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Case Report

Mesenteric thrombosis as a clinical presentation of antiphospholipid syndrome: a case report and literature review

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ABSTRACT

Mesenteric thrombosis is a rare pathology that results from the obstruction of the arterial or venous system of the mesenteric circulation by a thrombus or embolus. The etiology of thrombus formation is rarely evident; the population statistically most affected by this pathology are older adults, with an average age of 60-70 years. The main causes related to the presentation of mesenteric thrombosis are embolic cardiac arrhythmias such as atrial fibrillation, neoplasia and prolonged prostration. In rare cases, the presence of thrombophilia, for example antiphospholipid antibody syndrome (APS), is identified as an underlying cause of thrombotic events. APS is a disease characterized by a procoagulant state; the most frequent clinical manifestations are related to thrombotic events. The pathophysiology of the disease is related to the formation of antiphospholipid antibodies. The three antibodies identified in APS are lupus anticoagulant (LA), anticardiolipin antibody (aCL) and anti-b2glycoprotein 1 antibody (anti-b2GP1). Treatment is based on the use of anticoagulant drugs with the aim of preventing the appearance of new thromboembolic events. In this article we present a clinical case of mesenteric thrombosis in a male patient in whom the presence of antiphospholipid antibodies (aPL) was documented and who met the classification criteria for APS. For the review of the literature, a search was carried out for information related to pathophysiology, clinical manifestations, classification and treatment of APS, as well as literature related to mesenteric thrombosis in open access sources and databases such as PubMed. In conclusion, APS is a rare pathology, probably because it is underdiagnosed, since in developing countries there are no resources to carry out the diagnostic approach, likewise the delay in the start of treatment implies an increase in morbidity for the patient.

Keywords: Thrombosis, Antiphospholipid antibodies, Anticardiolipin antibodies, Anti-b2-glycoprotein 1 antibodies, Lupus anticoagulant, Antiphospholipid syndrome, Thrombophilia

INTRODUCTION

Antiphospholipid syndrome (APS) is considered an acquired thrombophilia, resulting from the formation of antibodies against phospholipids. It can be asymptomatic or cause devastating symptoms. The clinical manifestations are so varied that making a diagnosis can be complicated.[1] APS can be primary, when it is not related to other diseases, or secondary, when it occurs concomitantly with other diseases. It is common for it to occur in people with other autoimmune disorders. The most relevant clinical manifestations are related to thrombotic and obstetric events.[2] As for epidemiology, worldwide prevalence is estimated at 40-50 cases per 100,000 people, with an incidence of 1-2 cases per 100,000 people. Most patients in whom the diagnosis is established are young adults and only 12.5% of cases are people over 50 years of age. The most frequent association with another autoimmune pathology is with systemic lupus erythematosus (SLE).[3] The first descriptions of the disease were made between 1975 and 1985, and in 1983 the first article describing APS and its association with the lupus

anticoagulant and the anticardiolipin antibody, identified in patients with SLE, was published. Since then, the classification criteria have been modified, the most relevant being the Sapporo and Sydney criteria.[4] In 2023, the ACR and EULAR updated the APS classification criteria with the aim of creating criteria with greater specificity for the diagnosis of this pathology.[5]

CASE REPORT

This is a 52-year-old male patient, originally from Yucatan. Among the important pathological history, he mentions a diagnosis of deep vein thrombosis of the left lower limb in 2021, denies being a carrier of chronic-degenerative diseases or other important history, and denies previous surgical interventions.

He goes to the emergency room in March 2023, presenting generalized abdominal pain of 3 days' duration, with irradiation to the left flank, intensity 8/10 on the visual analogue scale (VAS), accompanied by nausea and vomiting; he says that he went to a private doctor who prescribed a non-steroidal anti-inflammatory drug (NSAID) without improvement of the condition. During his evaluation by the medical emergency service, the following findings are documented in his medical record. Vital signs like heart rate=140 bpm, respiratory rate=22 bpm, temperature=36.2°C, blood pressure=140/80 mmHg, oxygen saturation=99%.

The admission laboratories revealed leucocytosis of 32.75×109/l, with neutrophils of 88%. He was evaluated by the general surgery service, who found data of acute abdomen, reason why a simple and contrasted abdominal tomography was requested, in which data suggestive of ischemia (wall edema and intestinal pneumatosis) were observed. It was decided to perform exploratory laparotomy, finding necrosis of 70 cm of terminal ileum and colon, reason why ileocecectomy was performed (Figure 1).

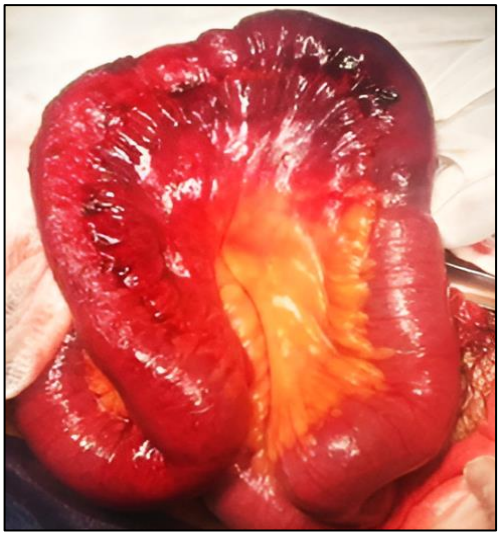

Figure 1: Intestine section with signs of ischemia.

