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Case Report

Patient With Stroke Secondary to Takayasu Arteritis Case Report☆

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ABSTRACT

Takayasu arteritis is a chronic inflammatory and idiopathic vasculopathy of the large arteries, whose consequences are occlusive, stenotic or aneurysmal disease of aorta and its immediate branches.

We describe a case of a 37 year old woman with focal neurological deficit, with magnetic resonance angiography demonstrating decreased flow of the left internal carotid and thinning of left cerebral artery. Thoracic aortogram and neck vessels confirms the findings and also demonstrates the left subclavian artery stenosis, meeting the classification criteria of Takayasu arteritis.

A review of Takayasu arteritis is performed with emphasis on the pathophysiology, clinical manifestations, diagnosis, treatment and prognosis.

Paciente con ataque cerebrovascular isquémico secundario a arteritis de Takayasu: reporte de caso

Palabras clave:
Arteritis de Takayasu
Enfermedad vascular
no aterosclerótica
Ataque cerebrovascular

RESUMEN

La arteritis de Takayasu es una vasculopatía crónica inflamatoria e idiopática de las grandes arterias, cuyas consecuencias son cambios oclusivos, estenóticos o aneurismáticos de la aorta y de sus ramas inmediatas.

Se presenta el caso de una mujer de 37 años con déficit neurológico focal y angiorresonancia, que demuestra disminución del flujo de la arteria carótida interna izquierda y adelgazamiento de la arteria cerebral izquierda con aortograma torácico y de vasos de cuello que confirma los hallazgos y, además, demuestra estenosis de la arteria subclavia izquierda, cumpliendo los criterios clasificatorios de una arteritis de Takayasu.

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Se realiza una revisión de la arteritis de Takayasu, haciendo énfasis en la fisiopatología, manifestaciones clínicas, diagnóstico, tratamiento y pronóstico.

Introduction

Takayasu arteritis (TA) is a type of large-vessel vasculitis that affects especially the aorta, resulting in ectatic occlusive changes, dilatation or aneurysm formation, mainly in the aorta and its immediate branches, as well as in the pulmonary artery.[1]

In the second decade of the 20th century, the ophthalmologist, Dr. Takayasu documented alterations of the flow of the retinal central vessels in the ocular fundus of a young woman.[2] Subsequently, the absence of pulses in the extremities, associated with the ocular changes described by Takayasu, configured the disease that bears his name.[3]

In 1952 Dr. Shimizu and Dr. Sano presented a detailed description of a brachiocephalic arteritis and called the condition as the pulseless disease, name that became popular and today is considered synonymous along with the term aortic arch syndrome.[4] In the next decades, Oriental researchers collected information from autopsies and arteriographies which demonstrated that the disease also involves the rest of the thoracic and abdominal aorta, as well as its branches.[5]

TA is considered a rare disease that mostly affects young women (82.9-97%) in the third and fourth decades of life,[6-9] mainly Asian, with a highest prevalence in Japan with 40 cases per million,[7] while in the Western world there is a similar incidence among these countries with a range between 0.4 and 2.6 cases per million.[8,10-13] In Latin America the largest series of patients was described by Soto et al., with 110 Mexican patients in which the disease has a behavior and prognosis similar to those observed in Oriental patients.[8] It is noteworthy that in the local context Cañas et al., described similar results in 35 Colombian patients.[14]

It is very important to establish a timely diagnosis of TA, since the inflammation and the progressive arterial remodeling increase the chances of developing stenosis, occlusion, dilatation or aneurysm formation.[15] Unfortunately, the delay in diagnosis since the onset of the first symptoms until diagnosis is most usual in our environment, taking from months to years.[16]

Usually, the initial clinical presentation is with constitutional symptoms, but occasionally it can start with ischemia of a particular organ, as for example, a cerebrovascular event, which is not infrequent, but few cases have been described at the time of the diagnosis.[17] This is a presentation of a case of a patient in the fourth decade of life which begins with an ischemic cerebrovascular attack associated with occlusion of the left carotid artery, confirmed by arteriography.

