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We started 2022 with full strength and determination to face a year full of uncertainties and challenges.

With each issue, Revista Científica CEREM Goiás exceeds initial expectations and makes it clear that resident physicians, residency services and the medical community as a whole received our publication in an extremely positive way, in addition to recognizing our uninterrupted efforts to ensure that this journal is a privileged and reliable space for the dissemination of unpublished scientific works.

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ORIGINAL ARTICLE

DERMATOLOGICAL PUNCH BIOPSY IN BREAST LESIONS

ANDRÉ MAROCCOLO DE SOUSA[1], VINÍCIUS DE MORAIS SANTOS[1], LILIANE CÂNDIDA DE PAULA SOUZA[2], GABRIELLA SILVA GARCIA TAGAWA[2], ELAINE XAVIER MACHADO[3], ANA LÚCIA OSÓRIO MAROCCOLO DE SOUSA[4], JUAREZ ANTÔNIO DE SOUSA[4]

ABSTRACT

Breast skin biopsy by punch is an easy, low-cost and accessible procedure, performed under local anesthesia, using a 4 or 5 mm cutting cylinder (punch), which, when rotated in a rotary motion, allows the removal of a fragment with the various layers of the skin, including the epidermis, dermis, and subcutaneous tissue. The fragment is fixed in formalin and later processed and stained with Hematoxylin-Eosin (HE) for histological diagnosis. The paraffin block can also be used to perform immunohistochemistry for diagnostic conclusion and/or study of prognostic factors in breast cancer (estrogen and progesterone receptors, human epidermal growth factor receptor 2 (HER-2) and cell proliferation markers (Ki-67) Punch biopsy is indicated in cases of suspected skin involvement by breast diseases such as areola and nipple eczema, psoriasis, Paget's disease and inflammatory carcinoma.

KEYWORDS: BREAST; PUNCH; PSORIASIS; INFLAMMATORY CARCINOMA; PAGET

INTRODUCTION

The introduction of punch skin biopsy of the breast was a great advance in the diagnosis of skin diseases in Mastology, as it is a method of easy applicability and low cost. Punches ranging from 1 to 8 mm are available, and can be made of metal or disposable plastic. It is a procedure performed under local anesthesia, and a 4 or 5 mm cutting cylinder (punch) is routinely used, which, when rotated in a rotational movement, allows the removal of a fragment with the various layers of the skin, including the epidermis, dermis, and subcutaneous cellular tissue1 (Figure 1).

After anesthesia, the punch must be placed on the skin lesion to be biopsied, making rotation movements, maintaining strong vertical pressure that will make the instrument penetrate the tissue. A cylindrical column of skin with its epidermis, dermis and subcutaneous tissue layers is removed. This skin cylinder is carefully removed with the aid of atraumatic forceps or a skin hook and then the base of the tissue is sectioned with fine scissors as deeply as possible The fragment is fixed in formalin and later processed and stained by the Hematoxylin-Eosin (HE) for histological diagnosis. Punch biopsy is indicated in cases of suspected skin involvement by breast diseases, such as areola and nipple eczema, psoriasis, Paget's disease and inflammatory carcinoma[2] (Figure 2).

LITERATURE REVIEW

The skin lesions of the breast that are most frequently biopsied by punch are eczema, psoriasis, inflammatory carcinoma and Paget's disease.

Eczema or dermatitis are terms used to refer to a polymorphic inflammatory reaction involving the epidermis and dermis. They can be of different etiologies and present varied clinical findings, from erythema, vesicles, xerosis and lichenification. Dermatological lesions are accompanied by pruritus. They may occur due to skin contact with irritating chemicals (primary irritant contact eczema) or an allergen (allergic contact eczema) that triggers a hypersensitivity reaction, or skin dryness3. The diagnosis is made by clinical history, physical examination, patch tests and histopathology (Figure 3).

Psoriasis is an immunologically and genetically based chronic inflammatory disease that clinically presents with erythematous squamous plaques with a symmetrical distribution. Lesions are usually recurrent and may be accompanied by pruritus4. The diagnosis is mainly clinical, but the histopathological examination helps in doubtful cases (Figure 4).

Inflammatory carcinoma is a neoplasm with a poor prognosis, which usually courses with edema of the subcutaneous tissue of the breast, described as "orange peel skin" (peau d'orange), with invasion of the lymphatic vessels of the dermis by emboli of neoplastic cells (Figure 5).

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It manifests with hyperemia, edema and breast enlargement, associated with a palpable nodule. In general, there is extensive involvement of the axillary, supraclavicular and ipsilateral mammary lymph nodes, as well as the contralateral axillary lymph node chain[5].

The diagnosis is based on clinical data and imaging tests. To characterize inflammatory breast cancer, it is necessary to have erythema and edema of the dermis in at least one third of the breast[6].

Histopathological examination by cutaneous incisional or punch biopsy of compromised skin and/or core biopsy of evident tumors.

Paget's disease is a clinical form of breast carcinoma, characterized as an eczematous change in the nipple associated with underlying breast cancer, with infiltration of the nipple epidermis by Paget's cells, which are large, pale-stained cells with round or oval nuclei. and prominent nucleoli[7]. The cells are among the normal keratinocytes of the nipple epidermis, occurring singly in the superficial layers and in clusters towards the basement membrane (Figure 6).

Most patients with Paget's disease initially present with eczema or long-standing nipple ulceration, which can progress to frank erosion, exudation and papillary effusion[8].

The histopathological evaluation of the nipple should be performed by incisional biopsy with a conventional scalpel or by dermatological punch. The breast skin punch biopsy is an outpatient procedure performed under local anesthesia, using a 4 or 5 mm cutting cylinder (punch), which, when rotated, goes deeper into the skin and allows the removal of a cone with the various skin layers, including epidermis, dermis and subcutaneous tissue1 (Figures 7 and 8).

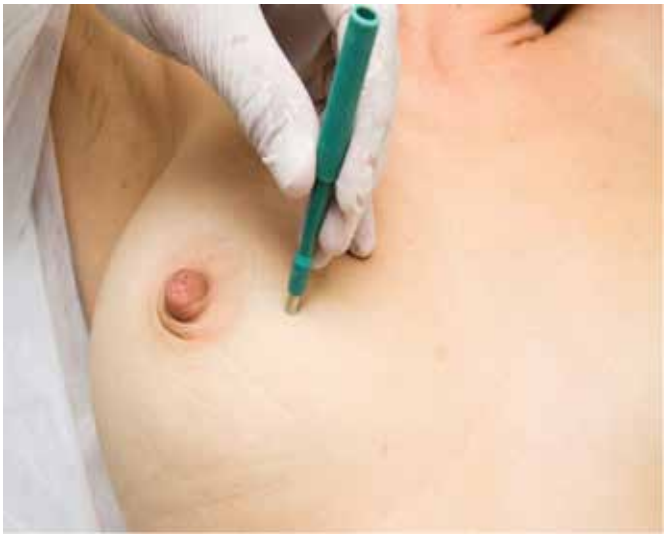

Figure 1: Use of Punch in breast dermatological lesions.

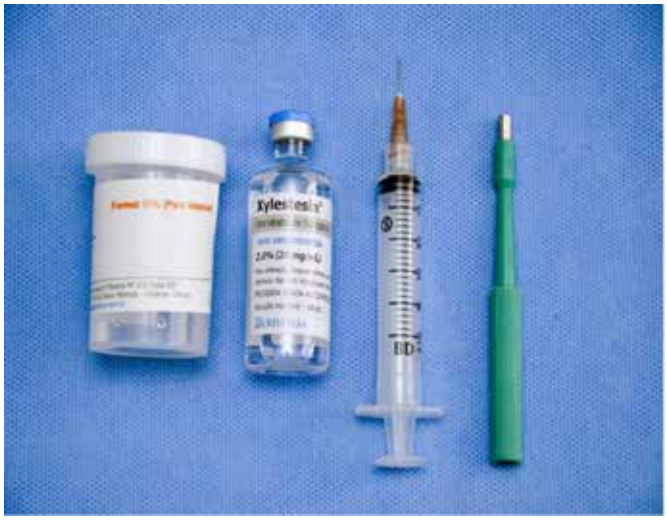

Figure 2: Material needed to perform the punch biopsy. Anesthetic (lidocaine), syringe, needle, punch and formaldehyde.

Figure 3: Left areola eczema.

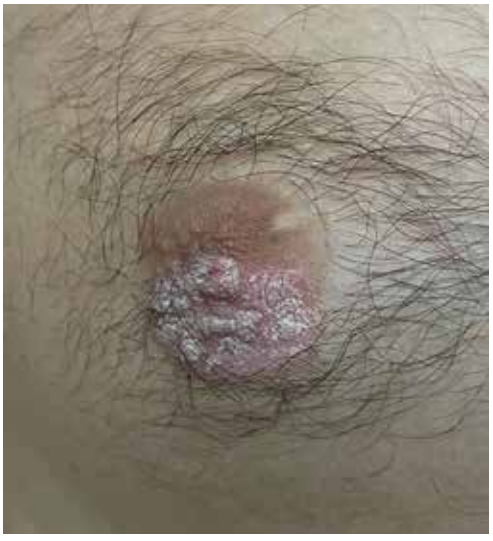

Figure 4 – Plaque psoriasis on the right breast.

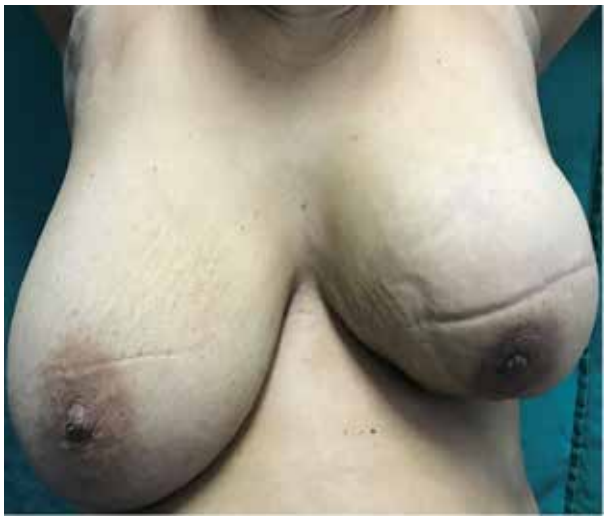

Figure 5: Inflammatory carcinoma in the left breast. Infiltrate the skin by neoplasm ("orange peel" skin).

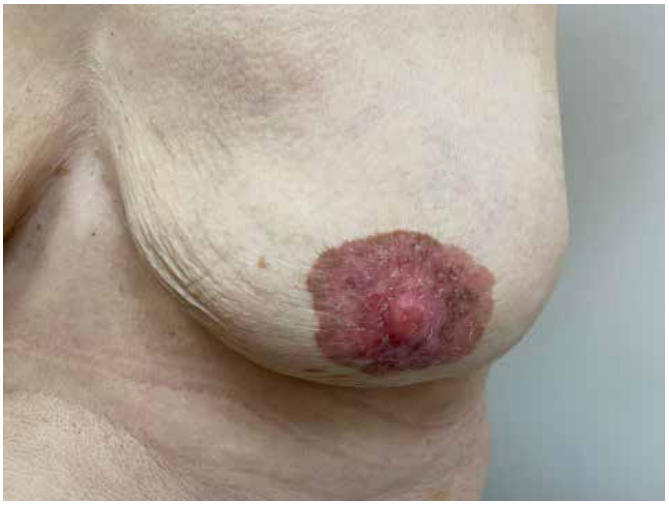

Photo 6: Paget's disease of the left breast.

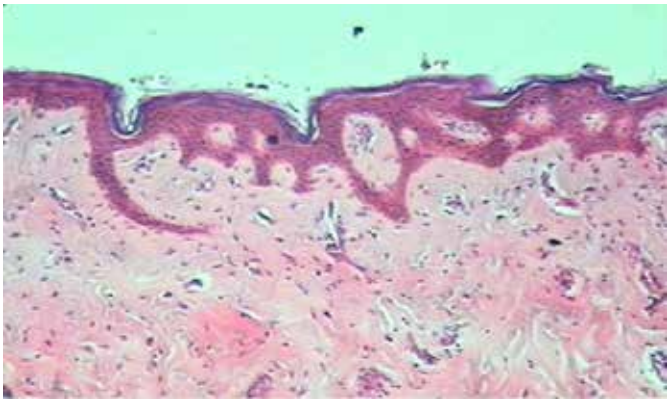

Photo 7: Microscopic appearance of normal skin with its keratin, epidermis and dermis layers.