During his follow-up, a consultation with the internal medicine service is requested for malabsorption syndrome and hydro electrolytic imbalances.

During the preparation of the clinical history, it catches our attention that the patient presented a second event of thrombosis, considering that he is a relatively young patient and at the time of the evaluation without documenting an apparent cause, we proceed to initially rule out cardiac arrhythmias that could cause embolisms, without finding any alteration, reason why it is decided to initiate an approach to rule out any type of thrombophilia.

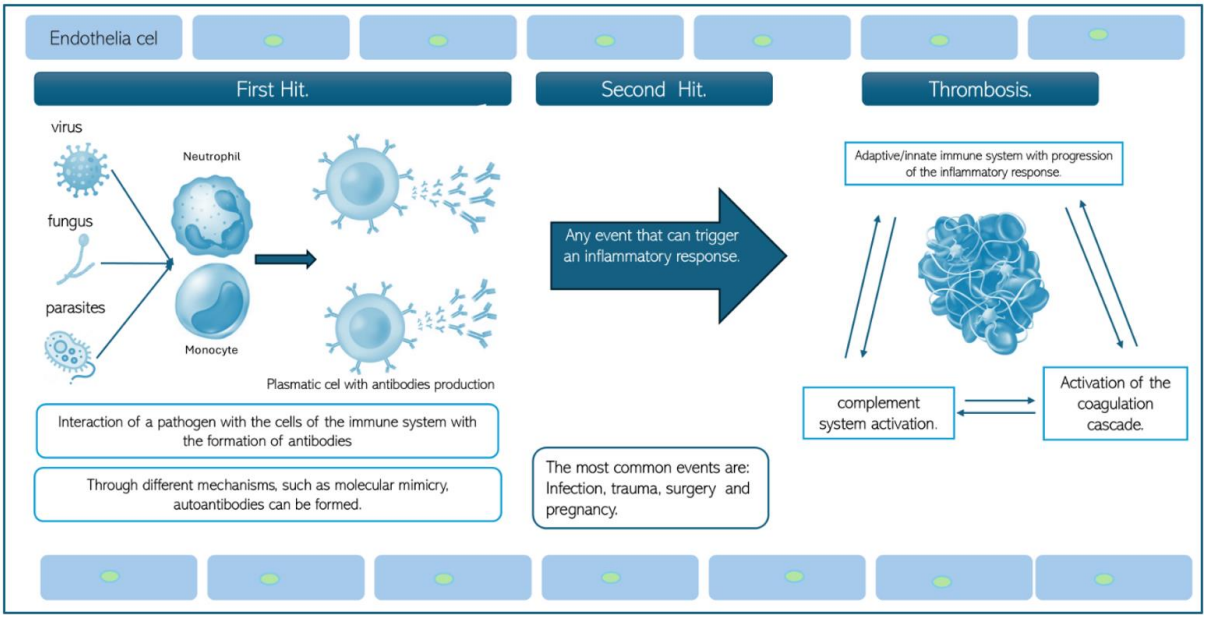

Figure 2: Summary of the double hit theory to explain the pathophysiology of antiphospholipid syndrome.

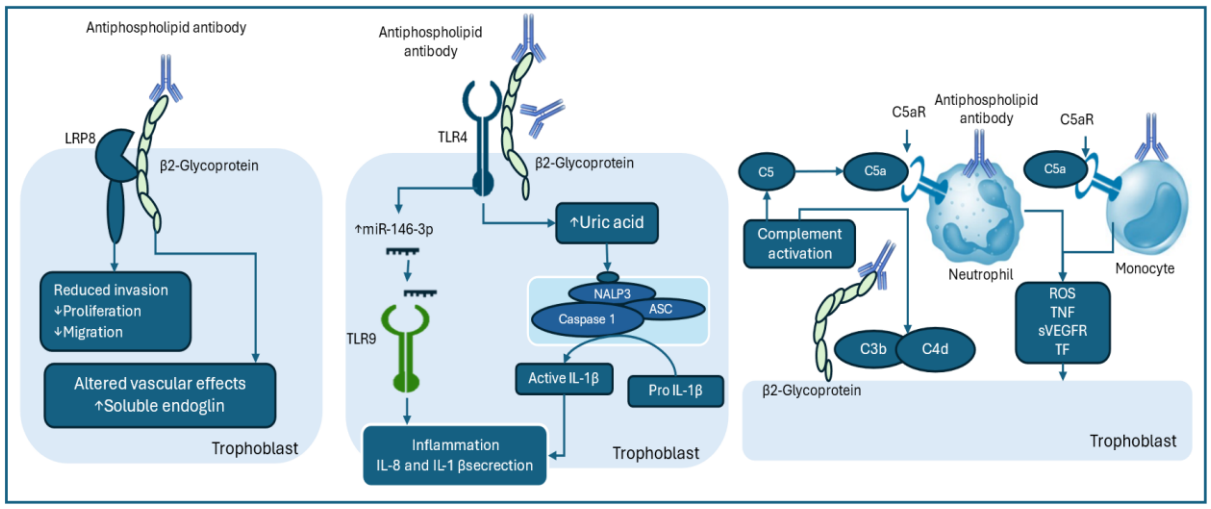

Figure 3: Molecular effects of antiphospholipid antibodies on trophoblasts.