Case Presentation

Patient with a clinical picture of headache and paresthesia of the right hand of 2 years of evolution, for which 10 days prior to the admission through the emergency unit, was being treated with carbamazepine 200 mg/day, prescribed by a general practitioner with a diagnostic impression of carpal tunnel syndrome. When she consults for the first time to the emergency service she presented 2 days of unsteady gait, a subjective sensation of dizziness and drowsiness, without affecting the thought contents. At this time it is thought that the referred symptoms are attributable to the side effects of carbamazepine and she is discharged. She is readmitted 24 hours after, having, in addition to the described symptoms, left brachial hemiparesis and motor aphasia. In the review of systems she denied headache, blurred vision, hallucinations or sensory alterations. She also denied red eyes, hair loss, dysphagia, oral ulcerations, skin rash, arthralgias, Raynaud's phenomenon, cough, dyspnea, abdominal pain, edemas and foamy urine.

Personal History

- Polycystic ovary syndrome.
- Obesity.
- Seizures. Two years ago she had two convulsive episodes and was treated with valproic acid which was discontinued a few months later.

Her vital signs were stable with a blood pressure of 120/85mmHg in the left arm and 130/90 mmHg in the right arm, with no murmurs in subclavian arteries. On the physical examination was evidenced a diminution of pulses in the lower limbs and the left upper extremity; and in the neurological evaluation the muscle strength was 3/5 in left upper limb and 5/5 in the rest of the extremities; bradylalic with coherent speech, with no alterations in cranial nerves, meningeal signs, dysmetria, adiadochokinesia, or gait alterations. NIHSS scale score 3. A tomography of the skull and an echocardiogram were performed, which reported no alteration, as well as the studies of autoimmunity. The exams with simple and contrasted resonance of the encephalon showed a hypointense image on T1, hyperintense on T2, with restricted diffusion in the left frontal region associated with reduced flow in the left internal carotid artery and thinning of the left middle cerebral artery.

It was decided to perform an aortography with arteriography of the vessels of the neck and the brain, which reported an occlusion of the left common carotid artery, from its origin. It was also observed the left subclavian artery with progressive diminution of its lumen, the left vertebral artery with significant dilatation, from which spinal branches are originated, generating important collateral circulation that reconstituted the flow of the left internal and external carotid arteries, as can be seen in Figure 1. Also, it was noted an amputation stump inferior to the left carotid bifurcation.

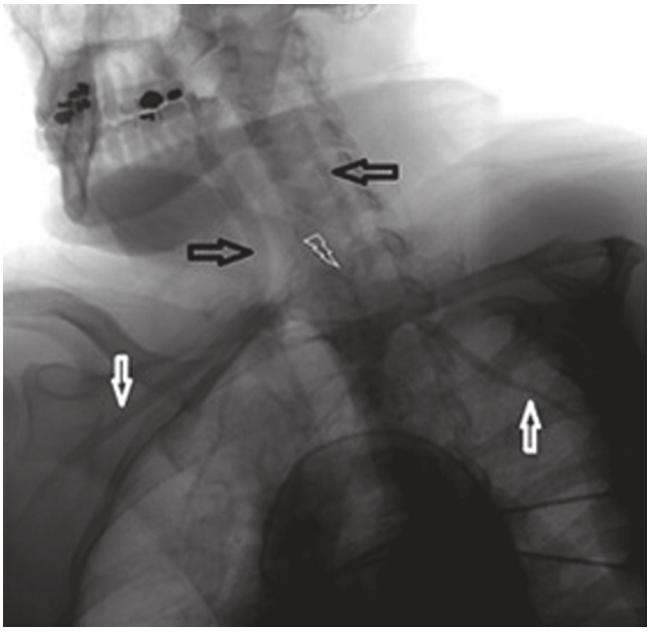

Figure 1 – Arteriography of the patient of the case. The white arrows show the asymmetry between the subclavian arteries. The arrowhead points to the amputation of the left common carotid artery. The arrows to the right and to the left mark the right internal carotid and left vertebral arteries, respectively, note the great compensatory dilatation of the latter vessel.