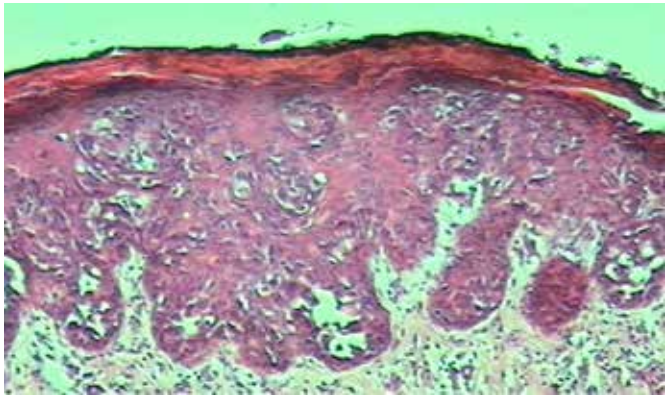

Photo 8: Microscopic appearance of the skin of Paget's disease showing thickening of the epidermis (acanthosis).

CONCLUSION

The development of punch for skin biopsies of breast lesions was a major advance in mastology and dermatology. It is an outpatient procedure that can be done in the office under local anesthesia, very tolerable, fast, practical and without the need for hospitalization. The fragment is fixed in formalin and processed for histopathological and immunohistochemical diagnosis for diagnostic conclusion and/or study of prognostic factors in breast cancer (estrogen and progesterone receptors, human epidermal growth factor receptor type 2 (HER- 2) and cell proliferation markers (Ki-67), which enables the correct treatment of benign and malignant breast diseases such as Paget's cancer and inflammatory carcinoma [9-10].

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CASE REPORT

BIOLOGICAL VALVE PROSTHESIS THROMBOSIS: A CASE REPORT

PAULA CORRÊA BÓÉL SOARES [1], DÉBORA FREIRE RIBEIRO ROCHA [1], CLOVES GERALDINO DA SILVA JUNIOR [2], JOÃO ALBERTO PANSANI [2], ARTUR HENRIQUE DE SOUSA [1,2], GIULLIANO GARDENGHI [2]

ABSTRACT

Valve bioprosthesis thrombosis is a possible complication inherent in cardiac valve replacement surgery. It is a delicate condition that often requires a new surgical intervention. Although little recognized in the literature, biological valve prosthesis thrombosis has been increasing, especially with the advent of transcatheter valve implantation. Despite this, research on the subject remains scarce, which makes the actual number of cases of this event underestimated, with a slow and inadequate treatment response. The present study aims to expose, from a case report, the thrombosis of a biological prosthesis implanted in the mitral position.

KEYWORDS: THROMBOSIS; BIOPROSTHESIS; MITRAL VALVE

INTRODUCTION

Heart valve diseases affect a large part of the world's population, being responsible for a considerable portion of interventions, in order to correct them. With the progress of the various prostheses currently available on the market, there has been a considerable improvement in terms of both hemodynamic characteristics and durability. Despite this, some complications inherent to the procedures can still be seen, valve thrombosis being one of them.[1]

Heart valve replacement for a prosthesis brings with it the risk of developing a dysfunction of it and consequently may lead to the need for a new intervention. Among the main causes the literature gives us the structural deterioration, which can be caused by a Pannus; non-structural deterioration; thrombosis; and endocarditis, and such complications can occur alone or simultaneously. Thrombosis, for example, is often seen associated with prosthetic degeneration or endocarditis.[2]

The replacement of a diseased valve, nowadays, can be performed in two ways: by conventional surgery, in which both mechanical valves (MV) and biological valves (BV) are used, depending on the indication of each patient; and through the percutaneous implant, which only uses BV.[3]

Although less thrombogenic than mechanical prostheses, thrombosis of bioprostheses has been increasingly seen as a cause of dysfunction, mainly due to the growth in the practice of catheter insertion. In a study by Hansson and his collaborators on transcatheter aortic valve implantation (TAVI), the presence of a thrombogenic component was evidenced in 7% of the cases studied, demonstrating the remarkable incidence and clinical relevance of this topic.[2]

Furthermore, bioprosthesis thrombosis (BT) has high morbidity and mortality, classically presenting as an acute heart failure, most often associated with inadequate anticoagulation.[4]

Based on this assumption, and in view of the increased use of BV both in conventional surgery and via catheter, the present work aims to expose, through a case report.

CASE REPORT

A 43-year-old female patient sought the health service for presenting with abdominal distension and pain in the right hypochondrium region, inappetence, gastric fullness and constipation that had started 15 days before treatment. She denied fever and/or chills, fecal acholia, jaundice or choluria.

As a previous history, she said she had fibromyalgia and denied smoking, alcohol consumption, family history of coronary heart disease or other comorbidities. She also said that, in 2012, she was diagnosed with mitral stenosis of rheumatic origin, requiring balloon mitral valvuloplasty, in the same year. Despite this, in 2019 she required mitral valve replacement surgery (MTS) with placement of a biological prosthesis, which was successfully performed.

In November 2020, she had a first episode of pulmonary thromboembolism (PTE), followed by another in April 2021, both treated with antithrombotics. In August 2021, she had mild COVID-19, conducted as a symptomatic patient at home.

On physical examination, the patient was in a regular general condition, hydrated, ruddy, acyanotic, anicteric, afebrile, Glasgow 15. Physiological vesicular murmur with fine bibas-

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al rales, regular heart rhythm, normophonetic sounds, in two stages, with diastolic murmur in mitral focus with irradiation to aortic and axillary focus. Hepatojugular regurgitation present.

On admission, computed tomography (CT) of the abdomen and ultrasound (USG) of the abdomen were performed, which respectively showed gallbladder with thickened and delaminated walls associated with densification of adjacent adipose planes, suggesting acute cholecystitis; and ultrasound signs of acute cholecystitis with positive Murphy. Inferior vena cava showing increased caliber.

In addition, the patient underwent an electrocardiogram (ECG) that showed sinus rhythm, with a heart rate of 64 bpm, a low amplitude QRS complex in the frontal plane, with a right bundle branch conduction disorder, as well as diffuse changes in ventricular repolarization (Figure 1).

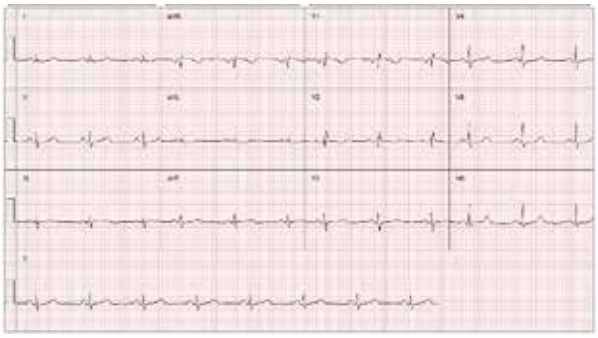

Figure 1. Electrocardiogram image on admission.

She was then referred for transesophageal echocardiogram (TEE) to investigate cardiac injury, the latter having demonstrated a significant increase in the right ventricle (RV), with a heterogeneous mass of wide mobility measuring 16.6 mm x 6.7 mm adhered to a of the mitral valve leaflets, causing significant stenosis-like dysfunction with a mean gradient of 11 mmHg and a peak gradient of 16 mmHg. A tricuspid valve with significant mitral regurgitation (MR) was also visualized. Other examination findings were within the normal range (Figure 2).

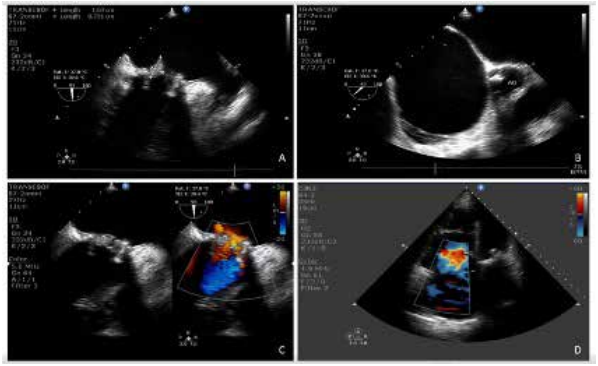

Figure 2. Perioperative transesophageal echocardiogram A) Thickening of the mitral valve leaflets. B) Right ventricular enlargement. C) Mitral stenosis demonstrating regurgitation by Collor. D) Tricuspid insufficiency.

Based on these findings, due to the hemodynamic repercussions presented by the patient, and after discussion with the Heart Team, a new surgical approach for MVR was chosen, proving its involvement intraoperatively (Figure 3). Long-term BV implant #29 (Medtronic-Hancock II®) was performed in the mitral position, closure of the left atrium and tricuspid valve repair with a semi-rigid #32 ring. Subsequently, she was referred to the cardiology ICU, where she remained for monitoring and postoperative recovery (PR).

Figure 3. Intraoperatively removed thrombosed prosthetic valve A) Biological valve removed from the mitral position, demonstrating involvement of its leaflets. B) Removed thrombus or biological valve side

The patient was discharged on the sixth postoperative day, after transthoracic echocardiogram (TTE) (Figure 4) proving good functionality of the implanted prosthesis, in addition to significant clinical improvement of the patient, who continued to use Apixaban 5 mg twice a day. She returned on the 30th PO for outpatient follow-up, in which the presence of atrial flutter was found on the follow-up ECG (Figure 5), requiring a new hospitalization. During this, she underwent laboratory investigations, showing significant anemia, requiring blood transfusion. She was also referred to the hematology service for causal investigation. She decided to be discharged again after 48 hours, having received two bags of blood and in sinus rhythm, controlled with Amiodarone 200 mg/day.

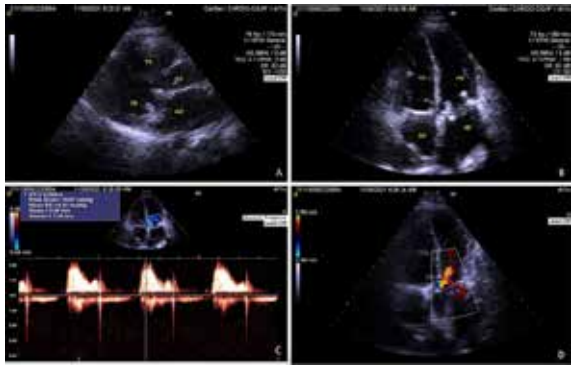

Figure 4. Postoperative transthoracic echocardiogram demonstrating good functionality of the new prosthesis

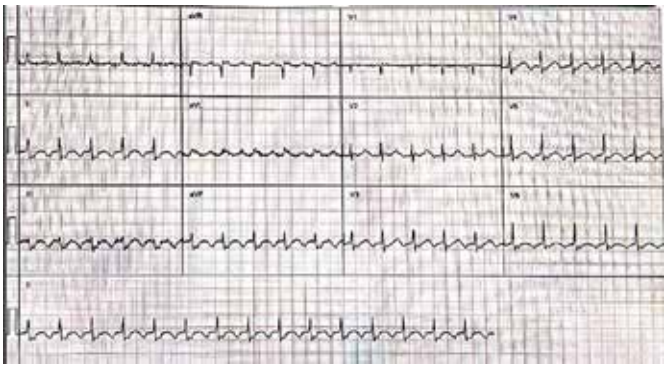

Figure 5. Admission ECG in the second hospitalization

DISCUSSION

In the above case, what stands out is the presence of a BPVT, in addition to multiple episodes of PTE, and its correlation with the therapeutic approach, both before and after the thromboembolic event occurred, and the diagnostic methods adopted for better resolution of the condition.