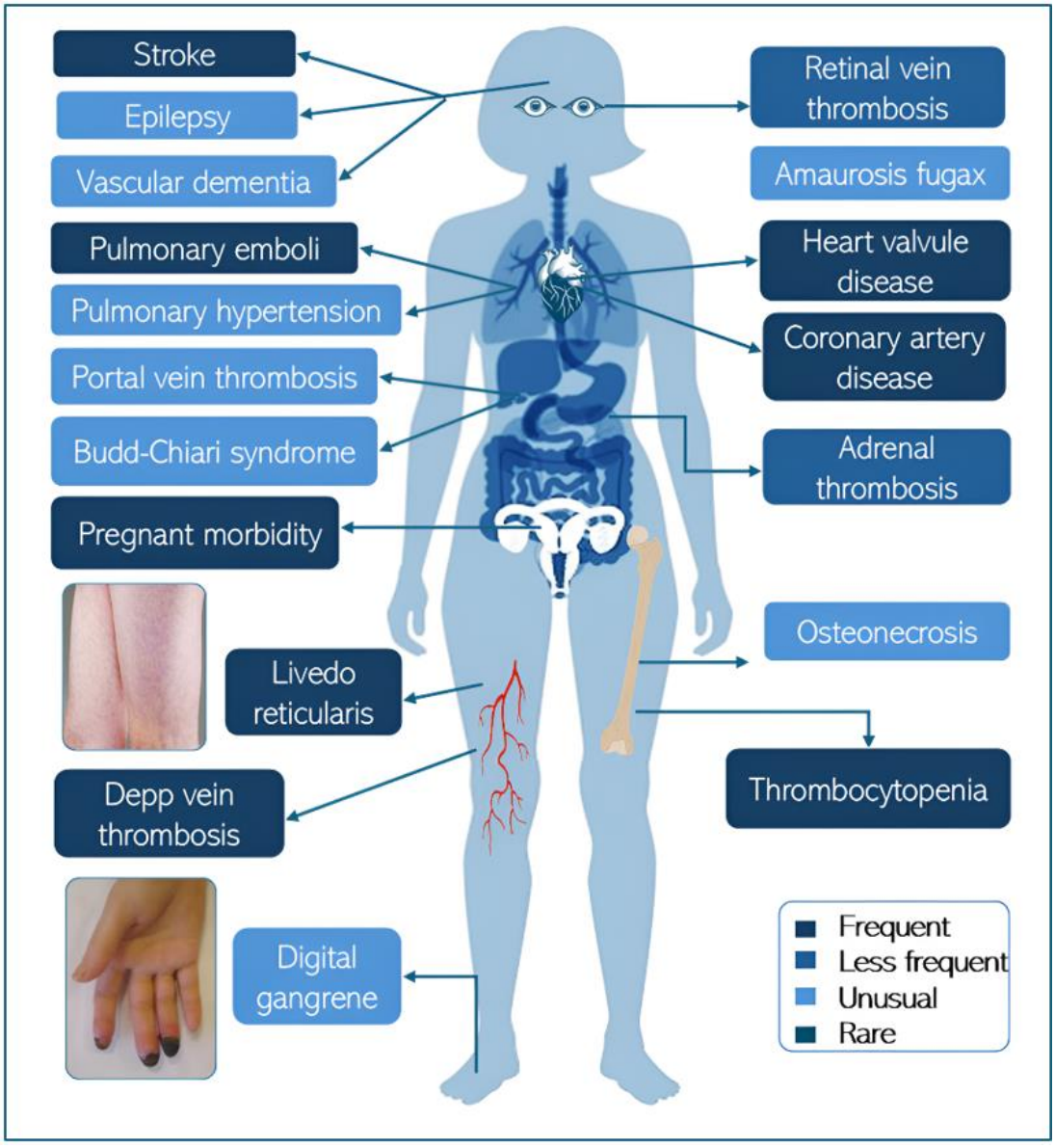

Figure 4: Clinical manifestations of APS; the main clinical manifestations are related to thrombotic events.

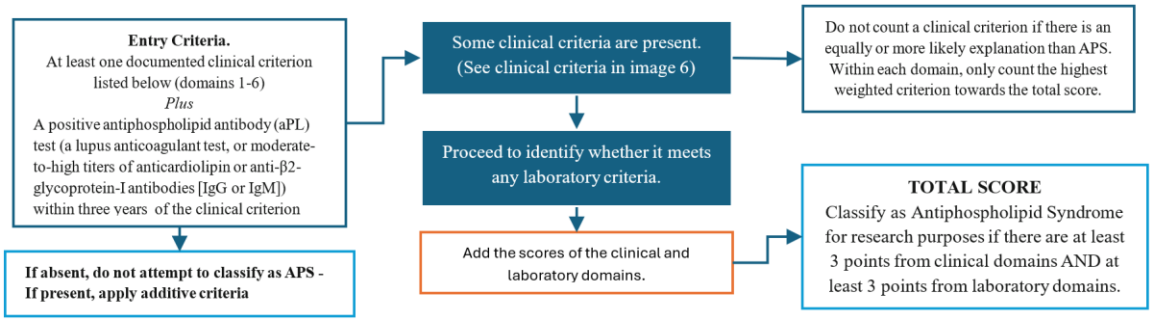

Figure 5: APS classification algorithm.