Already with the diagnosis of TA, it was started the management with high-dose steroids and methotrexate with adequate tolerance to therapy. The patient had a favorable clinical evolution, with no speech or cognitive alterations, walking without gait alterations, controlling sphincters, only with left hemiparesis (4/5), with a modified Rankin scale score of 2 (mild disability). She was discharged with the following treatment: prednisolone 70 mg/day and methotrexate 15 mg/weekly. The follow-up was carried out by Rheumatology and Neurology, with full recovery of the right hemiparesis after 2 months.

Discussion

Approximately 10-20% of patients with TA have been reported to have neurological manifestations. These include dizziness, headache and cerebrovascular events that are rarely described as the first manifestation of the disease.[17] There have also been described cases that begin with seizures and we do not rule out that the history of convulsions of the patient 2 years ago could be explained by the TA.[18,19]

In the approach of a young patient with an ischemic cerebrovascular attack, as that which occurred in our case, the involvement of major extracranial arterial vessels is the cardinal manifestation for considering the TA. However, a variety of systemic inflammatory disorders may be associated with large-vessel vasculitis. These include giant cell arteritis, Behçet's disease, Cogan's syndrome, relapsing polychondritis and sarcoidosis. Aortitis can also be associated with systemic autoimmune diseases such as systemic lupus erythematosus, rheumatoid arthritis and spondyloarthropathies. It should also be borne in mind the infectious etiology such as syphilis, tuberculosis and mycotic aneurysms caused by *Staphylococcus* spp. and *Salmonella* spp., which are the most common germs. Other non-inflammatory conditions such as Marfan syndrome, Ehlers Danlos syndrome, neurofibromatosis, and fibromuscular dysplasia of the media may exhibit lesions similar to those of our patient.[20-22]

In addition to the stenosis of extracranial vessels as an explanation of the cerebrovascular events, there also may occur emboli originated from inflamed areas in the arteries either with or without aneurysmal dilatation.[23] The combination of both phenomena can explain the cerebrovascular attack of the patient. Other postulated mechanisms are intracranial stenosis of arterial vessels, high blood pressure and premature atherosclerosis.[17]

Regarding the pathogenesis, the studies have shown CD4 and CD8 cell infiltration in 15%.[24] These cells play an important role in the pathogenesis of the large-vessel vasculitis.[25] Gamma interferon is responsible for the formation of giant cells and the activation of macrophages that release vascular endothelial growth factor (VEGF) and platelet-derived growth factor, resulting in an increase of neovascularization and intimal proliferation, respectively.[24]

TA can have a monophasic clinical course in 20% of the cases. The vast majority (80%) has a chronical course with relapses over time.[26,27]. At the onset of the disease predominate vague constitutional symptoms such as myalgias and fever that are accompanied subsequently by symptoms specific to the involved vessels, such as asymmetrical pulses in 57%, or discrepancy of the blood pressure of the extremities in 53%. Additionally, aneurysmal degeneration or dissection of the aorta may occur. It can also manifest itself as stroke, myocardial infarction and claudication of upper limbs.[28,29] Other manifestations include arthralgias, skin lesions simulating erythema nodosum and pyoderma gangrenosum.[27,30]

There are classificatory criteria of the American College of Rheumatology for TA (Table 1), and 3 of the 6 criteria must be met to be classified with 90.5% sensitivity and 97.8% specificity.[31] Our patient met 4 of them: age under 40 years, asymmetry of the pulse and the brachial blood pressure, and angiographic abnormalities. Let's remember that these classificatory criteria were developed to identify homogeneous groups that can be distinguished from other forms of similar diseases, in order to carry out epidemiological an treatment studies, rather than to be used in clinical practice as a diagnostic tool, thereby excluding a considerable number of patients with the disease in early stages or with atypical manifestations. On the other hand, the diagnostic criteria help to distinguish the disease from normal controls of the general population and other conditions that may simulate the disease.[31-33]

