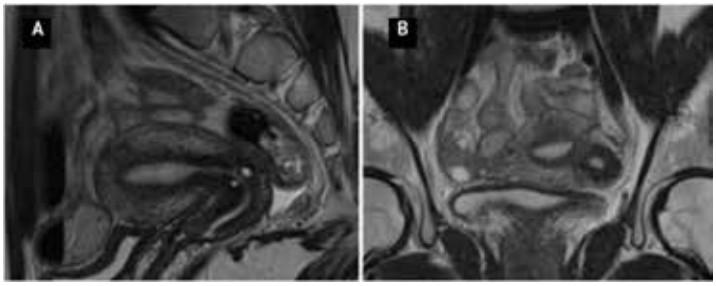

FIGURE 8. T2-weighted images: A. Sagittal and B. Coronal views showing a nodular uterine lesion with a centrally located high-signal cavity, alongside a normal uterus.[10]

DIFFERENTIAL DIAGNOSIS

Leiomyoma

Leiomyomas (Figure 10) are benign tumors composed of smooth muscle cells and are the most common type of tumor in the female genital tract. Adenomyomas and leiomyomas both appear as low-signal-intensity lesions on T2-weighted images, but they present differently on MRI. Adenomyomas typically appear as ill-defined lesions with minimal mass effect, whereas leiomyomas usually present as well-defined masses associated with prominent peripheral vessels. The differential diagnosis becomes even more challenging in cases of cystic or hemorrhagic degeneration of leiomyomas.[1,2,10]

Accessory cavitated uterine mass (ACUM)

ACUM (Figure 11) is a rare uterine anomaly that presents as a cystic or hemorrhagic mass that does not communicate with the uterine cavity and is located within the myometrial wall. The diagnosis of ACUM requires the presence of a T1 hyperintense cavitated mass, completely isolated from the endometrial cavity. The differential diagnosis should include several gynecological conditions, such as rudimentary or cavitated uterine horns, intramural cystic adenomyoma, and red degenerating leiomyomas. Diagnosis may be guided by clinical data, such as patient age and the presence of severe dysmenorrhea and chronic cyclic pain.[2,10,11]

Endometrial carcinoma with melf pattern

A differential diagnosis includes a fibromyxoid stromal reaction. This tumor is often associated with myometrial invasion, lymphovascular involvement, and lymph node metastasis, despite having a low histological grade. On magnetic resonance imaging, it appears as a hypointense thickening on T2-weighted images in the inner myometrium, with a small cystic component resembling adenomyosis. MRI is an important tool for assessing myometrial invasion and identifying pathologic lymph nodes.[6,9]

Effectiveness of magnetic resonance imaging in the diagnosis of adenomyosis

Currently, ultrasonography is used as the first-line imaging method for the initial diagnosis of adenomyosis. However, due to its high operator dependence, MRI is recommended in

inconclusive cases because of its high sensitivity and specificity and low operator dependence. Additionally, magnetic resonance imaging offers excellent soft tissue differentiation, allowing the identification of coexisting diseases, evaluation of structures adjacent to the pelvic region, and detection of adenomyosis subtypes.[6] Furthermore, the subendometrial myometrium, which is composed of densely packed smooth muscle cells, is clearly visualized on MRI as a low-signal-intensity zone, known as the junctional zone (JZ). In this context, MRI has been shown to be superior to transvaginal ultrasonography (TVUS) in the diagnosis of adenomyosis based on imaging studies. However, the combination of MRI and TVUS provides a high level of accuracy for ruling out adenomyosis. Measuring the difference in junctional zone thickness may further optimize the diagnosis of adenomyosis by MRI.[12] Another important factor to consider is the high cost of MRI, which limits its use as a first-line investigation. According to the 2017 guidelines of the European Society of Urogenital Radiology (ESUR), MRI should be considered a second-line imaging technique for female pelvic disease when TVUS results are inconclusive. In addition, MRI is recommended in cases of endometriotic disease due to its accurate preoperative staging.[13]

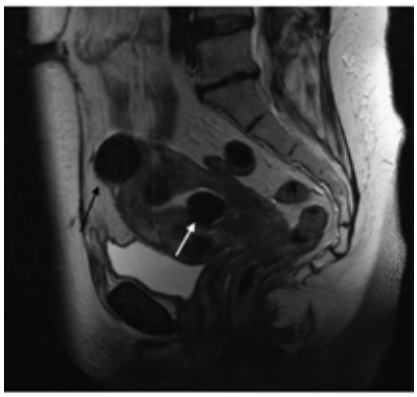

FIGURE 9. Subserosal and submucosal leiomyomas. Sagittal T2-weighted FSE image (1100/121 repetition time/ echo time). Well-circumscribed subserosal (black arrow) and submucosal (white arrow) leiomyomas, both showing low signal intensity.[1]