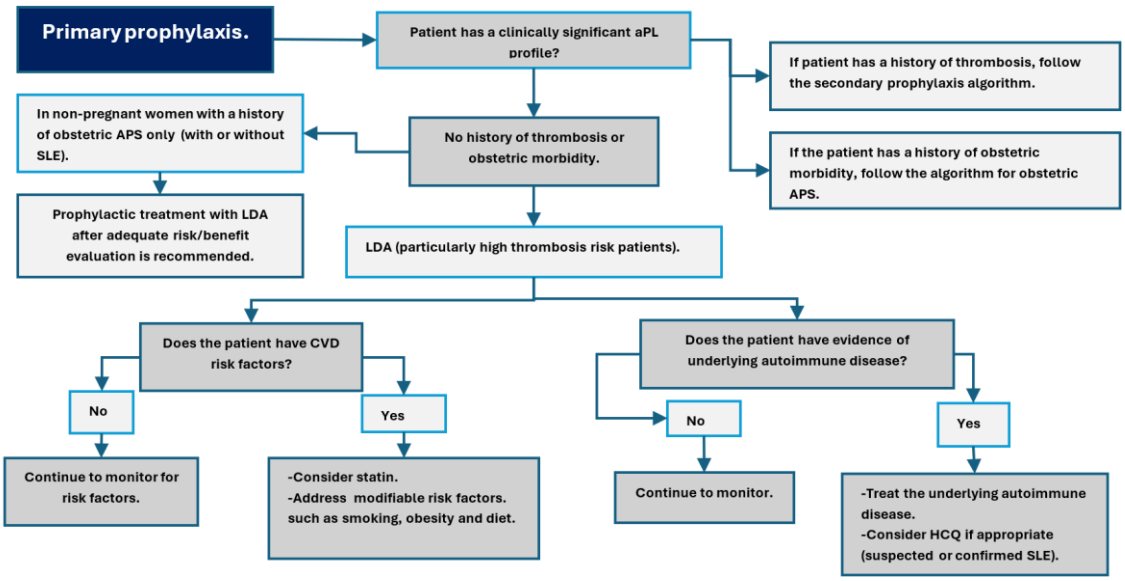

Figure 6: Algorithm for starting primary prophylaxis.

HCQ: hydroxychloroquine, LAD: low doses of acetylsalicylic acid, SLE: systemic lupus erythematosus.

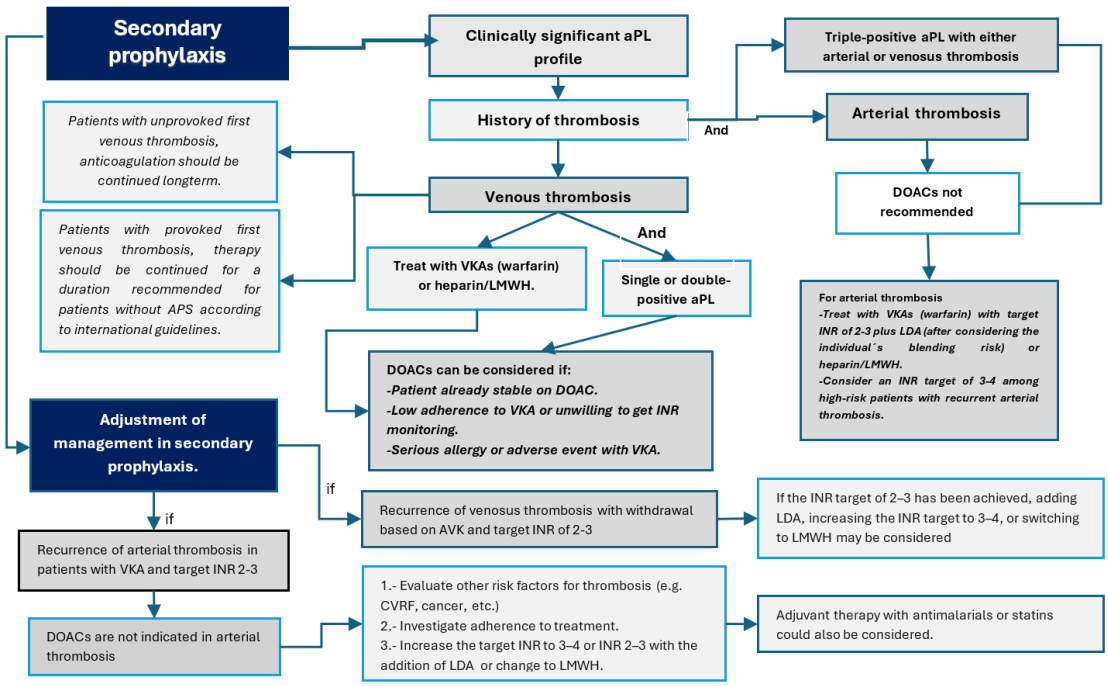

Figure 7: Algorithm for secondary prophylaxis.