In 1988, Ishikawa proposed his diagnostic criteria (Table 2) based on the age of onset and on clinical, laboratory and angiographic parameters, grouped into major and minor criteria, with a sensitivity of 84% and specificity higher than

Table 1 – Classificatory Criteria of the American College of Rheumatology for Takayasu Arteritis.

Age of onset	Signs or symptoms of Takayasu arteritis before 40 years old
Claudication	Upper of lower limbs fatigue with exercise
Diminished brachial pulses	Diminished unilateral or bilateral brachial pulses
Asymmetry of brachial arterial blood pressure	Difference between brachial systolic blood pressure >10 mmHg
Murmur	Audible over the aorta or the subclavian artery
Angiographic abnormalities	Occlusion or stenosis of the aorta, or of its branches or of the large caliber arteries in upper extremities, either localized or segmental. Not explained by atherosclerosis, fibromuscular dysplasia or other causes

Source: Arend et al.[31]

Table 2 – Ishikawa's Diagnostic Criteria for Takayasu Arteritis Modified by Sharma.

Three major criteria	
Lesion of the middle portion of the left subclavian artery	The most severe stenosis or occlusion present in the middle portion from the point 1 cm proximal of the vertebral artery or 3 cm distal of the orifice determined by angiography
Lesion of the middle portion of the right subclavian artery	The most severe stenosis or occlusion present in the middle portion from the right vertebral artery orifice to the point 3 cm distal of the orifice determined by angiography
Characteristic signs and symptoms of at least one month of duration	Includes claudication in the limbs, abolition of pulses of difference between pulses in limbs, a significant difference in blood pressure (difference between the systolic arterial blood pressure in limbs >10 mmHg), fever, neck pain, transient amaurosis, blurred vision, syncope, dyspnea or palpitations
Ten major criteria	
Elevated sedimentation rate	Unexplained persistent elevation >20 mm/h at the diagnosis or presence of evidence in the history of the patient
Weak carotid pulse	Unilateral or bilateral weak pulse upon palpation
Hypertension	Persistence of a brachial arterial blood pressure >140/90 mmHg or popliteal >160/90 mmHg
Aortic regurgitation or annuloaortic ectasia	Aortic regurgitation by auscultation or Doppler echocardiography or angiography; or annuloaortic ectasia by angiography or two-dimensional echocardiography
Lesion in the pulmonary artery	Lobar, segmental arterial occlusion or equivalent, determined by angiography, perfusion gammagraphy, or presence of stenosis, aneurysm, luminal irregularities or any combination determined by angiography in the trunk of the pulmonary artery or in the pulmonary arteries, unilateral or bilateral
Lesion in the middle portion of the left common carotid artery	Presence of severe stenosis or occlusion in the middle portion, 5 cm length and 2 cm distal to its orifice determined by angiography
Distal lesion of the brachiocephalic trunk	Presence of severe stenosis or occlusion of the distal third determined by angiography
Lesion of the descending thoracic aorta	Narrowing, dilatation or aneurysm, or irregularity of the lumen or any combination of aneurysms, determined by angiography; isolated tortuosity is not accepted
Lesion of the abdominal aorta	Narrowing, dilatation or aneurysm, or irregularity of the lumen or combination of aneurysms
Lesion of the coronary arteries	Documented by angiography before 30 years of age in the absence of risk factors such as hyperlipidemia or diabetes mellitus

Source: Sharma et al.[35]

95%.[33] Subsequently, these criteria were applied to patients from India with TA proven by angiography, the sensitivity decreased to 60.4%.[34] In 1995, Sharma modified Ishikawa's criteria with the removal of the age < 40 years, the exclusion of aortoiliac lesions and the incorporation of lesions of coronary arteries in young patients.