It is known that prosthetic heart valve thrombosis has a low incidence, with a percentage between 0.03% and 4.3% patients per year. Within this statistic, 0.5% occur on the left side of the heart, 0.1% of which are in the mitral position. In relation to biological valves, the occurrence of thrombosis is even more uncommon, which is why the main advantage of their use over mechanical valves. In addition, as it is not very thrombogenic, the long-term use of oral anticoagulants is unnecessary. Although rare, especially in the mitral position, there are few studies or reports evidenced in the literature, and when present, they are about MV. Because of this, the real prevalence of thrombosis in BV is unknown and may even be underestimated. [5]

In studies carried out with patients who underwent MVR with the use of biological prostheses, followed up for a period of five years, it was seen that in 10% of the evaluated cases there was the presence of thrombi in the prosthetic valve leaflets, a percentage that is similar to other reports found in the works. performed so far, which is a considerable statistic that should not be ignored.[6]

Most cases of biological prosthetic valve thrombosis (BPVT) are related to insufficient oral anticoagulation therapy. It is known that in the first year after surgery, there is a greater risk of having a valve thrombosis due to recently handled perivalvular tissue that has not yet been endothelialized, with a high thrombogenic potential. In addition, atrial fibrillation secondary to fibrosis that occurs at the expense of the procedure predisposes to thrombus formation. Thus, the institution of adequate anticoagulation is necessary. [5]

In addition, careful follow-up should be employed in both the immediate and late PO. In the present studies, BPVT, although uncommon, when it occurs, appears in the first months after the surgery, with a good response in relation to the use of vitamin K antagonists. However, this fact does not exclude the possibility of such an event occurring later. However, current guidelines only give us a restricted period of three to six months in relation to the use of this therapy. In the works carried out by Egbe and his collaborators, it was evidenced that BPVT has a prevalence of 11.6%, which is much higher than that reported at the present time, which denotes the importance of developing strategies for an early diagnosis, since that a TTE with the presence of three echocardiographic signs of a thrombus is highly sensitive and specific to confirm the diagnosis.[7]

Previously related only to the surgical procedure, today we see an exponential increase in transcatheter procedures, which use only biological valves. Thus, the ways of evaluating the consequences in relation to the growth in the use of biological prostheses should be expanded, considering that there is a possibility of subclinical thrombosis of the valve leaflets.

In a survey carried out by Chakravarty et al. between 2014 and 2017, the presence of changes in BV implanted by both means already known via tomographic images was found, and such changes are of great significance.8 Based on this assumption, in view of the scarcity of studies in relation to this specific topic , the view of the authors of the present case report remains that it is necessary to expand research regarding BPVT, as well as to optimize the therapies used after performing such procedures.

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CASE REPORT

INTERMEDIATE UVEITIS SECONDARY TO COVID-19 INFECTION: A CASE REPORT

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ABSTRACT

Objective: a case of intermediate uveitis, post-acute period infection, mediated by SARS-CoV-2, documenting the multiform clinical presentation of COVID-19. Materials and method: case report and image exams, with bibliographic review. Results: A 50-year-old man, with a positive Polymerase Chain Reaction (PCR) in a nasal swab for SARS-CoV-2, 10 days after isolation, complained of low visual acuity and bilateral blurring. Vitreitis in both eyes, 2 + / 4 in OR and 1 + / 4 + in OS and vitreous haze were documented in retinography. 15 days after the early diagnosis and the start of treatment, the patient evolved with improved visual acuity. In the reassessment of biomicroscopy and fundoscopy, there was an improvement in the vitritis pattern. Conclusions: the patient denied a medical history of chronic autoimmune and inflammatory diseases, and possible etiologies were excluded. Clinical presentation, early diagnosis and clinical response, with gradual reduction and satisfactory response, shows an intermediate uveitis. We present this case of ocular involvement, days after a systemic inflammatory condition by COVID - 19, to document the extraordinary and multifaceted capacity for clinical viral manifestation.

KEYWORDS: INTERMEDIATE UVEITIS; COVID-19; PUBLIC HEALTH

INTRODUCTION

A new epidemic of the RNA virus, with envelopes belonging to the family Coronaviridae [1], capable of causing a severe acute respiratory syndrome coronavirus - 2 (SARS-CoV-2), emerged from China in late 2019. Literature descriptions conceptualize "COVID-19" as an inflammatory storm, supported by cytokines, of a multisystem nature[2].

Viruses of the Coronaviridae family (CoVs) are also known to manifest in regions other than the respiratory tract, including the gastrointestinal tract and ocular tissues [1]. In 2004, near the end of the SARS-CoV crisis, the polymerase chain reaction (PCR) in tears from patients with SARS-CoV infection demonstrated the presence of the virus. The discovery of SARS-CoV in tears was the first of its kind in emphasizing the need for adequate precautions to prevent potential transmission through ocular tissues and secretions [3].

In felines and murine models, it is known that viruses of the Coronaviridae family are known to cause various ocular involvements, with conjunctivitis, anterior uveitis, retinitis and optic neuritis. In SARS-CoV-2, the ocular pathology manifests itself, as expected, in a variety of ways [3].

Recently, in the "SERPICO-19" study, 54 patients, among the 133 exposed, were identified with retinal changes, where the main changes were microvascular, especially microhemorrhages and cotton-wool exudates [4]. It is believed that this correlation between retinal manifestations and uveal and COVID-19 is related to the ACE 2 cell receptor, detected in the human retina, retinal pigment epithelium, choroid, cornea and conjunctival epithelium [1,4].

A recent survey showed that the main eye complaints of patients with SARS-CoV-2 are dry eyes, blurred vision and foreign body sensation. It is believed that they are related much more to the more intense use of electronic devices in quarantine phases than to the infectious manifestation. However, in some patients, keratoconjunctivitis was the first clinical manifestation 3,5. Some studies indicate that the presentation of SARS-CoV-2 and keratoconjunctivitis may be associated with a more severe form of the disease [3,5]. It may be present in conjunctival secretions, requiring greater attention and caution on the part of the patient and the multidisciplinary team that will manage the patient [3].

Furthermore, there are, in the literature, varied descriptions of infrequent ocular presentations of COVID-19. Bettach et al., as an example, postulated the first case of bilateral anterior uveitis secondary to multisystemic inflammation of SARS-CoV-2 [6]. The word uveitis was created to describe an inflammatory process of the ulvea,

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the region that constitutes the bulbi vasculosa (iris, body ciliary and choroid), but the current term is synonymous with intraocular inflammation [7].
There are several etiologies, of autoimmune or infectious origin, that can develop the pathology, and the forms of clinical presentation are also varied, depending on the inflammatory anatomical site. However, it is known that all are mediated by the immune system, where the genes of the MHC complex regulate the production of cytokines and are involved in the susceptibility to the development of uveitis [7].

CASE REPORT

A 50-year-old man sought care at the Ophthalmological Emergency Room with a complaint of low vision after treatment for a COVID-19 infection, confirmed in a nasopharyngeal swab Polymerase Chain Reaction (PCR). He reports that he was hospitalized for treatment of dyspnea, fever and cough with analgesics associated with systemic corticosteroids.
He complained of bilateral blurring of vision after 10 days of hospital discharge, with no previous ocular pathological history, on examination: distance visual acuity of 20/50 in the right eye and 20/40 in the left eye (Snellen chart at 6 meters). Biomicroscopy showed an anterior chamber with mild anterior chamber reaction and fine paracentral keratic precipitates (PKS) in both eyes. The retinal mapping exam showed a clinically preserved retina up to the ora serrata, however vitritis in both eyes (BE), 2+/4 in the Right Eye (RE) and 1+/4+ in the Left Eye (LE), documented by the simple retinography (figure 1).
It is worth remembering that, for the evaluation of the vitreous haze scale, characteristic of this clinical presentation, it is graded from 0-4, where the main factors evaluated are the presence of blurring of the optic nerve and retinal vessels. For evaluation of the anterior chamber, the scale for counting cells scattered in the light beam in biomicroscopy is used. However, vitreous haze, according to the American Academy of Ophthalmology (AAO), is the best way to indicate intermediate uveitis activity[7].
On fluorescein angiography (Figure 1), no vascular, macular or papillary abnormalities were observed in both eyes and Optical Coherence Tomography (Figure 3) showed a macula with preserved neurosensory retinal architecture and retinal pigment epithelium. A diagnostic hypothesis of subacute, bilateral, asymmetric intermediate uveitis secondary to COVID-19 was raised.

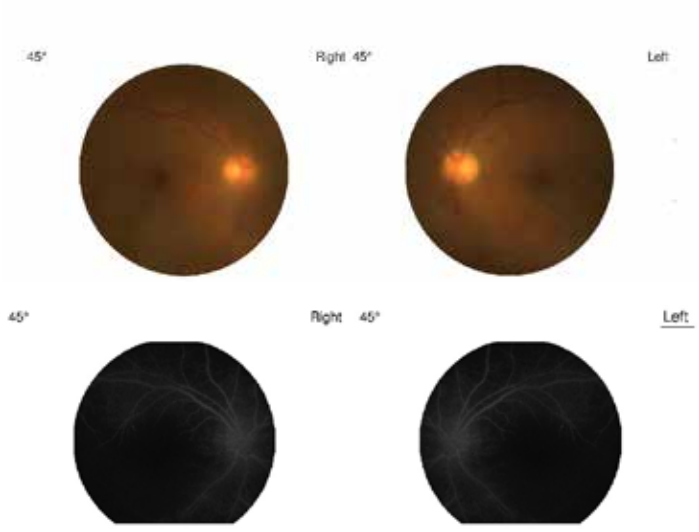

Figure 1. Color Retinography (upper): Vitreous haze 2+/4+ in the right eye and 1+/4+ in the left eye. Fluorescein angiography (lower): Intermediate phase of the exam without changes in circulation under sodium fluorescein.

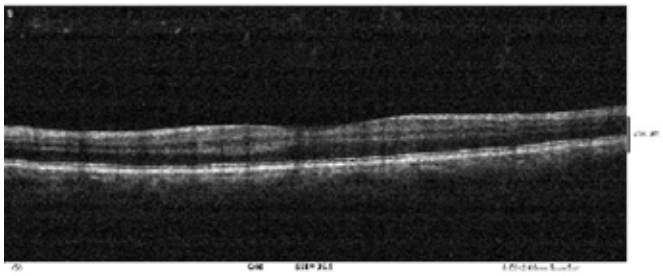

Fig 2. RE macular optical coherence tomography: Posterior optical shadow foci from the vitreous cavity.

Topical treatment was started with 1.0% prednisolone acetate eye drops, one drop, in both eyes, every 4 hours. The patient evolved with significant improvement in visual acuity, and on examination after 15 days: 20/25 in the right eye and 20/20 in the left eye. In the reassessment of biomicroscopy and funduscopy, there was important resolution of the vitritis pattern and placid anterior chamber without keratic precipitates (PKS). There was weaning from the topical treatment and progressive improvement without reactivation of the condition.
Infectious diseases such as syphilis, herpes, tuberculosis, HTLV, toxocariasis and viral hepatitis were ruled out. Cat-scratch disease, sarcoidosis, Lyme disease and multiple sclerosis were also excluded. It is worth mentioning that the patient has no medical history of other previous eye diseases or chronic systemic autoimmune, inflammatory and infectious diseases.

DISCUSSION

It is known that SARS-CoV-19 resembles a hyperferritinemic syndrome, in its main stages, coursing with:

lymphopenia, reduction in the number and activity of NK lymphocytes, coagulopathy and hyperferritinemia, which demonstrates the great pro-inflammatory capacity, which induces the expression of different inflammatory mediators, mainly IL-1β [1].

According to Colanfresco et al., despite the numerous etiologies that can develop the hyperferritinemic syndrome, they can converge in at least two mechanisms that cause hyperferritinemia: T lymphocyte hyperactivation and IFN-γ [1,2] hyperactivity. However, recent evidence has described the direct role of ferritin H chain in macrophage activation to increase the secretion of inflammatory cytokines, evolving with macrophage activation syndrome (MAS), antiphospholipid syndrome (CAPS) and septic shock [1].

This pro-inflammatory condition can be observed in several observational studies, where an increase in the number of autoimmune conditions, such as Kawasaki syndrome, was found. In children, in cities such as Paris8, France, and Bergamo, Italy9, the SARS-CoV-2 epidemic has been associated with a high incidence of a severe form of Kawasaki disease, such as children's multisystem inflammatory syndrome (KDSS) and macrophage activation syndrome (MAS) [8,9].

In a peculiar way, kawasaki syndrome is an acute vasculitis of medium-sized vessels, with systemic decompensation, with an immune-mediated trigger, which often leads to anterior uveitis [8,9]. It is believed that the correlation between intraocular inflammation and Kawasaki syndrome lies in the large inflammatory storm present in the pathology, with high levels of IL-6, C-reactive protein and procalcitonin [8,9].

There are reports of bilateral acute anterior uveitis (iridocyclitis) associated with blurred vision, associated with a multisystem inflammatory condition secondary to COVID-19, coursing with corneal edema, diffuse Descemet folds and keratic precipitates (PKs) in both eyes, with good prognosis after topical and systemic therapy with corticosteroids described in the literature [1,3,5].

In time, intermediate uveitis is a subgroup of uveitis, where the main site of inflammation is the vitreous, peripheral retina and pars plana, epidemiologically, it is not usually associated with gender or race, and the involvement tends to be bilateral in 70% of cases. The most frequent initial symptom is the perception of floaters and decreased visual acuity [7].