We proceed to request the panel of paraclinical tests available in our unit, obtaining the following results: protein C with activity of 28%, protein S with activity 85%, complement C3 1.375 g/l, C4 0.455 g/l, anti-nuclear antibodies<0.50 IU/ml, anti-cardiolipin antibodies IgG<2 IU/ml, anticardiolipin antibodies IgM<2 IU/ml, antithrombin III 30.7 mg/dl, lupus anticoagulant positive, beta 2 glycoprotein-1 IgG 2 U/ml, beta 2 glycoprotein-1 IgM 6.5 2 U/ml (Table 1).

During follow-up, control laboratories were requested again, in which protein C levels were >70% active and lupus anticoagulant persistently positive, therefore the patient was classified as having primary APS since no other pathology was identified and he met the current classification criteria (clinical and radiographic evidence of a thrombotic event in a patient with a low risk profile for venous thromboembolic events, persistently positive lupus anticoagulant, meets 3 points of the clinical domain and 5 points of the laboratory domain).

In this patient, it was decided to start management with rivaroxaban since he had no arterial disease and could not continue with adequate adherence and control of management with vitamin K antagonists or low molecular weight heparin. The patient has not had any new episodes of thrombosis during more than 1 year of follow-up.

For this review, a search was carried out for publications related to the keywords (thrombosis, antiphospholipid antibodies, anticardiolipin antibodies, anti-b2-glycoprotein 1 antibodies, lupus anticoagulant, antiphospholipid syndrome, thrombophilia), using freely accessible search engines. Within the inclusion and exclusion criteria for the selection of information.

Publication date: publications less than 10 years old were selected. Relevance: those publications were selected which provided relevant data for the understanding of the disease or updates on it, the impact factor of the publications and the reliability of the media (magazines, platforms, etc).

Where they were published. Repeated articles. Articles published in unreliable or low-impact media. Articles published more than 10 years ago.

During our search for information, 60 articles were obtained that were related to the key words. Only 17 articles were selected that met all the inclusion criteria, from which the following narrative of the pathophysiology, clinical manifestations, diagnosis and treatment of APS is made.

Table 1: Patient's laboratory results.

Study	Result	Normal value
Anti-nuclear antibodies	<0.5 UI/ml	Negative <1.5
Complement C3	1.375 g/l	0.811-1.570
Complement C4	0.455 g/l	0.129-0.394
Anti-cardiolipin IgG antibodies	<2 U.I/ml	Negative <20
Anti-cardiolipin IgM antibodies	<2 U.I/ml	Negative <13
Antithrombin III	30.7mg/dl	22.5-33.3
Lupus anticoagulant	+	-
Anti-beta 2 glycoproteinin-1 IgG	2 U/ml	Negative <20
Anti-beta 2 glycoproteinin-1 IgM	6.5U/ml	Negative <20
Protein C	Activity 28%	
Protein S	Activity 85%	

Table 2: New classification criteria for APS 2023 EULAR.

Classification criteria for APS 2023 EULAR	
Clinical domains and criteria	**Weight**
D1. Macrovascular (Venous Thromboembolism [VTE])	
VTE with a high-risk VTE profile	1
VTE without a high-risk VTE profile	3
D2. Macrovascular (Arterial Thrombosis [AT])	
AT with a high-risk CVD profile	2
AT without a high-risk CVD profile	4
D3. Microvascular	
Suspected (one or more of the following) -Livedo racemosa (exam) -Livedoid vasculopathy lesions (exam) -Acute/chronic aPL-nephropathy (exam or lab) -Pulmonary hemorrhage (symptoms and imaging)	2

Continued.

Classification criteria for APS 2023 EULAR	
Established (one of more of the following) -Livedoid vasculopathy (pathology -Acute/chronic aPL-nephropathy (pathology -Pulmonary hemorrhage (BAL or pathology -Myocardial disease (imaging or pathology) -Adrenal hemorrhage (imaging or pathology)	5
D4. Obstetric	
>3 Consecutive pre-fetal (<10w) and/or early fetal (10w 0d -15w 6d) deaths	1
-Fetal death (16w 0d – 33w 6d) in the absence of pre-eclampsia (PEC) with severe features or placental insufficiency (PI) with severe features	1
-PEC with severe features (<34w 0d) or PI with severe features (<34w 0d) with/without fetal death	3
-PEC with severe features (<34w 0d) and PI with severe features (<34w 0d) with/without fetal death	4
D5. Cardiac Valve	
Thickening	2
Vegetation	4
D6. Hematology	
Thrombocytopenia (lowest 20-130x109/L)	2
Laboratory (aPL) domains and criteria	
Laboratory (aPL) domains and criteria	**Weight**
D7. aPL test by coagulation-based functional assay (lupus anticoagulant test [LA])	
Positive LA (single – one time)	1
Positive LA (persistent)	5
D8. aPL test by solid phase assay (anti-cardiolipin antibody [aCL] ELISA and/or anti-β2-glycoprotein-I antibody [aβ2GPI] ELISA [persistent])	
Moderate or high positive (IgM) (aCL and/or aβ2GPI)	1
Moderate positive (IgG) (aCL and/or aβ2GPI)	4
High positive (IgG) (aCL or aβ2GPI)	5
High positive (IgG) (aCL and aβ2GPI)	7

Table 3: Summary of risk stratification in patients with APS.