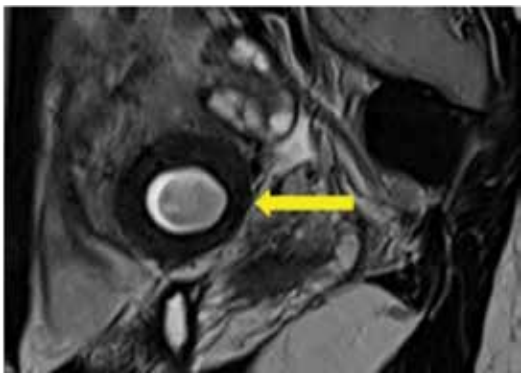

FIGURE 10. Sagittal magnetic resonance image showing a cavitated lesion on the left side of the uterus, with its wall formed by T2 hypointense myometrium (yellow arrow) and intermediate signal with "shading." There is no communication between this lesion and the normal endometrial cavity.[11]

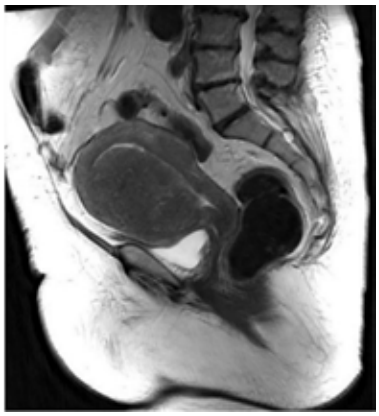

FIGURE 11. Endometrial carcinoma with MELF pattern. Sagittal T2-weighted image showing thickening of the inner anterior myometrium and a low-signal mass resembling adenomyosis, with small cystic components.

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SCIENTIFIC ARTICLE - INTEGRATIVE REVIEW

ULTRASONOGRAPHY IN THE DIAGNOSIS OF ADENOMYOSIS: AN INTEGRATIVE REVIEW

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ABSTRACT

Adenomyosis presents a diagnostic challenge in gynecological practice due to its nonspecific symptoms, such as dysmenorrhea and abnormal uterine bleeding, which overlap with various other uterine pathologies. For many years, definitive diagnosis was only possible through hysterectomy, limiting early detection and conservative treatment strategies. With advances in imaging techniques, there is a growing need to evaluate non-invasive, effective, and accessible diagnostic tools.

This integrative review explores the role of ultrasonography—particularly transvaginal ultrasound (TVUS)—as the primary screening and diagnostic modality for adenomyosis. TVUS demonstrates good sensitivity and specificity, being capable of identifying features such as a heterogeneous myometrium, myometrial cysts, hypoechoic striations, and alterations in the junctional zone (JZ). The use of three-dimensional TVUS improves visualization of the JZ, although it does not significantly enhance diagnostic accuracy compared to the two-dimensional mode.

The review also addresses the utility of color Doppler and elastography in differentiating adenomyosis from leiomyomas, as well as the potential of contrast-enhanced ultrasound, which remains under investigation. Despite its diagnostic efficacy, TVUS is operator-dependent, and in inconclusive cases, magnetic resonance imaging (MRI) should be used as a complementary method. In conclusion, this article emphasizes that ultrasonography, when properly performed, is a fundamental diagnostic tool for the early detection of adenomyosis, enabling less invasive and more personalized treatment approaches for patients.

Keywords: Adenomyosis, Diagnostic imaging, Review, Ultrasonography, Doppler ultrasonography.

INTRODUCTION

Adenomyosis is a gynecological condition characterized by the presence of endometrial glands and stroma within the myometrium, often associated with symptoms such as dysmenorrhea, abnormal uterine bleeding, and, in some cases, infertility. Historically, the diagnosis of adenomyosis

was made exclusively through histopathological examination following hysterectomy, which significantly limited the early clinical recognition of the disease.[1]

With advances in imaging technologies—especially ultrasonography—it has become possible to identify suggestive features of adenomyosis in a non-invasive manner. Pelvic ultrasonography has become the first-line imaging exam when clinical signs point to the disease.[1,2] In this context, it is essential to understand the role of ultrasonography in its different modalities, its characteristic findings, limitations, and the recent advances that contribute to a more accurate diagnosis.