The eye generally has a lower inflammatory pattern compared to presentations of anterior uveitis, with mild hyperemia and moderate anterior chamber reaction. The clinical presentation also includes small, white, thin keratic precipitates, usually in the lower half of the cornea. Vitreitis is the hallmark of the disease, ranging from mild to severe, becoming more condensed and classically focal, such as snowballs, are observed during progression [7].

Snowballs are peculiar vitreous infiltrations, containing mononuclear leukocytes and fibrocyte-like cells, muller cells, and fibrous astrocytes. Apparently, the pathophysiology is related to a disease mediated by T cells, which, by immunotaxis initiated by an unknown antigen, leads to a picture of vasculitis and vitreous inflammation [7].

It is possible that the antigen is infectious because intermediate uveitis is seen in infectious diseases such as Lyme, syphilis, and cat-scratch fever. The disease may be autoimmune as the pathology is also seen in non-infectious diseases such as multiple sclerosis and sarcoidosis. Type II collagen in the vitreous may be an autoantigen in some patients [7].

HLA associations have been reported in intermediate uveitis, in which HLA-DR is the most significant, occurring in 67-72%. Promising studies correlate human leukocyte antigen (HLA), which are proteins encoded in the major histocompatibility complex, for the recognition and immune defenses of COVID-19, which may condition an individual more susceptible or more resistant to the inflammatory storm typical of the acute phase of the disease, such as HLA-B*46:01 and HLA-B*15:03 [10].

In general, the condition of intermediate uveitis is usually benign, where its complications are due to chronicity. Glaucoma, cataracts, macular edema and maculopathy, secondary to intraocular inflammation, are possible complications [7]. Early diagnosis and therapeutic intervention can avoid these conditions, therefore, it is of fundamental importance to discuss the clinical and inflammatory presentations, as well as the therapeutic approach of this multisystemic viral condition, in this ongoing Pandemic, and therefore, an important public health issue.

CONCLUSION

Regarding this COVID-19 case, it was not possible to perform the tear swab PCR or the vitreous humor PCR, so we cannot say that the uveitis presented was caused by the coronavirus. The good response to early clinical treatment speaks in favor of a self-limited subacute intermediate uveitis. After excluding other causes and possible etiologies, we considered a presumptive diagnosis of intermediate uveitis secondary to coronavirus.

The manifestation of intermediate uveitis, in this case reported, occurred shortly after treatment of acute systemic illness by COVID-19. One hypothesis raised is the post-infectious immune-mediated presentation. Another hypothesis raised is that uveitis did not manifest early due to the concomitant use of systemic corticosteroids and that, after discontinuation of the same, intraocular inflammation set in.

We report this case of ocular involvement, days after the systemic inflammatory condition by SARS-CoV-2, to document the extraordinary and multifaceted capacity of viral clinical manifestation, as a cause of low visual acuity, in an alarming pandemic scenario.

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CASE REPORT

INFLAMMATORY ABDOMINAL AORTIC ANEURYSM – ENDOVASCULAR TREATMENT: CASE REPORT

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ABSTRACT

INTRODUCTION: Inflammatory Abdominal Aortic Aneurysms are characterized by a dense perianeurysmal inflammatory reaction, characterized by the presence of a thickened arterial wall and increased laboratory tests of inflammatory activity. CASE REPORT: A 55-year-old female patient with a CT scan of the abdomen showing the presence of an infrarenal Abdominal Aortic Aneurysm measuring 14.1 x 8.1 x 9.1 cm. She underwent endovascular repair after remission of the inflammatory activity with the use of corticosteroids and methotrexate. After 6 months of treatment, the presence of type II Endoleak was observed. DISCUSSION: Inflammatory aneurysms should be suspected in patients with aortic aneurysms with an atypical epidemiological history. The incidence of rupture tends to be similar to that of non-inflammatory aneurysms. Open surgical resection is difficult due to the extensive fibrous and inflammatory reaction and adhesion to adjacent structures. The application of endovascular surgery for this type of aneurysm is considered promising. Type II endoleak is the most frequent and is characterized by a retrograde reflux through the aortic branches. For cases in which there is no expansion, the recommendation is to carry out follow-up with serial imaging tests.

KEYWORDS: INFLAMMATORY ABDOMINAL AORTIC ANEURYSM; ENDOVASCULAR SURGERY; TYPE II ENDOLEAK

INTRODUCTION

Inflammatory Abdominal Aortic Aneurysms correspond to approximately 3 to 10% of abdominal aortic aneurysms. They are characterized by a dense perianeurysmal inflammatory and fibrotic reaction that encompasses neighboring structures, rarely reaching the aorta above the emergence of the renal arteries.[1]

The diagnosis is suspected because of abdominal or back pain, weight loss, increased erythrocyte sedimentation rate (ESR), and symptoms of involvement and ureteral stricture with hydronephrosis. [3, 4]

The tomographic findings that suggest its diagnosis are the presence of contrasted aortic lumen, with non-opacified mural thrombus and thickened arterial wall, tending to involve mainly the anterior and lateral walls, preserving the posterior.[1, 4, 5]

The first open surgical repair to correct an abdominal aortic aneurysm was performed in 1051 by Charles Dubost, with a homologous graft.[4] Since then, such techniques have been improved, and the advent of endovascular surgery has brought surgical correction as a less invasive option.

CASE REPORT

Patient MMBX, 55 years old, female, referred for outpatient care in November 2020, with a report of recurrent abdominal pain and distension, which started about a year before. The condition was associated with the sensation of a pulsating abdominal mass in the periumbilical region. Patient denies smoking history, denies previous personal history of vascular disease or family history. She denied other associated comorbidities.

During the investigation of abdominal pain, an abdominal CT scan identified the presence of an infrarenal Abdominal Aortic Aneurysm measuring 14.1 x 8.1 x 9.1 cm, approximately 2.8 cm below the renal arteries, with wall thickening, extending to the proximal portion of the iliac arteries bilaterally. Carotid Doppler showed hyperplasia of the bilateral intimal-medial complex of common carotid arteries.

In laboratory tests, he had an ESR of 122 and a CRP of 1.1. Follow-up with the Rheumatology team began, which started treatment for large vessel vasculitis, with corticosteroid therapy followed by methotrexate for a period of 60 days. After reducing the inflammatory activity, preoperative laboratory control tests showed an ESR of 8 and CRP of 0.5. Surgical treatment was then indicated to correct the Abdominal Aortic Aneurysm.

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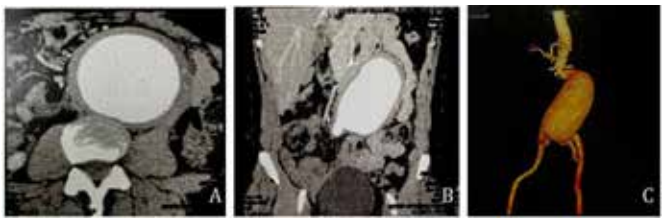

Figure 1: Computed Tomography Angiography of the abdomen – A: axial section showing an Abdominal Aortic Aneurysm measuring 9.1 cm in its largest diameter, with thickened walls; B: coronal section showing an Abdominal Aortic Aneurysm with an extension of 14.1 cm; C: 3D Reconstruction of Infrarenal Abdominal Aortic Aneurysm.

Endovascular surgical correction was opted, with a customized endoprosthesis. The procedure was performed in February 2021 without complications.

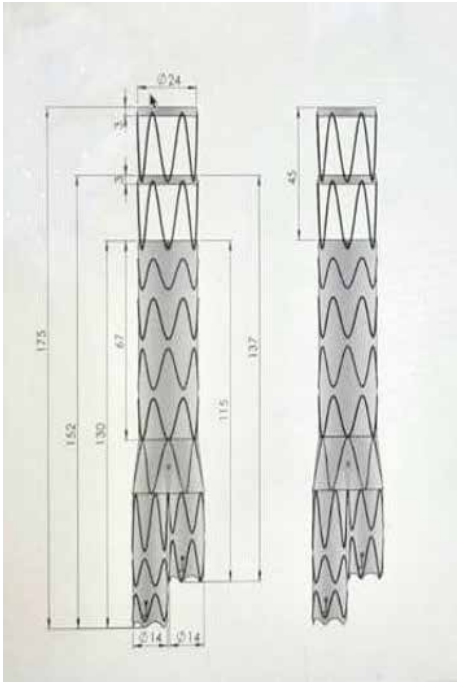

Figure 2: Customization project for a "double free-flow" stent in proximal modules.

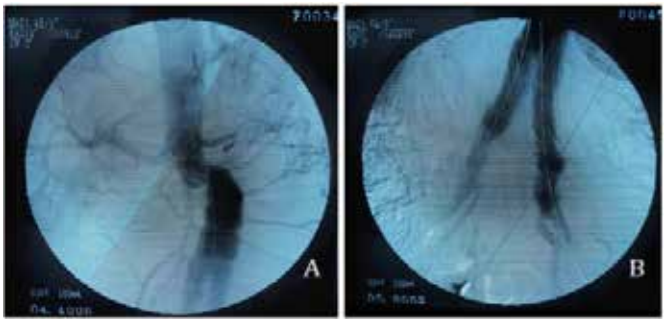

Figure 3: Intraoperative arteriography. A: proximal neck; B: iliac branches of the prosthesis.

The patient had a good postoperative evolution, being discharged on the 12th postoperative day in good clinical condition, for outpatient follow-up.

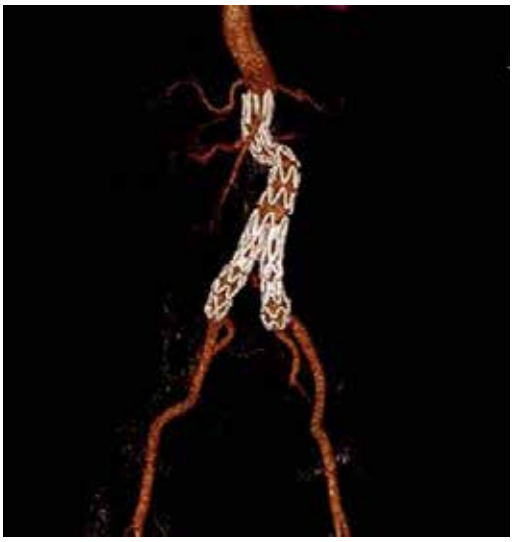

Figure 4: 3D reconstruction of abdominal CT angiography, showing satisfactory early postoperative control of endovascular repair of Inflammatory Abdominal Aortic Aneurysm.

After 6 months of treatment, a new contrast-enhanced tomography of the abdomen was performed, which confirmed the presence of type II endoleak, with a normopositioned prosthesis, without evidence of aneurysm expansion. Follow-up with serial angiotomography every 6 months was opted.

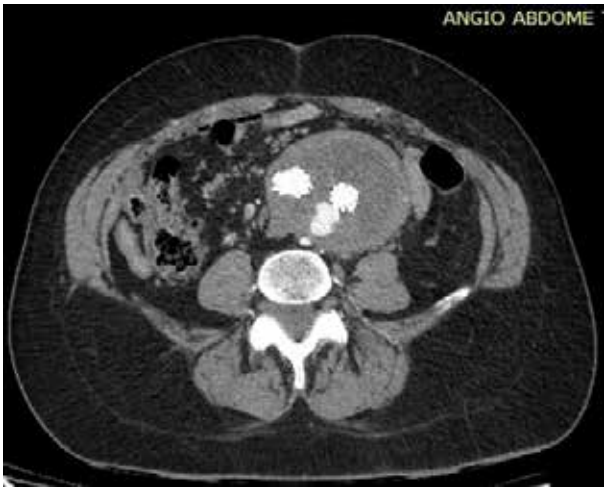

Figure 5: Axial section Angiotomography of the abdomen, showing type II Endoleak.