Risk stratification		
Risk profiles for venous thromboembolism	**Risk profiles for cardiovascular disease.**	**Antibody risk profiles.**
Major VTE risk factors (any of the following at the time of the event): -Active malignancy, Hospital admission confined to bed (only bathroom privilege), Major trauma with fractures, spinal cord injury or Surgery. **Minor VTE risk factors (2 or more of the following at the time of the event):** Active systemic autoimmune disease or active inflammatory bowel disease , Acute/active severe infection, Central venous catheter in the same vascular bed, Hormone replacement therapy, Long distance travel (≥8 hours), Obesity, Pregnancy or postpartum, Prolonged immobilization not counted above, Surgery High-risk VTE profile is defined based on 1 or more major OR 2 or more minor VTE risk factors.	**High CVD risk factors (any of the following at the time of the event):** -AHT with systolic blood pressure ≥180/110 mm Hg. Chronic kidney disease FG ≤60 ml/min. DM with organ damage. Hyperlipidemia (severe) with total cholesterol ≥310 mg/dl or LDL >190 mg/dl. **Moderate CVD risk factors (3 or more of the following at the time of the event):** -AHT treatment, or with persistent >140/90. Current tobacco smoking. DM with no organ damage and short disease duration. Hyperlipidemia (moderate) on treatment, or with TC <310 mg/dl, or LDL above normal range and <190 mg/dl, Obesity (BMI ≥30 kg/m²). High-risk CVD profile is defined based on 1 or more high CVD risk factors OR 3 or more moderate CVD risk factors.	**High risk profile:** The presence (on 2 or more occasions at least 12 weeks apart) of positive Lupus Anticoagulant – measured according to the ISHT guidelines. Double positivity of anti-phospholipid (aPL) antibodies, any combination of lupus anticoagulant, anticardiolipin (aCL) antibodies or anti b2 glycoprotein I antibodies (anti-B2GP1), or triple positivity (the three subtypes). -The presence of persistently high aPL titles. High-risk antiphospholipid antibody (aPL) profile is associated with increased risk of thrombotic and obstetric APS. **Low risk profile:** -aCL or anti-b2 glycoprotein I antibodies, as an isolated laboratory finding, at low-medium titles, especially if they are transiently positive. aPL medium-high antibody titles: aCL IgG or IgM in serum or plasma present in titles >40 IgG/IgM phospholipid units or >99th percentile, measured by standardized ELISA. aB2GP1 IgG or IgM in serum or plasma in titles >99th percentile, measured by standardized ELISA.

DISCUSSION

APS is a pathology that is difficult to diagnose due to its diverse and non-specific clinical manifestations, in addition to the low availability of the corresponding laboratory studies (antiphospholipid antibodies) in medical care units.

The identification of patients with the presence of antiphospholipid antibodies and subsequent risk stratification for the development of thrombotic events allows for timely initiation of primary prophylaxis to avoid such events or reduce their severity. On the other hand, documenting the presence of APS as a cause of thrombotic events allows the initiation of secondary prophylaxis measures, thereby reducing patient morbidity.

In this article, after carrying out a systematic search for information, selecting the most relevant information on the topic, a summary is made exposing the most relevant points about APS, emphasizing the pathophysiology, clinical manifestations, classification criteria and treatment.

We can summarize the pathophysiology in 3 events that occur simultaneously: the formation of antibodies, a triggering event/injury (double hit) and the activation of the coagulation cascade, activation of the complement system and activation of the immune system (Figure 2).[3,6]

Antibody formation

The first postulated step is the formation of antibodies, which is the result of the interaction of some pathogen with the immune system, the three main antibodies documented in APS are.

Lupus anticoagulant

They are IgG/IgM immunoglobulins that inhibit phospholipid-dependent (PL) coagulation reactions in vitro. They affect the activation of prothrombin by the prothrombinase enzyme complex.[7,8]

Anti-cardiolipin antibodies

Cardiolipin is a phospholipid found in the cell membrane, which in addition to having a structural role has other functions in the cell. On its own it is not immunogenic, however, when it binds to beta 2-glycoprotein 1 it can generate an immune response, that is, it could be considered a hapten.[8,9]

Anti B2 glycoprotein 1 antibodies

B2-glycoprotein 1 (b2GP1) is a protein found in plasma in free form or bound to lipoproteins and can bind to phospholipids in cell membranes. Anti-b2GP1 antibodies are the most studied in APS, as they play different roles in the pathogenesis of the disease. The formation of the anti-b2GP1 complex with one of its receptors triggers a different cellular response depending on the receptor and cell to which it binds. In the case of APS, most cellular responses to this antibody are associated with thrombosis.[8,9] It is important to remember that the presence of these antibodies alone is not considered pathological, they are present in up to 5% of the population.