OBJECTIVE

To analyze, through an integrative review, the role of ultrasonography in the diagnosis of adenomyosis, considering the available imaging modalities, the main ultrasound findings, the clinical impact, and the challenges in differentiating it from other gynecological pathologies.

METHODOLOGY

This integrative review aimed to analyze the role of ultrasonography in the diagnosis of adenomyosis, with an emphasis on the different modalities used, the main ultrasound findings, and the clinical applicability of the technique. Studies published between 2005 and 2025 were considered, in Portuguese, English, and Spanish, that addressed ultrasonography as an imaging method in the evaluation of adenomyosis.

The search was conducted in the PubMed, Scopus, Google Scholar, and LILACS databases using the following descriptors combined with Boolean operators: "adenomyosis," "transvaginal ultrasonography," "transabdominal ultrasonography," "diagnostic imaging," "three-dimensional ultrasonography," and "elastography."

Articles discussing the use of ultrasonography in the diagnosis, differentiation, and clinical monitoring of adenomyosis were included, encompassing clinical studies, systematic reviews, consensus statements, and guidelines. Studies addressing other gynecological pathologies without a direct relationship to adenomyosis, as well as experimental research in non-human populations, were excluded.

The analysis was conducted with a focus on the characteristics and limitations of transabdominal and transvaginal ultrasonography; the characteristic ultrasound signs of adenomyosis; the contributions of color Doppler and elastography in differentiating it from other uterine pathologies; and the potential advancements with three-dimensional and contrast-enhanced ultrasonography. The selection and interpretation of the studies were carried out qualitatively, prioritizing the clinical relevance of the findings and their applicability in the context of imaging diagnosis of adenomyosis.

RESULTS

Transabdominal Ultrasonography (TAUS)

TAUS has low sensitivity (around 30%) and high specificity (>95%) in the diagnosis of adenomyosis.[3,4] Although limited in resolution, it can be useful in cases of an enlarged uterus or when the vaginal route cannot be used[3]. Ultrasound findings include increased uterine volume, regular (possibly asymmetric) outer contour, myometrial heterogeneity, and intramyometrial cystic images.[2,4,5] However, TAUS does not allow differentiation between the different types of adenomyosis.

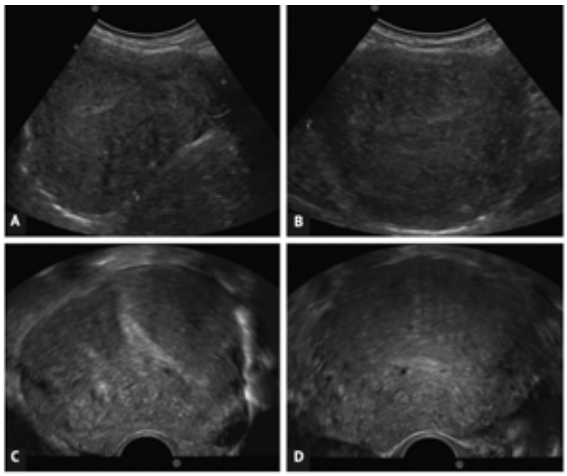

FIGURE 1. A and B. Comparison using TAUS, showing a globular uterus. C and D. 2D TVUS, showing subendometrial microcysts, heterogeneous myometrium, hyperechoic linear striations in the myometrium, and asymmetry of the myometrial walls.[2]

Transvaginal Ultrasonography (TVUS)

TVUS is the method of choice for investigating adenomyosis due to its accessibility, non-invasiveness, low cost, and high accuracy, especially when performed by experienced professionals[6,7]. It has a sensitivity of 83.8% and a specificity of 63.9%[7]. Studies indicate that, in cases of increased uterine bleeding with positive TVUS findings, the probability of adenomyosis is 68.1%; with a normal TVUS, the likelihood drops to 10%[8].