DISCUSSION

Inflammatory aneurysms should be suspected in patients diagnosed with Abdominal Aortic Aneurysm,

in which the clinical history is not compatible with the main known risk factors, such as male gender (about 4 to 6 times more frequent, in relation to women), advanced age, smoking, positive family history for first-degree relatives, obesity and Caucasian race.[2, 5, 6] Reinforced by the findings of imaging tests, which may show the presence of non-opacified mural thrombus and thickened arterial wall. [1, 4]

The incidence of rupture tends to be similar to that of non-inflammatory aneurysms, occurring most commonly in the posterior wall of the aneurysm, which is not thickened. Thus, the surgical indication is the same for non-inflammatory patients.[3, 10]

In inflammatory aneurysm, open surgical resection is difficult due to extensive fibrous and inflammatory reaction and adhesion to adjacent structures such as ureters and duodenum. This makes dissection of the proximal and iliac aorta difficult, and favors the occurrence of lesions in other structures that are also encompassed and difficult to identify.[7, 8] For this reason, the application of endovascular surgery for this type of aneurysm is considered promising.9

Endoleaks are persistent blood leaks into an aneurysmal sac after endovascular repair of the aneurysm. Type II Endoleak is the most frequent and is characterized by being a retrograde reflux through the aortic branches.9, 6 Interventional treatment for this leak is recommended if there is aneurysmal expansion or with the onset of symptoms attributable to the leak. For cases in which there is no expansion, the recommendation is to carry out follow-up with serial imaging tests, since 30% to 50% will be resolved without any intervention.[9]

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CASE REPORT

ACUTE OBSTRUCTIVE ABDOMEN RESULTING FROM LEFT OBTURATOR FORAMEN HERNIA - CASE REPORT

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ABSTRACT

INTRODUCTION: Obturator foramen hernias are rare and have high morbidity and mortality due to delayed diagnosis. It is more frequent in women and its treatment is imminently surgical.

CASE REPORT: Female patient, 74 years old, referred for evaluation by the general surgery team with colic-like left iliac fossa pain radiating to the medial face of the ipsilateral thigh with 14 days of evolution. Associated with the condition, he reported hyporexia, nausea, vomiting and evacuation failure for 13 days. He denied previous pathologies. The patient had a computed tomography (CT) scan of the abdomen with a diagnosis of left inguinal hernia. Physical examination of the left inguinal region incompatible with diagnosis. A new CT scan was performed with evidence of obturator foramen hernia. Patient submitted to exploratory laparotomy, observing non-viable jejunum segment insinuated in obturator foramen. Enterectomy with enteroanastomosis was performed. The patient had a good clinical evolution. DISCUSSION: The obturator foramen hernia is a rare entity whose main predisposing factors are female sex, advanced age, low BMI associated with factors that lead to increased intra-abdominal pressure. It is difficult to diagnose clinically, and is commonly performed during an exploratory laparotomy procedure due to an obstructive acute abdomen. Attention should always be paid to this etiology in cases of obstructive acute abdomen without a defined cause. Its early identification and treatment promotes a considerable reduction in the morbidity and mortality of this pathology.

KEYWORDS: FORAMEN, OBTURATORY HERNIA; ACUTE OBSTRUCTIVE ABDOMEN

INTRODUCTION

Obturator foramen hernias are rare, accounting for less than 1% of all hernias and 0.2 to 1.6% of the etiologies of mechanical obstruction of the small intestine. It has a high mortality rate, ranging from 38 to 81%. This fact is due to the delay in diagnosis, which occurs when the herniated segments are already unfeasible, leading the patient to cases of sepsis with an abdominal focus. Its first case was reported in the 18th century, in 1724 by Arnaud de Ronsil. Only in 1851 was the first case successfully operated by laparotomy performed by Henri Obre described.[1]

They occur more frequently on the right, considering that the presence of the sigmoid colon in the left iliac fossa covers the obturator foramen in this region.[2] It is more frequent in women (6:1) due to anatomical and physiological factors inherent to the female sex: wider pelvis , greater opening and inclination of the obturator canal associated with greater transverse diameter of the pelvis. Other associated factors are peritoneal laxity, chronic constipation, multiparity and age.[1]

In this hernia, there is an inferior projection of the hernial sac, below the pectineus muscle or posterior to the external obturator muscle. It is a hernia that is difficult to palpate. Its contents usually consist of omentum or lateral clamping of the intestine (Ritcher's hernia), but incarceration of other abdomino-pelvic structures such as appendix, fallopian tube, ovary may occur.[2]

The clinical picture is triggered by compression of the obturator nerve, generating pain and/or paresthesia on the inner side of the thigh, which may extend to the medial side of the ipsilateral knee (Howship-Romberg sign). Another clinical sign presented as a result of compression of the obturator nerve is that of Hannington-Kiff, which consists of the absence of the reflex of the adductor muscle of the thigh when performing percussion in the region of the ipsilateral adductors. Acute abdominal pain is usually associated with signs and symptoms of intestinal obstruction, or sciatica-like pain. The incidence of intestinal necrosis associated with obstruction is high.[3]

The main surgical access routes for correction of obturator foramen hernias are: transperitoneal abdominal, preperitoneal abdominal, inguinal and femoral.[1]

CASE REPORT

A 74-year-old female patient referred for evaluation by the general surgery team with colic-like left iliac fossa pain radiating to the medial face of the ipsilateral thigh within 14 days of evolution. Associated with the condition,

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he reported hyporexia, nausea, vomiting and evacuation failure for 13 days. He denies previous pathologies. He brought with him an abdominal computed tomography (CT) report performed outside our service with a report of an incarcerated left inguinal hernia with dilatation of the upstream small intestine.

On physical examination, she was in a regular general condition, normocardia, normotensive, dehydrated 2+/4+, lucid and oriented in time and space. Cardiovascular and respiratory systems with no alterations. Abdominal examination showed distended abdomen, with pain on deep palpation in the left iliac fossa with positive sudden decompression in this region. In the inguinal region, no signs of inguinal or femoral hernias were found. Rectal examination with the presence of small fecal remnants, without blood on a gloved finger.

After physical examination, an open nasogastric tube was requested, X-ray of the acute abdomen (standing and lying down) was requested, followed by laboratory tests. Acute abdominal X-ray showed diffuse distention of small bowel loops with no evidence of obstruction points to the method. A new CT scan of the abdomen was then requested, with evidence of left obturator foramen hernia.

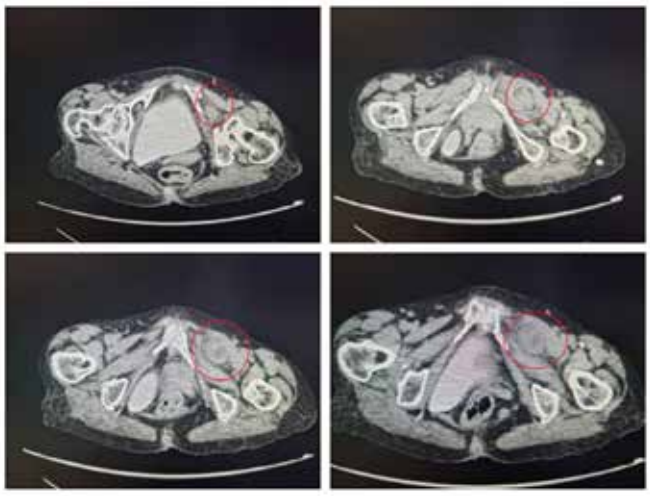

Figure 1 - Abdominal tomography without contrast showing left obturator foramen hernia.

It was then decided to submit the patient to an exploratory laparotomy. A supra and infraumbilical incision was made. The cavity inventory showed the presence of a jejunal loop at 120 cm from the angle of Treitz, herniated through the left obturator foramen, non-reducible and with signs of necrosis. There was no evidence of free fluid in the abdominal cavity. Due to the impossibility of reducing it, opening of the space of Retzius was performed with resection of the herniated loop (approximately 30 cm). Due to the absence of contamination of the abdominal cavity and the hemodynamic stability of the patient, an end-to-end enteroenteric anastomosis was performed with suture in two planes using non-absorbable 3-0 polypropylene thread. After anastomosis, the obturator foramen failure was corrected with the placement of a thick marlex mesh anteriorly to the obturator foramen in the space of Retzius and posteriorly to the obturator foramen, anteriorly to the peritoneum. Peritoneum was closed with absorbable polyglactin thread. The patient had a good clinical evolution and was discharged 7 days after admission.

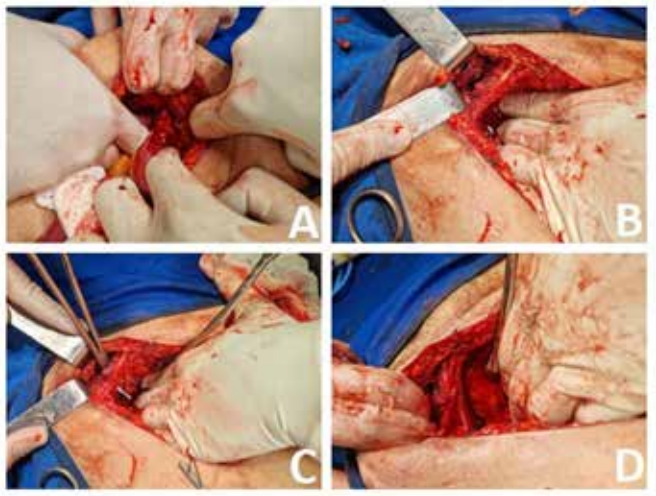

Figure 2: A - Jejunal loop herniated in the left obturator foramen with sign of distress. B - Retzius space dissection with subsequent non-viable jejunal follow-up enterectomy. C - Identification of failure in the obturator foramen. D - Final appearance after placing the marlex mesh anteriorly and posteriorly to the obturator foramen.

DISCUSSION

Obturator foramen hernia is a rare entity whose main predisposing factors are female sex, advanced age, low BMI associated with factors that lead to increased intra-abdominal pressure such as COPD, chronic constipation and multiparity.[4] The patient referred to in this case was a female, had advanced age, multiparity (11 pregnancies) and reports of chronic constipation as risk factors.

It is a pathological entity of difficult clinical diagnosis, being commonly performed only during abdominal inspection during exploratory laparotomy procedure due to an obstructive acute abdomen.[3] In a smaller percentage of cases, its diagnosis can be made by complementary exams. , such as computed tomography of the abdomen, as occurred in the case reported.

The surgical approach indicated in the emergency is wide exploratory laparotomy, as it allows a better inspection of the abdominal cavity with better identification of the hernia, its reduction or resection in case of non-viability of segments, as in the case presented. The laparoscopic approach can be performed, being more indicated in a context outside the emergency, in patients who have not yet progressed to frank obstructive conditions and with possible suffering from intestinal loops.[5,6]

The repair indicated for the failure of the obturator foramen is performed with synthetic meshes. Their use is not recommended in the case of intestinal gangrene or perforation. In the case presented here, we chose to place a synthetic mesh despite the fact that the herniated segment showed signs of necrosis because there were no signs of contamination of the abdominal cavity or the space of Retzius. One option for such cases is to close the gap with a purse-string suture using non-absorbable threads or interrupted sutures.[1]

CONCLUSION

The obturator foramen hernia presents a clinical picture of difficult diagnosis and high morbidity and mortality for the patient if its identification and correct approach are not performed early. The importance of this case is due to the fact that it occurred in a less frequent anatomical region (obturator foramen on the left) associated with a clinical picture of obstructive acute abdomen with the first imaging test suggesting left inguinal hernia, not being corroborated by physical examination. There is still no consensus in the literature on the best way to approach the failure in the obturator foramen, with the option of closing with a synthetic fabric mesh. Attention should always be paid to this etiology in cases of obstructive acute abdomen without a defined cause. Its early identification and treatment promotes a considerable reduction in the morbidity and mortality of this pathological entity.

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CASE REPORT

TRANSCATHETER AORTIC VALVE REIMPLANTATION (TAVI) VALVE IN VALVE: CASE REPORT

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ABSTRACT

Introduction: The advent of transcatheter aortic valve replacement (TAVI) has changed the paradigm for managing aortic valve disease. TAVI has become specific in the last decade as a less invasive treatment alternative to the conventional surgical procedure, for inoperable, medium and high surgical risk patients. Objective: to report a case of a previous patient who underwent TAVI 5 years ago due to severe symptomatic aortic stenosis that evolved with degeneration of the prosthesis, progressing with implicit worsening of the functional class, undergoing TAVI valve-in-valve (ViV). Case report: This is a patient with multiple comorbidities who underwent TAVI in 2016 due to severe aortic stenosis. She returned to the hemodynamics service with dyspnea at rest, orthopnea, paroxysmal nocturnal dyspnea and typical precordial pain. Echocardiogram of aortic prosthesis dysfunction with severe stenosis. So, it was decided to perform TAVI ViV to treat an elderly patient with severe organic fragility and high surgical risk. Intraoperatively, a reduction in transvalvular gradients was evidenced. Postoperatively, the patient evolved with significant improvement in her previous symptoms and was discharged after 3 days for outpatient follow-up. Conclusion: The ViV procedure is a safe and less invasive alternative for the treatment of dysfunctional bioprostheses. The current literature reports low morbidity and mortality rates of patients related to ViV improvement and survival.

KEYWORDS: TRANSCATHETER AORTIC VALVE REPLACEMENT, AORTIC VALVE STENOSIS, HEMODYNAMICS.