Double hit, the double hit theory postulates that despite the presence of antibodies, a triggering event is necessary to initiate an inflammatory response, the most frequent situations that can initiate such a response are infections, trauma or pregnancy. Activation of the coagulation cascade, complement and immune system.

As previously mentioned, these are events that occur at the same time. When antibodies bind to their target receptor, they produce cellular activation. The main target cells are endothelial cells, platelets, neutrophils and monocytes, which when activated release proinflammatory and procoagulant cytokines. Likewise, antiphospholipid antibodies can directly activate the complement or through products released by activated immune cells and can directly activate or inactivate coagulation factors.[10]

Pathophysiology of obstetric APS

The mechanisms by which antiphospholipid antibodies can cause adverse obstetric events, are not fully understood, although the thrombotic phenomena related to these may justify them. Other alterations induced by aPL have been demonstrated that are closely related to the elevation of sVEGFR1 levels (anti-angiogenic molecule related to hypertensive disorders of pregnancy). The most relevant findings of the obstetric condition in APS are summarized below (Figure 3).[3,11]

At the trophoblast level, antiphospholipid antibodies, specifically the anti b2-glycoprotein1/ b2-glycoprotein 1 antibody complex, can perform the following actions. Produces the activation of the low-density lipoprotein receptor-related protein 8; (LRP8) found in the membrane of the trophoblast cells, decreasing the proliferation and invasion of the trophoblast and increasing the levels of endoglin, resulting in inadequate implantation. Produces activation of the inflammasome system in trophoblast cells by binding to toll-like receptor 4 (TLR 4), increasing the release of interleukin 1b and interleukin 8, causing local inflammation. Produces activation of the complement system by binding to the c5 fraction of complement and transforming it into the c5a fraction, which binds to its receptor on the cell membrane of neutrophils and stimulates the release of sVEGFR1 by these cells.

As a result, alterations occur at the placental level that produce fetal losses. Figure 4 summarizes the alterations at the trophoblast level due to the action of antiphospholipid antibodies.

The clinical manifestations are diverse and may even go unnoticed. The main clinical manifestations are related to thrombosis, which can affect venous or arterial territories. Following in frequency are obstetric events and the less frequent but more fatal presentation is catastrophic antiphospholipid syndrome (CAPS) (Figure 3). Summarizes the alterations at the trophoblast level due to the action of antiphospholipid antibodies (Figure 4).

Thrombosis is the most common cause of the signs and symptoms that patients present. Single or multiple thrombi may appear in veins, arteries and/or in the microvasculature. The most common venous system condition is deep vein thrombosis in the lower limbs (39% of cases). Other affected sites are pulmonary, renal, retinal, hepatic veins and portal veins. Arterial disease is less common, but more serious, the most common condition is stroke (20% of cases). Other arterial manifestations include coronary, renal and adrenal artery thrombosis, etc.[1-3,12]

Obstetric manifestations, the most frequent alterations in pregnant patients with APS are preeclampsia, eclampsia and placental abruption, which can cause pregnancy loss. The main and most serious complication is recurrent spontaneous abortion, usually 10 weeks before gestation. It is difficult to differentiate between hypertensive complications of pregnancy per se and obstetric manifestations of APS. Both share a common point regarding pathophysiology: Abnormal elevation of sFLT-1 (sVEGFR).[11]

Catastrophic antiphospholipid syndrome, it is the most severe manifestation of APS, occurring in less than 1% of cases. It is defined as the simultaneous involvement of 3 or more organs in patients in whom the presence of aPL is demonstrated.[13] The precipitating factor can be identified in approximately half of the cases, with the most frequent causes being Infection (49%), surgery or trauma (17%), malignancy (16%), withdrawal of anticoagulation or subtherapeutic anticoagulation (8%), pregnancy complications or initiation of oral contraception (8%), medications (5%) and flare of an underlying autoimmune disease (e.g., SLE) (3%).[13,14]

The main manifestations of catastrophic APS are renal failure (75%), respiratory involvement (60%, acute respiratory distress syndrome is a common initial presentation of (CAPS), neurological involvement (50%), cardiac involvement (45%) and skin manifestations (~40%).[14,15]

Classification criteria, the new classification criteria score the findings in the clinical and laboratory domains to classify patients with APS. To make this classification, they require at least 3 points in the clinical domains and 3 points in the laboratory domains (it is worth remembering again that like other diseases such as SLE, there are no diagnostic criteria but rather classification), this new classification increases the sensitivity and specificity compared to the previous Sydney classification, with specificity of 99% vs 86% and a sensitivity of 84% vs. 99%, respectively.[5] The algorithm and classification criteria are summarized in figure 5 and table 2.

Classification criteria for catastrophic APS, catastrophic APS is the least frequent but potentially fatal presentation of APS defined as the involvement of more than 3 organs simultaneously. To support the diagnosis, we have the 2003 criteria. A definitive diagnosis of CAPS is made when the patient presents the following criteria.