Among the characteristic ultrasound signs are:

- Myometrial heterogeneity;[3,9] (Figure 2)
- Radiating striations originating from the endometrium[3]; (Figure 3)
- Anechoic lacunae and myometrial cysts measuring 1–7 mm (high specificity);[3,9] (Figure 4)
- Increased or decreased echogenicity;[3]
- Combinations such as myometrial cysts with hypoechoic striations show 90% sensitivity and 100% specificity[3]. (Figure 5)

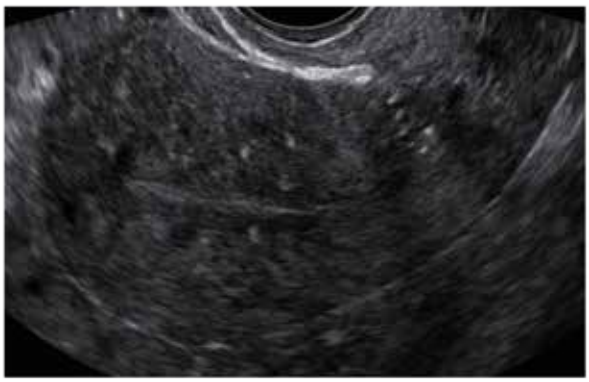

FIGURE 2. Heterogeneous myometrium on TVUS.[10]

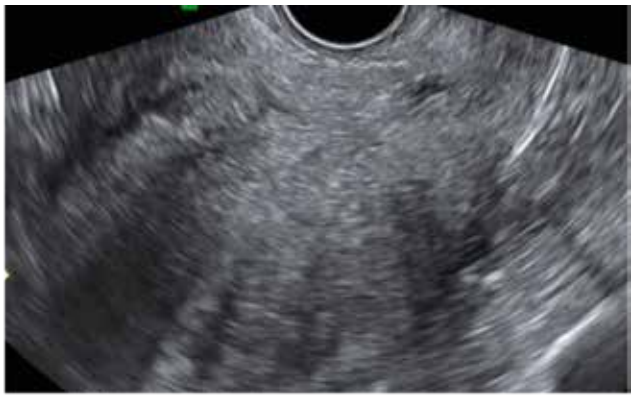

FIGURE 3. Hypoechoic myometrial striations on TVUS.[10]

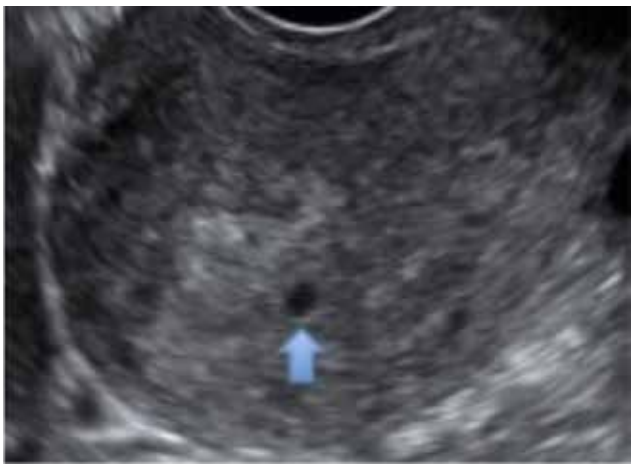

FIGURE 4. Myometrial cyst (arrow) and irregular endometrial–myometrial junction on TVUS.[10]

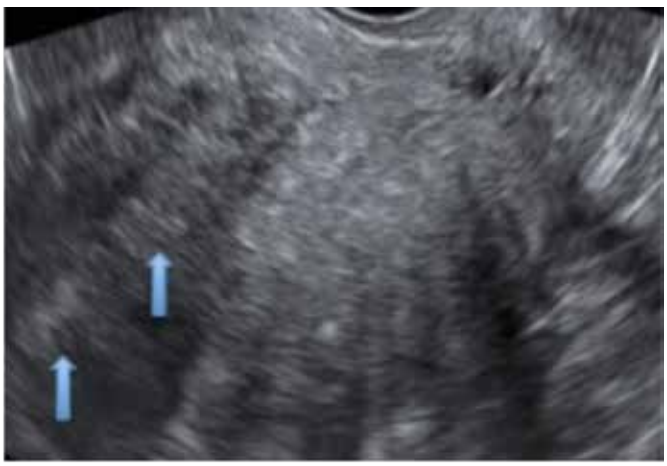

FIGURE 5. Hyperechoic myometrial islands (arrows), hypoechoic myometrial striations, and indistinct endometrial–myometrial junction on TVUS.[10]

For diagnosis, the presence of three or more of these findings is recommended.[10,11]

Three-Dimensional Transvaginal Ultrasonography (3D-TVUS)