INTRODUCTION

Degenerative calcific aortic stenosis (AS) is more common in the elderly population, with a predominance of males. It is present in 29% of individuals over 65 years of age[1].

According to the ESC (European Society of Cardiology) and the ACC (American College of Cardiology) guidelines, surgical aortic valve replacement (SAVR) was the standard treatment for symptomatic AS until 2017. However, after publication of the CoreValve High Risk trial and other randomized studies, it is recommended that the decision and choice of treatment for AS should be based on a multidisciplinary discussion involving the Heart Team. Currently, it is recommended for symptomatic patients with severe AS and age over 80 years or for younger patients with life expectancy < 10 years and no anatomic contraindications to transcatheter aortic valve implantation (TAVI), this is recommended in preference to Conventional Aortic Valve Replacement surgery (SAVR) for patients at high surgical risk (Degree of Recommendation I, Level of Evidence A) or intermediate (Degree of Recommendation IIa, Level of Evidence B) [2].

In this context, the TAVI method was developed in 2002 by Alan Cribier, a minimally invasive procedure, as an effective and safe alternative in the surgical treatment of patients with severe symptomatic AS and with restrictions to the procedure by SAVR (contraindication to surgical aortic valve replacement due to high risk surgery or technical conditions that make the surgery unfeasible, such as porcelain aorta, previous thoracic radiation, and others) [3,4].

TAVI-in-TAVI was initially used in the acute management of suboptimal bioprosthesis function during a TAVI procedure. However, with the technological advances of the devices and the initial experience of TAVI-in-TAVI, there was an expansion of the potential indications and use for the correction of degenerated prostheses [5].

The present report aims to describe the performance of a case of TAVI-in-TAVI five years after the original implant. Approval was obtained from the Research Ethics Committee of Hospital de Urgências de Goiânia, under CAAE: 85497418.2.0000.0033.

CASE REPORT

A 77-year-old female patient with multiple comorbidities was admitted to the hemodynamics service in April 2021 for TAVI ViV. It is important to mention that in 2016 this patient underwent TAVI due to severe AS with implantation of the Edwards XT® 23 mm prosthesis.

She recently evolved with dyspnea at rest, orthopnea, and paroxysmal nocturnal dyspnea associated with typical chest pain on minimal exertion.

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Transthoracic echocardiogram (TTE) performed on 12/23/2020 showed severe AS (peak gradient of 51 mmHg, mean gradient of 28 mmHg, peak velocity of 4.5 m/s and valve area of 1.0 cm^2), aortic insufficiency, high probability of PAH (PASP 75 mmHg), preserved left ventricular (LV) systolic function (LVEF 66%) and significant LV diastolic dysfunction.

In view of these circumstances, we chose to perform ViV because she was a symptomatic patient (stage D1), an elderly woman with marked organic fragility and high surgical risk (STS score, mortality 4.4 and morbidity 22%), but with life expectancy longer than one year regardless of the AS.

Preoperatively, the patient underwent cardiac catheterization with manometry that revealed the presence of a pressure gradient between the left ventricle and the aorta; divergent pressure in aorta; prosthesis in incompetent aortic position with double lesion (important insufficiency and stenosis); predominance of stenosis with a transprosthetic gradient of 50 mmHg and coronary circulation with mild obstructive lesions (Figures 1A and 1B).

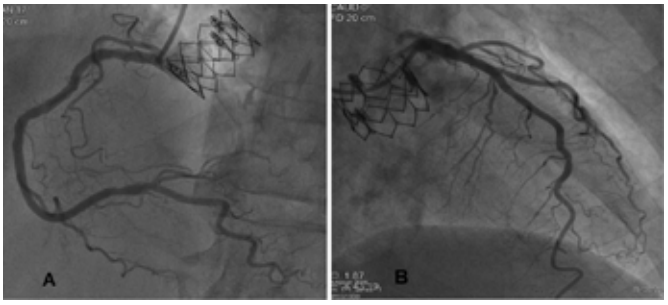

Figure 1: Coronary angiography of 02/25/2021 showing right (A) and left coronary circulation (B) with mild obstructive lesions.

In view of this situation, on 04/12/2021, the Sapien® 23 mm prosthesis was implanted without clinical or angiographic complications (Figures 2A and 2B), whose post-procedure manometry showed a significant reduction in the pressure gradient between LV and aorta from 38 mmHg to 8 mmHg.

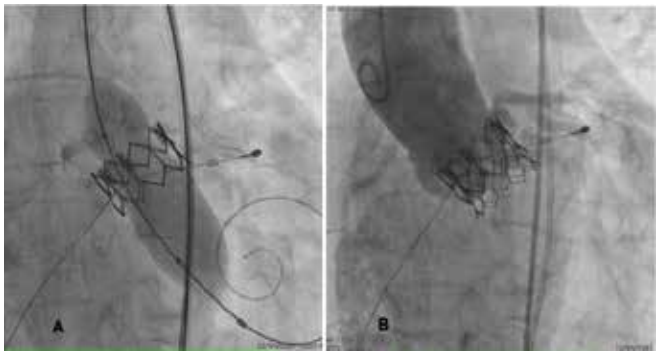

Figura 2: Implante de prótese Evolut R® 23 mm – A: pré-intervenção e B: pós-intervenção

On 04/13/2021, she underwent postoperative control TT ECHO, which showed aortic biological prosthesis with good mobility of the leaflets, with peak left ventricle-aorta (LV-Ao) gradients of 33 mmHg and mean of 18 mmHg; moderate dilatation of the left atrium (indexed LAV of 38ml/m2) (Figures 3A and 3B).

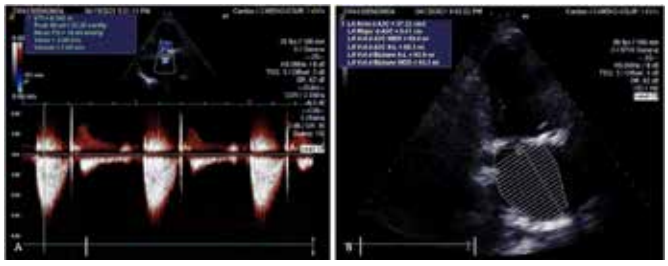

Figure 3: Doppler echocardiogram tracings. A – Doppler evidencing left ventricle (LV) - Aortic (Ao) gradients. B – Doppler demonstrating moderate dilatation of the left atrium (LA)

With good clinical evolution and significant improvement in previous symptoms, the patient was discharged on 04/17/2021 for outpatient follow-up.

DISCUSSION

With the increase in survival of this population treated with TAVI, a progressive increase in a portion of patients who develop implanted valve dysfunction is observed 6,7.

Thus, ViV for replacement of degenerated surgical aortic bioprostheses is a very interesting technique due to the high risk associated with surgical valve replacement in elderly patients [5].

In several meta-analyses published between 2018 and 2021 comparing ViV with Redo-SAVR, there was no significant difference in perioperative or late mortality between the groups, with lower rates of permanent pacemaker implantation, shorter hospital stay in the ViV group [8-10].

In the Global Valve-in-Valve Registry including 202 patients with degenerated bioprostheses, there were no significant differences in mortality between two types of prostheses (CoreValve and Edwards-SAPIEN), major vascular complication or stroke at 30 days and 1-year survival. The implantation of Edwards-SAPIEN models, however, was an independent predictor for high post-procedural gradients (p: 0.02)[11].

Takagi et al9 emphasize in their study that in patients with degenerated aortic valve bioprostheses, especially elderly or high-risk patients, VIV-TAVI may be a safe and viable alternative to Redo-SAVR. They cite, and the authors of the present case report agree with this statement, that the publications so far involve only observational studies with important differences in the baseline characteristics of the patients studied, making it necessary to carry out randomized clinical trials to elucidate this knowledge gap.

CONCLUSION

The ViV procedure is a new, promising, safe and less invasive alternative for the treatment of dysfunctional bioprostheses that has shown low morbidity and mortality rates, being a possibility that may change the indication of prosthesis selection in the initial procedure, favoring biological prostheses. Therefore, we emphasize the need for randomized studies to determine the efficacy and safety of the ViV procedure in patients with aortic prosthetic valve dysfunction.

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CASE REPORT

OCCLUSION OF PATENT FORAMEN OVALE AFTER MYOCARDIAL INFARCTION DUE TO CORONARY ARTERY EMBOLISM. CASE REPORT

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ABSTRACT

Patent foramen ovale is a congenital heart disease prevalent in about 30% of the adult population and is associated in the genesis of ischemic cerebrovascular events, peripheral arterial occlusions, and less commonly, with acute coronary syndrome. Paradoxical coronary artery embolism is considered rare, being an underdiagnosed cause of acute myocardial infarction in patients with a low risk profile for coronary heart disease. We report a case of acute myocardial infarction with ST-segment elevation of the anterior wall due to presumed paradoxical embolism with subsequent percutaneous occlusion of the patent foramen ovale.

KEYWORDS: FORAMEN OVALE, PATENT; EMBOLISM, PARADOXICAL; MYOCARDIAL INFARCTION; SEPTAL OCCLUSION DEVICE; HEART DEFECTS, CONGENITAL

INTRODUCTION

From the end of the fourth week of embryonic development, the primitive atrium divides into right and left atria by the fusion of two septa, the septum primum and the septum secundum, the latter forming an incomplete division between the atria, receiving the denomination of foramen ovale[1]. It consists of an embryonic formation essential for the maintenance of fetal circulation, since it allows the continuous diversion of oxygenated blood from the right atrium to the left atrium[2]. After birth, due to the increase in pressure in the left atrium, due to the increase in pulmonary venous return, the septum primum is pressed against the septum secundum, adhering to it, succeeding the closure of the foramen and the formation of the fossa ovale. When, for some reason, this fusion does not happen or it occurs inappropriately, we characterize the patent foramen ovale (PFO)[1].

Considered a common abnormality, the prevalence of foramen ovale patency in the adult population is approximately 30%3. The PFO is functionally closed most of the time and in most cases it does not cause systemic repercussions, and may go undetected [4,5]. However, in situations such as deep inspiration, coughing and Valsalva maneuver where the pressure of the right atrium exceeds that of the left atrium, the PFO opens, allowing the passage of emboli from the venous to the systemic circulation, establishing a paradoxical embolism (PE)[4,6]. Deep venous thrombosis (DVT) of the lower limbs is an important emboligenic source, but other mechanisms have been suggested, such as the formation of the thrombus in the foramen itself due to blood stasis[2].

Two diagnostic methods can be used to document PFO: transesophageal echocardiography (TEE), traditionally considered standard, and transcranial Doppler, both sensitized with the bubble study and Vasalva maneuver[7]. In TEE, in patients with PFO, at least three microbubbles should be visualized inside the left atrium, between the third and fifth cardiac cycle after maximum contrast opacification in the right atrium. In transcranial Doppler, the test is confirmed when a hyperintense signal is observed within 10 seconds after infusion of the stirred saline solution8. However, since most patients are asymptomatic, the diagnosis may occur as an occasional finding in tests requested for another purpose[4,5].

In 1877, pathologist Julius Cohnheim hypothesized that the passage of a paradoxical embolism through the PFO could be the cause of a cerebrovascular accident (CVA)[2]. Since then, numerous statistical analyzes have been published demonstrating PFO as an important risk factor for ischemic stroke, especially in patients younger than 55 years of age and with no other apparent cause to trigger the insult9. Although the ischemic cerebrovascular event

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is the predominant clinical manifestation of PE, a fact that can be explained by the predisposition of embolisms to reach the cerebral arteries due to anatomical aspects and the distribution of blood flow in the aortic arch, cases of peripheral arterial occlusions, and less commonly, acute coronary syndrome (ACS) are reported in the literature[5].

The aim of this study is to report a case of acute myocardial infarction (AMI) with ST-segment elevation of the anterior wall due to presumed paradoxical embolism and subsequent PFO occlusion using a percutaneous device.

The Research Ethics Committee of the Hospital de Urgências de Goiânia, linked to Plataforma Brasil, approved the present study (CAAE: 52695421.2.0000.0033).

CASE REPORT

A 48-year-old male, married patient sought care in the cardiology emergency department after waking up at night due to severe tight chest pain accompanied by nausea, vomiting and profuse sweating. In his previous medical history, he reported having systemic arterial hypertension and being treated with olmesartan. He denied other known comorbidities and has no family history of cardiovascular disease.

On physical examination, he was conscious, oriented, cutaneous-mucous pallor, diaphoretic, eupneic, without adventitious sounds on respiratory auscultation and oxygen saturation of 98% on room air. Blood pressure 90/70 mmHg, heart rate 63 beats per minute, regular heart rhythm, two beats, normophonetic sounds, no murmurs. The peripheral pulses of the four limbs were palpable and symmetrical.