A high probability of TA is considered when there are 2 major criteria or one major criterion and 2 minor criteria or 4 minor criteria, with a sensitivity of 92.5% and the same specificity.[34,35] In any case, the scientific community considers that there is a need to create diagnostic criteria validated in a greater and diverse population, for the majority of the vasculitis and, in this sense, there is currently an international effort: the Diagnostic and Classification Criteria for Vasculitis Study, in order to include 2000 patients with systemic vasculitis and 1500 with conditions that resemble a vasculitis, which is in the phase of recollection of patients.[36]

As for the laboratory studies, there are no characteristic findings, it is usual to find negative autoimmunity studies, anemia of chronic disease, elevation of the C-reactive protein and the erythrocyte sedimentation rate. However, up to 11% of patients have normal acute-phase reactants at the time of

the diagnosis,[26,29] as in the case of our patient. This opens the possibility of non-inflammatory pathologies, described previously, specifically fibromuscular dysplasia of the media, since the phenotype of the patient ruled out congenital conditions. Nevertheless, the angiographic pattern of commitment makes it unlikely, as will be explained later. It is noteworthy that acute-phase reactants do not serve either for the follow-up of the treatment since up to 25% of patients would be normal despite having the active disease.[26,27]

Having in mind that the biopsy of large arteries cannot be performed easily, images are essential for the diagnosis. Catheter-directed angiography has traditionally been the gold standard, however, it only detects lumen abnormalities, dilatation and stenosis, which are absent in the early stages of the disease, and therefore other techniques such as angiotomography and angioresonance are emerging as useful techniques that are non-invasive and capable of detecting luminal and mural alterations.[37] Resonance has the problem of poor standardization and ultrasonography is operator-dependent.[38]

The angiographic classification of the TA Consensus of 1994 (Fig. 2) defines the following types of lesions: Type I, involves the vessels that arise from the aortic arch, Type IIa involves the ascending aorta, the aortic arch and their branches. In the Type IIb, besides the foregoing, the descending thoracic aorta is added; Type III includes the abdominal, the descending thoracic aorta, or the renal arteries: Type IV affects the abdominal aorta or the renal arteries; and finally the Type V combines the Types IIb and IV. The letters C or P are added depending on whether there is compromise of the coronary or pulmonary arteries, respectively.[39] The affection of the middle cerebral and left subclavian arteries, without involvement of the thoracic aorta, of our patient, probably classifies as Type I (the abdominal aorta and the renal arteries were not evaluated), which is more frequent in Japanese patients, along with Type II; while in other Asian and Latin American patients its frequency decreases. These variations suggest the role of ethnicity in the pathophysiology of TA.[26,39] In the series of Cañas et al., the most frequent was type I with 34%, followed by type V and type IV with 28.8 and 20%, respectively.[16]

Previous works suggest that mural thickening is the most important finding in early stages.[37] Calcifications in the thickened wall is another important sign, they are usually transmural and have been observed in 27% of patients.[38] In the exploration by angiotomography before contrast, the mural thickening is of high attenuation with respect to the lumen, while in the images after the contrast, the double ring enhancement becomes apparent, being more evident in the venous phase. The most common is to find a poor enhancement within the ring and increased on the periphery. It has been proposed that the internal part represents the intimal edema, while the external ring indicates inflammation of the media and the adventitia.[38,40] Some studies show that this double ring pattern is useful for assessing the efficacy of the treatment.[40] The positron emission tomography with 18-fluorodioxyglucose is a promising exam since by evaluating the metabolic activity of the vascular wall it highlights the inflamed areas, and as the inflammation precedes the morphological changes it can facilitate an earlier diagnosis, with a sensitivity of 92% and specificity of 100% in the diagnosis.[41,42]