3D-TVUS allows for better assessment of the junctional zone (JZ), especially in coronal planes. High-sensitivity findings include distortion and infiltration of the JZ, thickening >8 mm, a difference >4 mm in JZ thickness, and hypoechoic myometrial striations[4,9,11,12]. Despite its anatomical superiority, no significant improvement in overall diagnostic accuracy has been observed compared to the two-dimensional mode[11,13]. (Table 1)

TABLE 1. Main ultrasound findings in adenomyosis. Sensitivity (Se), specificity (Sp), negative predictive value (NPV), and positive predictive value (PPV).[12]

ULTRASOUND CRITERIA	SE (%)	SP (%)	VPN (%)	PPV(%)
Subendometrial cyst	53	83	81	55
Predominantly hypoechoic myometrial echotexture	60	82	83	58
Predominantly heterogeneous myometrial echotexture	35	76	74	38
Subendometrial linear striations	37	92	78	67
Poorly defined endometrial–myometrial junction	45	83	78	53
Nodule at the endometrial–myometrial junction	53	83	83	83
Subendometrial echogenic nodules	32	96	77	76
Asymmetric myometrium	23	94	75	67
Globular uterus	43	79	77	46

Differential Diagnosis: Leiomyomas vs. Adenomyosis

Differentiating between leiomyomas and adenomyosis is challenging, as both conditions can cause a globular or asymmetric uterus.[2] Leiomyomas typically present with well-defined margins, a rounded shape, mass effect, and possible acoustic shadowing. On color Doppler, they show peripheral vascularization. In contrast, adenomyosis is characterized by poorly defined margins, absence of a mass effect, and multiple attenuation foci, with a translesional vascular pattern that crosses the hypertrophied myometrium.[2,11]

The concomitant presence of fibroids can reduce the sensitivity of TVUS to around 30%, reinforcing the importance of clinical correlation and the complementary use of magnetic resonance imaging when necessary[6]. (Figure 6)

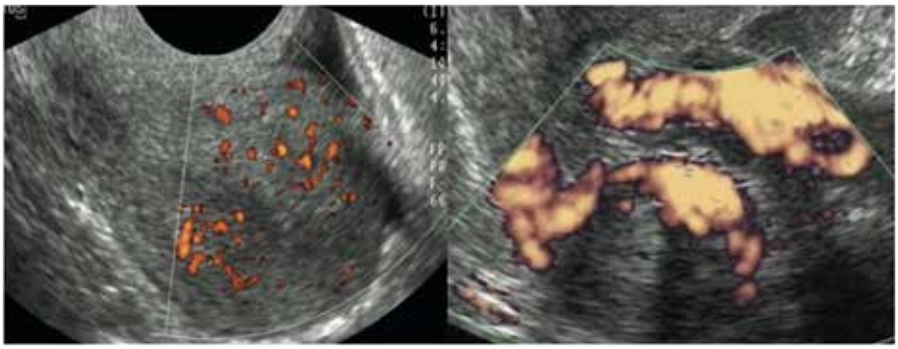

FIGURE 6. Differences in vascularization in adenomyosis and leiomyoma, shown on TVUS with Power Doppler. A. Vascularization crossing the hypertrophied myometrium in diffuse adenomyosis. B. Peripheral vascular projections in leiomyoma.[2]

Technical Advances: Elastography and Contrast-Enhanced Ultrasonography

Elastography, which measures tissue stiffness through gentle compression, has shown promise in distinguishing between adenomyosis and leiomyomas due to the difference in tension generated between the tissues[5]. Studies have already demonstrated improved specificity of TVUS elastography compared to two-dimensional ultrasonography[13],[14].

Moreover, there is evidence that adenomyotic tissue presents angiogenesis and vascular alterations compared to normal myometrium[15], suggesting a potential future role for contrast-enhanced ultrasonography in the diagnosis of adenomyosis. However, no clinical reports have been published to date.[16]

CONCLUSION

Ultrasonography—especially transvaginal ultrasonography—is currently the main imaging method for the non-invasive diagnosis of adenomyosis. The identification of specific ultrasound signs—particularly when three or more are present—allows for an effective and accessible diagnostic approach. Technologies such as Doppler, elastography, and 3D-TVUS provide additional support in differentiating adenomyosis from other pathologies, such as leiomyomas, although ultrasonography remains highly operator-dependent.

In inconclusive or difficult-to-interpret cases, magnetic resonance imaging remains the recommended complementary examination. The continuous advancement of ultrasound techniques, combined with the training of professionals, has the potential to further optimize the diagnosis of adenomyosis and improve clinical management for patients.

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