The electrocardiogram (ECG) showed ST-segment elevation in leads V2 to V4, establishing the diagnosis of AMI in the anterior wall in the hyperacute phase (Figure 1).

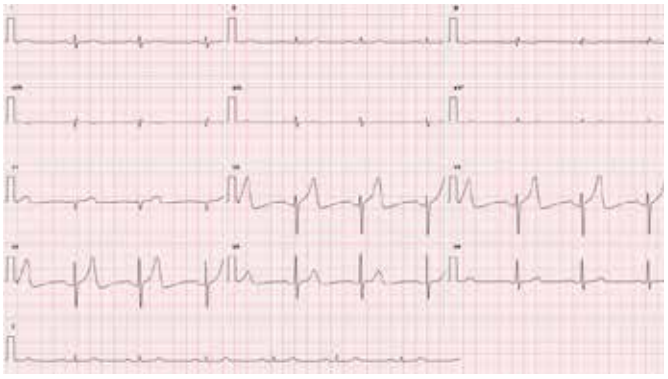

Figure 1. Electrocardiogram image on admission showing ST-segment elevation leads V2 to V4.

In view of the ECG, an urgent coronary angiography was performed, which showed obstruction with high thrombotic load in the proximal third of the anterior descending artery and absence of atherosclerotic lesions (Figure 2 A and B). Intracoronary thrombus aspiration was performed with a catheter associated with an intracoronary glycoprotein IIb/IIIa inhibitor (Tirofiban®) and alteplase (Actilyse®), in addition to heparinization and primary balloon angioplasty. At the end, Thrombolysis in Myocardial Infarction (TIMI) III flow was obtained. Procedure performed without clinical or angiographic complications (Figure 2 C).

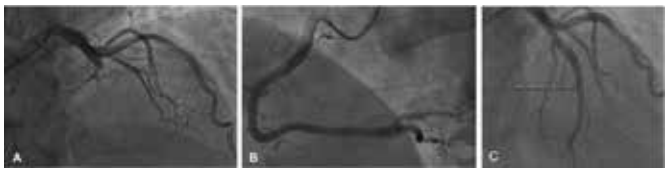

Figure 2. Coronary angiography image. A: total obstruction in the proximal third of the anterior descending artery with no atherosclerotic lesions. B: dominant right coronary artery, with normal angiographic appearance. C: Post-intervention result. Image of residual thrombus is observed, but without flow impairment.

Due to the angiographic appearance with the absence of atherosclerotic plaques, the medical approach consisted of keeping the patient anticoagulated and proceeding with the etiological investigation for possible causes of non-atherothrombotic AMI. The following were performed: lower limb Doppler without evidence of DVT; hematological investigation for thrombophilia without alterations; transesophageal echocardiogram (TEE) with normal ventricular function, contractility and valves, in addition to the absence of thrombi in the left atrial appendage. The infusion test with agitated saline solution was performed, showing the passage of numerous microbubbles from the right atrium to the left atrium during the Valsalva maneuver, compatible with the diagnosis of PFO (Figure 3).

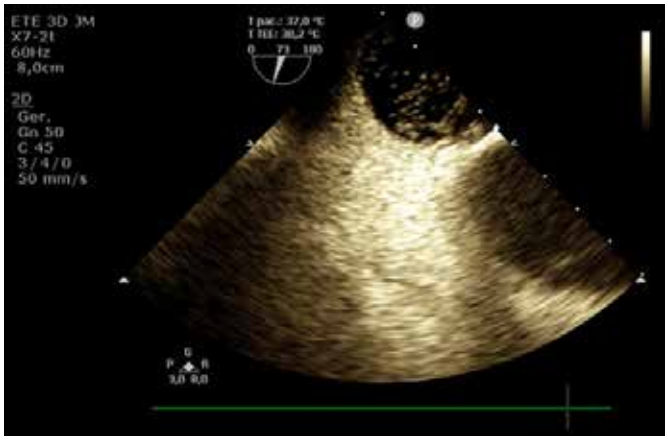

Figure 3. Transesophageal echocardiogram image showing microbubbles crossing from the right atrium to the left atrium through the patent foramen ovale.

There being no other justification for the occurrence of AMI, paradoxical embolism was presumed. Forty-five days

after the infarction and after discussion with the Heart Team, it was decided to perform a new catheterization, which showed a normal angiographic appearance, followed by closing the PFO with an Occlutech® prosthesis, guided by three-dimensional TEE, with an excellent final result (Figure 4 A, B and C).

Figure 4. A and B: Three-dimensional transesophageal echocardiographic image guiding the opening and positioning of the prosthesis. C: Fluoroscopy image demonstrating the prosthesis after its release for PFO closure.

The patient was discharged from the hospital with dual antiplatelet therapy. A control transthoracic echocardiogram was performed one week after the intervention, demonstrating adequate positioning of the prosthesis and absence of residual shunts.

DISCUSSION

In the present case, what stands out is the correlation between AMI in a middle-aged patient with intermediate cardiovascular risk, with PFO and the therapeutic approaches adopted, emphasizing the presumed diagnosis of PE.

It has been recognized since the 1970s that coronary atherosclerosis is the main cause of ACS, however a variety of causes of non-atherosclerotic AMI have been described, including arteritis, Takotsubo cardiomyopathy, aortic stenosis and insufficiency, trauma, spasm, dissection, congenital anomalies and coronary artery embolization10. Paradoxical coronary artery embolism is considered uncommon, representing approximately 10 to 15% of all paradoxical emboli, and in patients younger than 35 years, it corresponds to 25% of acute coronary events[6].

Usually, the clinical and electrocardiographic characteristics are similar to those with classic obstructive atherosclerotic disease10. As a result, PE is, in general, an underdiagnosed cause in individuals with AMI, and should be considered in patients with acute AMI and with a low risk profile for coronary artery disease (CAD)[6]. There are no differences in the approach to AMI by PE in the emergency department. The identification of ST-segment elevation on the ECG associated with the presenting signs and symptoms should trigger a rapid assessment to define the reperfusion strategy10. In this context, the treatment of choice is manual aspiration of the thrombus, followed or not by angioplasty and stent placement associated with antiplatelet agents[6].

The use of a glycoprotein IIb/IIIa inhibitor receives a grade IIb/IIIa recommendation by the American College of Cardiology and American Heart Association (ACC/AHA) guidelines committee for those with ST-segment elevation AMI, especially those with extensive anterior wall and/or large thrombotic burden[11]. On the other hand, the use of thrombolytics via the intracoronary route, in turn, is not routinely recommended due to their potential risk of causing bleeding and the lack of studies demonstrating their effectiveness, and their use is currently restricted to exceptional cases[12]. In the case presented here, in view of the catastrophic situation with high thrombotic load in the proximal third of the left anterior descending artery, we chose to combine drugs with manual thrombectomy, however, we know that the success of the procedure cannot be attributed to such conduct and we recognize that this can be a confounding factor.

The definitive diagnosis of PE is made by autopsy or by direct visualization of the passage of the thrombus through the PFO during echocardiography, being considered a rare diagnosis[13]. However, PE can be considered a presumed cause of infarction when in the presence of a right-to-left shunt and the following conditions are ruled out: coronary artery spasm, atrial fibrillation, vascular and myocardial disease, vasculitis, atherosclerosis and intracavitary thrombus[5]. In our case, it was the most likely cause of AMI, considering the presence of a thrombus in the left anterior descending artery, atherosclerotic plaques not apparent on coronary angiography, tests for hematological disorders without alterations, ultrasound study of normal lower limb veins and exclusion of others. possible causes, in addition to the presence of PFO. We recognize as a limitation of the study the lack of complementary intracoronary imaging to strengthen the diagnostic conclusion of the absence of atherosclerotic plaque, ulcer or endothelial rupture. However, it is known that the panorama of health services in Brazil presents different scenarios and different realities, in addition to regional inequalities with regard to accessibility to more complex services. Thus, a careful analysis of each case individually is necessary, so that there is a balance between the risks and benefits involved in the percutaneous closure of PFO, given its high prevalence in the population.

The indications for secondary prevention in patients with PFO are still the subject of extensive debate. The therapeutic arsenal available includes antiplatelet aggregation, oral anticoagulation and percutaneous or surgical PFO closure. With the advent of the percutaneous approach, the surgical procedure is reserved for selected cases. The results obtained with percutaneous treatment in published series describe results equivalent to those obtained through surgery with the advantage of lower occurrence of complications[14].

There is scientific evidence that percutaneous occlusion in PFO is safe and beneficial in preventing the recurrence of ischemic cerebrovascular events in patients with cryptogenic stroke[3]. In a meta-analysis study published in 2020, involving six randomized trials and 3750 patients, PFO closure demonstrated superiority in reducing the

rate of recurrent stroke over medical therapy alone - risk ratio of PFO closure versus medical therapy of 0.37; 95% confidence interval, 0.17 to 0.78; p=0.01. The occlusion procedure has been implicated in an increased risk of atrial fibrillation[15].

The reference standard for secondary prevention remains controversial and the decision must be individualized and shared, considering age, risk of recurrence of ischemic events, adverse events, long-term clinical consequences and patient preferences[15]. In cases where the outcome was AMI, there are no studies with strong evidence demonstrating that these patients would also benefit from this strategy, however, most authors suggest that the occlusion of the venoarterial communication should follow the same indications[6].

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CASE REPORT

CONGENITAL CYTOMEGALOVIRUS INFECTION

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ABSTRACT

Cytomegalovirus (CMV) is a virus that can cause vertical transmission.

Pregnant women can be affected by a primary infection or secondary to reactivation or reinfection. After a primary infection or virus it can become latent in white cells with periodic reactivations and there is also the possibility of reinfection with a new strain since an initial infection does not guarantee cross-immunity.

Newborn (NB) CMV carriers may be affected in the future by neurological disorders in childhood and deafness. In the present work, we teach a case report of a male NB with probable CMV infection whose mother had tests compatible with immunity to the virus. It opens up possibilities for discussing other forms of transmission for the concept of a previously immune pregnant woman.

KEYWORDS: REINFECTION, REACTIVATION, VERTICAL TRANSMISSION

INTRODUCTION

Cytomegalovirus (CMV), of the Herpesviridae family, is the most common agent of congenital infection in humans. After a primary infection, the virus evades the host's cellular immunity, making the infection persistent or latent throughout life, with periodic reactivations being frequent. There is also the possibility of reinfection with different viral strains since cross-immunity does not occur [7].

In Brazil, the prevalence of congenital CMV infection at birth was 1% in a population with high maternal seroprevalence, that is, greater than 97%. [7]

In women not immune to CMV, primary infection results in transmission in about 33% of fetuses. Women with CMV immunity before conception transmit the virus to 1-2% of fetuses. [7]

CMV appears to be transmitted efficiently in all trimesters of pregnancy [2].

According to the National Registry of Symptomatic CMV Infection in the United States (CDC), the disease is defined by the simultaneous presence of three criteria: presence of one or more clinical signs (small for gestational age, petechiae, hepatosplenomegaly, microcephaly, among others); Detection of CMV in saliva, urine or other clinical specimens and the exclusion of other etiologies that cause abnormalities (syphilis, toxoplasmosis and congenital infections, respectively). [4]

For diagnostic purposes, the presence of specific IgG anti-CMV antibodies in the serum of a previously negative pregnant woman confirms a primary maternal infection. Within the context of maternal primary infection, we have the diagnosis of anti-CMV serum IgM detection, which reveals three different conditions: acute phase of primary infection (from one to three months after infection); convalescent phase of primary infection, which is more common when there is a decline in IgM levels and persistence of IgM antibodies, usually occurring more than three months after the onset of primary infection. Additionally, when the presence of anti-CMV IgM in the serum of a pregnant woman is not sufficient to identify a primary infection, it is suggested that an antibody avidity test be performed [8].

The detection of anti-CMV serum IGM in NB is suggestive of congenital infection by this virus, but it must always be confirmed by its detection in urine and/or saliva. [4]

CLINICAL CASE

NB admitted to the neonatal ICU of SANTA CASA in Anapolis, coming from Goianésia-Goias, gestational age by the Capurro method of 35 weeks and 3 days old, birth weight -1800g, male, who underwent emergency cesarean section in that city due to acute fetal distress. Report of being born bathed in meconium amniotic fluid, with Apgar 5/8; physical examination also presented

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jaundice, diffuse petechiae and hepatosplenomegaly (liver at 5 cm from the RCM and spleen at 4 cm from the LCM). Evolved with progressive respiratory distress. Admitted to the ICU at 8 hours of life, in respiratory failure, started mechanical ventilation, vasoactive drugs and antibiotic therapy to cover early neonatal sepsis.