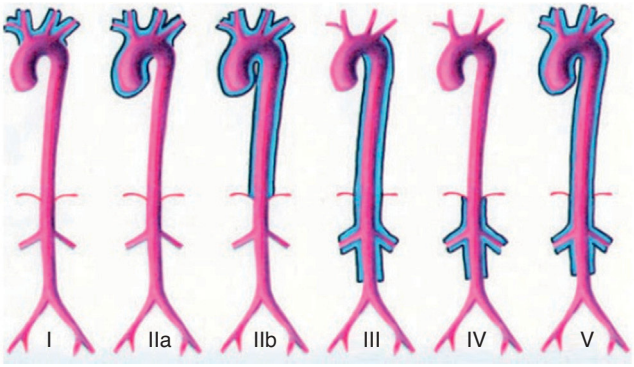

Figure 2 – Angiographic classification of the Takayasu Arteritis Consensus of 1994. Taken from Nastri et al.[50]

Likewise, markers such as ICAM-1, VCAM-1 and IL-1 have been proposed to determine the activity of the disease, however, a consensus has not been reached in this regard.[43] Recently, the measurement of pentraxin 3 has emerged as a more specific marker of vascular inflammation; according to a study published in Annals of Internal Medicine, measurements of pentraxin 3 higher than 1 ng/ml are more accurate to indicate disease activity compared with the measurements of C-reactive protein and erythrocyte sedimentation rate.[44]

With regard to the treatment, high doses of steroids are the first-line therapy, however, the majority of patients relapse when these drugs are discontinued.[45] According to some studies, persistent remissions only can be achieved in less than 30% of patients after reducing the dose of prednisolone to 10 mg or less. Combined therapy with immunosuppressive agents, such as azathioprine, methotrexate, tacrolimus and cyclosporine is used in 46- 84% of patients, in whom the discontinuation of steroids is difficult. The use of infliximab is recommended even in patients who do not respond to a second-line therapy.[46]

Therapeutic alternatives such as the anti-TNFα are useful to reduce the duration of the disease, as evidenced by the series of cases reported by Hoffman,[47] conducted in 15 patients with active disease or relapse despite the treatment with steroids and second-line agents. The average duration of the disease, prior to the use of the anti-TNF, was 6 years; after receiving infliximab, 93% showed sustained remission during an average of 1 to 3.3 years.

When a patient has uncontrolled symptoms, surgical treatment is an option that can prevent further deterioration. However, the surgical indications for TA are controversial, because they depend of the number of vessels involved, the extent of the occlusion, the presence of collateral circulation and the clinical manifestations; it is important to bear in mind that in the phase of acute inflammation, the surgery implies a high risk due to the great potential of graft rejection and complications.[48]

The prognosis of these patients is usually variable, and the presence of aortic insufficiency, hypertension and heart failure has been proposed as a determining factor.[46] The mortality data are limited, with mortality rates of 3-15% reported in different series with different managements and of diverse

causes such as heart failure, kidney failure, stroke, myocardial infarction and aneurysm rupture.[49] In Eastern countries the prognosis has improved in the last decade, thanks to early diagnosis favored by the implementation of non-invasive methods of aid and the advent of new therapeutic alternatives.[15]

Conclusion

The TA is still a rare pathology in our environment; it can be clinically manifested in an aggressive form such a cerebrovascular attack, as it happened to the patient in this case. Even though a variety of diagnostic aids are currently available and there is an urgent need of therapeutic options, the high suspicion of the clinician and, therefore, a timely diagnosis is the main determining factor in the prognosis of the patients.

Ethical Disclosures

Protection of people and animals. The authors declare that no experiments were performed on human beings or animals for this research.

Data confidentiality. The authors state that patient data do not appear in this article.

Right to privacy and informed consent. The authors state that patient data do not appear in this article.

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Conflict of Interest

The authors declare that they have no conflict of interest.

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