Clinical manifestation of damage to 3 or more organs and systems. Simultaneous and rapid development of clinical manifestations (less than 1 week). Occlusion of small-caliber vessels on histological examination. Laboratory confirmation of the presence of aPL (according to the Sydney recommendations). A diagnosis of probable CAPS is made if: criteria 2, 3, and 4 are present, but only two organs or systems are involved in the pathological process, instead of 3 and more. Criteria 1, 2, and 3 are observed, but there is no laboratory confirmation of the presence of aPL; Criteria 1, 2, and 4 are present. Criteria 1, 3, and 4 are present, but clinical manifestations develop within 1 month, despite anticoagulant therapy. The treatment of APS is complex and is based on the following principles.

Perform individual risk stratification of the patient with antiphospholipid antibodies+, which includes staging according to the cardiovascular risk profile and risk profile determined by the type of antibody found.[1,5]

Perform adequate control of cardiovascular risk factors and risk factors for modifiable thrombotic events.

Offer advice and educate the patient on adherence to treatment, INR control in patients treated with vitamin K antagonists (VKA). Table 3 summarizes the risks due to antibodies, cardiovascular risk factors, and risk factors for thrombotic events. After determining the risks based on the cardiovascular profile and antibody profile, appropriate alternatives for pharmacological management can be established. Pharmacological management is practically based on the use of anticoagulant and antiplatelet agents. This management is divided into two approaches, primary prophylaxis and secondary prophylaxis. In some situations, adjustments must be made to the therapeutic plan, or it must be individualized, for example, in pregnancy. The treatment strategies based on the patient's clinical situation are summarized below.[1,2,12]

Primary prophylaxis It is the management offered to patients who have not suffered thrombotic or obstetric events but have documented the presence of antibodies and who belong to a high-risk profile. Management consists of the use of low doses of acetylsalicylic acid and statins (if present a high cardiovascular risk).[1,17] The therapeutic algorithm for primary prophylaxis is summarized in Figure 6.

Secondary prophylaxis is the management given to patients who present thrombotic or obstetric events. In this case, the management is based on the use of anticoagulants, which may be vitamin K antagonists or direct anticoagulants (such as rivaroxaban). The type of anticoagulant is defined according to the characteristics of the patient. Image 10 presents a practical algorithm for starting secondary prophylaxis (Figure 7). [1,2,17]

Adjustment in the management of secondary prophylaxis, in those patients who have already been started on anticoagulant therapy and have presented new thrombotic events despite reaching or not the INR goal, it is necessary to make pertinent adjustments (Figure 7).[1,2,17]

Treatment of catastrophic APS, the primary goal of therapy should be to suppress thromboinflammation by reducing excessive complement activation, cytokine levels, NET formation, and excessive thrombin generation. Due to the low incidence (less than 1% of APS patient cases), no standard therapy has been established for the management of patients who develop this complication.[14,15] Management of patients with CAPS can be resumed as follows.

Initial stabilization of the patient, ABCDE, organic support therapy and assessment by intensive care service. Use of immunomodulatory therapy, being able to use the drugs and therapies available in the unit, the following therapeutic measures are recommended. Anticoagulation with heparin, it is the most important treatment, initially infusion of unfractionated heparin is recommended, but LMWH is useful, later changing to VKA. LMWH at a dose of 1 mg/kg (in the case of enoxaparin every twelve hours).

Steroid, it is because CAPS is an inflammatory state or is associated with the presence of an underlying rheumatological disease, not useful as monotherapy, for example doses of 250-750 mg/day of metheil prednisole for 3 days. Plasma exchange, may be preferred in patients with microangiopathic haemolytic anaemia or renal dysfunction, this approach reduced mortality by 37% and increased the recovery rate by 78%.

Immunoglobulin, preferred in autoimmune thrombocytopenia, given evidence of benefit in this condition. Dose 0.4 g/kg/day for five days or 1 g/kg/day for two days. Other drugs may use for refractory disease such as rituximab, eculizumab, or cyclophosphamide.

CONCLUSION

Antiphospholipid syndrome is a rare disease and probably underdiagnosed in primary and secondary care centres due to lack of clinical suspicion or resources (paraclinical studies).

In our environment, there is a lack of reliable epidemiological data to estimate the incidence and prevalence of this disease. APS or some other thrombophilia should be suspected in patients with recurrent thromboembolic events, especially in young patients, with thrombosis in unusual sites, with a family history of thrombosis, spontaneous thrombosis during pregnancy and women who, in the absence of evidence of a thrombotic event, have had recurrent early and late abortions.

Current recommendations for the diagnostic and therapeutic approach focus on the timely identification of those patients with the presence of antiphospholipid antibodies and stratify them according to their risk factors to assess and initiate or modify pharmacological therapy. To date, the most used drugs for management continue to be low molecular weight heparins, vitamin K antagonists, and direct inhibitors of coagulation factors, for example rivaroxaban or apixaban, are increasingly being used in selected groups. of patients, hoping that in the future there will be more convenient and safe drugs to use.

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