The NB is the son of young, healthy, non-consanguineous parents, with no history of heredofamilial pathology. First pregnancy, the mother had 2 prenatal consultations. Ten days before delivery, she remained hospitalized for 1 week for treatment of a urinary tract infection (UTI). Maternal examinations and obstetric ultrasounds were within normal limits.

	1st Trimester	3rd Trimester	Reference value
Toxoplasmosis (IGG)	>200 UI/mL		Reagent >10ui/Ml Non-Reagent <10ui/mL
Rubella (IGG)	12,31 UI/mL		Reagent >10ui/Ml Non-reagent <10ui/mL
Cytomegalovirus Inclusion Disease	4,51 UI/mL		Reagent >0,5ui/Ml Non-reagent <0,5ui/mL
Syphilis	Non-reagent	Non-reagent	
AIDS	Non-reagent	Non-reagent	
Chagas	Non-reagent		
Hepatitis B	Non-reagent		
Hepatitis C	Non-reagent		
HTLV	Non-reagent		

Table 1- Serology performed by the pregnant woman.

Serologies were requested to clarify the clinical picture, according to the table below (Table 2):

Date	17/11	18/11	19/11	20/11	21/11	22/11
Hb	6,0	9,9	7,17	9,49	8,4	7,34
Ht	16,1	26,4	20	24,8	21,3	19,2
Plq	23600	17000	15000	14000	13000	17200
Leuc	25400	9600	4800	4500	12200	5400
Citomegalovirus			IGM-4,5 IGG-108,7			
VDRL			NON-REAGENT			

Table 2 – Serology performed on the patient from November 17 to 22.

He evolved with anemia, thrombocytopenia and hemodynamic instability in continuous use of vasoactive drugs and died on the seventh day of hospitalization.

DISCUSSION

We report the case of an NB with a clinical picture compatible with congenital neonatal infection. The mother had prenatal exams with immunity to rubeola, toxoplasmosis and CMV. According to the National Registry of Symptomatic CMV Infection in the United States (CDC), the disease is defined by the simultaneous presence of three criteria: presence of one or more signs (small newborn with gestational age, petechiae, hepatosplenomegaly, microcephaly, among others); Detection of CMV in saliva, urine or other clinical specimens and the exclusion of other etiologies that cause abnormalities (syphilis, toxoplasmosis and other congenital infections) [4]. In the case discussed, the NB had petechiae, cholestatic jaundice, hepatosplenomegaly, thrombocytopenia and from the serologies requested in the NB, the presence of CMV reagent IgM and IgG was observed. The detection of anti-CMV serum IgM in newborns is suggestive of congenital infection by this virus, but it should always be confirmed by its detection in urine and/or saliva [4]. There was no time to collect CRP in urine/saliva.

It is important to discuss the possibilities of vertical transmission of CMV in a mother with previous immunity.

The main mechanisms for non-primary infection include reactivation in an existing persistent infection or reinfection with a new strain of CMV [5].

On reactivation, in the latency period, there is the activation of CD3-4 and HPCs, which participate in the activation of the immune system, contemplating molecules

such as hematopoietic stem cells, pluripotent, myeloid progenitor, myeloblast, monocytes, macrophages and dendritic cells, leading to a quiescent or latent infection through superior activation of monocytes, which leads to an acute or chronic infection, in epithelial or dendritic cells and macrophages [3].

Regarding reinfection, it is known that there is no cross-immunity to other strains, being a risk factor for vertical transmission by the reinfected pregnant woman. [6]

CONCLUSION

This work presents a picture compatible with vertical transmission of CMV in a pregnant woman who had prenatal exams with immunity to the virus. The possibilities of fetal involvement due to non-primary causes, such as reactivation or reinfection, were discussed. With this, there is a need for greater attention for pregnant women in prenatal care in relation to preventive care, since even with a pre-existing immunity, contagions can occur. The reflection of the entire health team about this problem may prevent some children from having disabilities in the future, especially hearing impairments. In the meantime, we continue to look forward to the large-scale development and use of the vaccine.

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REVIEW ARTICLE

SURGICAL TREATMENT OF GYNECOMASTIA

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ABSTRACT

Gynecomastia is the growth of the male mammary gland resulting from secondary branching of the ducts and proliferation of the fibroelastic stroma, usually resulting from an imbalance between the estrogenic stimulating action and the androgenic inhibitory effects. Differential diagnosis should be made with breast carcinoma, dermoid cyst, hematoma, lipoma, lymphangioma and neurofibroma. The treatment of choice is surgical since drugs such as clomiphene, tamoxifen, danazol and testolactone have low efficacy. Reducing adenomastectomy with an inferior periareolar incision is the technique of choice. Liposuction should be considered in cases of severe lipomastia.

KEYWORDS: BREAST; GYNECOMASTIA; SURGERY

INTRODUCTION

Gynecomastia is the growth of the male mammary gland due to secondary branching of the ducts and proliferation of the fibroelastic stroma. In most cases, it seems to result from an imbalance between estrogenic stimulating action and androgenic inhibitory effects[1].

The imbalance between estrogen and androgen, pituitary gonadotropins, corticosteroids, prolactin, thyroid and growth hormones may play a role in the origin of gynecomastia[1].

It predominates at puberty and after 65 years of age, especially in overweight or obese people.

LITERATURE REVISION

The macroscopic appearance of glandular tissue in gynecomastia is similar to that of the female breast. It must be distinguished from the increase in volume caused by the accumulation of fat called lipomastia[2].

In the hypertrophic breast, dense, hyaline, periductal and collagenous connective tissue is observed, in addition to hyperplasia of the lining of the ducts and plasma cell infiltrate[3].

A ginecomastia pode ser classificada em:

Gynecomastia can be classified into:

Physiological: when it appears in the neonatal period, puberty, adolescence and senility.

Primary hormonal alterations: Klinefelter syndrome, in which there is a chromosomal alteration (47, XXY). Familial prepubertal gynecomastia which is a rare autosomal dominant disorder due to increased aromatase activity.

Sexual development alterations: Male pseudohermaphroditism, which is characterized by individuals with hypospadias, post-pubertal atrophy of the seminiferous tubules, azoospermia, infertility and gynecomastia.

Non-hormonal clinical conditions: Liver cirrhosis, hyperthyroidism, malnutrition, trauma, tumors, liver and kidney failure.

Induced by drugs and psychoactive substances: Anabolic steroids, estrogens, digitalis, spironolactone, cimetidine, ketoconazole, amphetamine, antihypertensives, antidepressants, cytotoxic agents, alcoholic beverages and illicit drugs (heroin and marijuana).

Diagnosis is based on clinical data, supplemented by mammography and ultrasound. It rarely requires punctures and percutaneous biopsy[4].

Withdrawal of the drug causing gynecomastia or correction of the underlying condition that altered the balance of estrogens and androgens causes regression of gynecomastia, particularly if breast growth is of recent onset.

Therapeutic management is usually expectant in adolescents who have physiological gynecomastia, as many cases regress spontaneously. The parenchyma/fat ratio and breast consistency will help determine the most effective treatment modality. Treatment with antiestrogenic drugs has no scientific evidence[5].

When gynecomastia persists in adults, causing psychological disorders due to aesthetics, surgical treatment with subcutaneous adenomastectomy, with or without liposuction, is the most used method.

Subcutaneous adenomastectomy: The procedure is

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performed in the operating room under local anesthesia with lidocaine without vasoconstrictor, associated with anesthetic sedation. An inferior periareolar incision is made, the subcutaneous flap is made with a scalpel or scissors up to the plane of the pectoralis major muscle, removing the entire mammary gland, preserving the areola and nipple[6].

Figure 1: Gynecomastia in a 14-year-old adolescent.

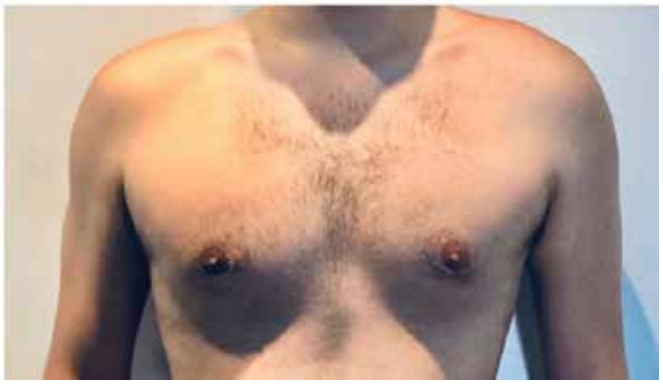

Figure 2: Result after 2 years of surgical treatment of gynecomastia with an inferior periareolar incision.

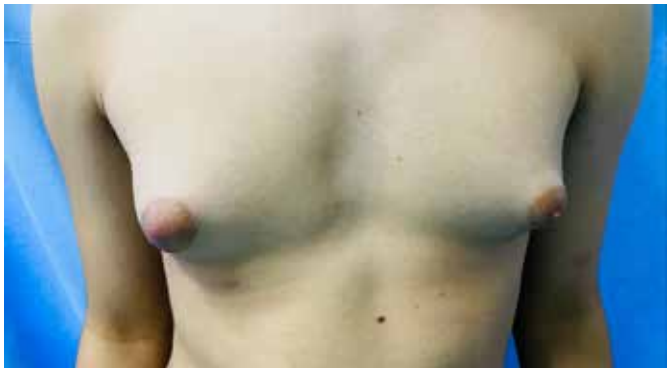

Photo 3: Gynecomastia in a 15-year-old adolescent.

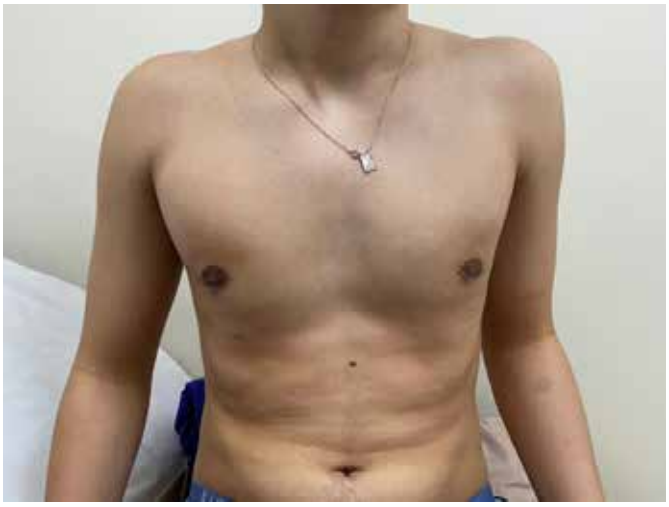

Photo 4: Result after 15 days of surgical treatment with bilateral adenomastectomy with inferior periareolar incision.

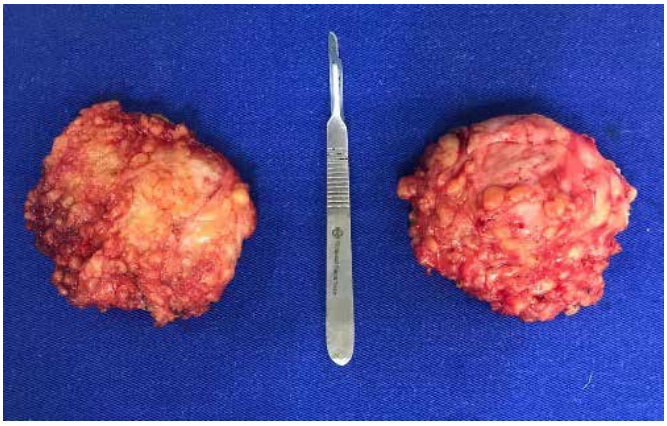

Photo 5: Macroscopic appearance of the surgical specimen of bilateral adenomastectomy for gynecomastia.

CONCLUSION

Healthy men with palpable breast tissue is not uncommon. Thus, these asymptomatic patients should not receive a diagnostic investigation, let alone be treated. Treatment of gynecomastia depends on the underlying cause. Physiological pubertal gynecomastia usually does not require treatment and resolves spontaneously within 3 years in approximately 90% of patients. When gynecomastia is drug-induced, it may regress after drug withdrawal. Surgical treatment with the subcutaneous adenomastectomy technique, using the inferior periareolar incision, is the technique of choice in most cases when gynecomastia persists in adults[5].

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CEREM-GOIÁS

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