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CASE REPORT: ACUTE PERICARDITIS DUE TO MYCOPLASMA PNEUMONIAE IN A SCHOOL AGE PATIENT

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ABSTRACT Approach Strategies for pericarditis management in pediatric patients are based on information collected from adults. In pediatric population mainly exists case reports, where the etiology of the disease is reached. Mycoplasma pneumoniae is a microorganism mainly characterized by infection respiratory tract, but cardio-tropic characteristics have been described in the last decade , by leading colonization and myopericardial infection, with pericardial effusion or acute pericarditis. Even so, the incidence of Mycoplasma pneumoniae associated pericarditis worldwide is low. Based on what is described, our case intends to expand the understanding of this disease.

KEYWORDS : Pediatric Pericarditis, Mycoplasma pneumoniae, Echocardiographic, NSAID, colchicine .

INTRODUCTION

Acute pericarditis (AP) is an inflammatory disorder affecting the layers of the pericardium, which responds to multiple causes , so the etiological approach is complex and the idiophatic disease remains the most common (1). AP is often accompanied by myocarditis, because usual causative agents such as cardiotropic viruses (coxackie, enterovirus, adenovirus), affect both cardiac layers (2). Mycoplasma pneumoniae is a bacterium of the Mycoplasmastacaceae family of the Millicutes class that mainly affects the respiratory system(3). This microorganism can cause extrapulmonary complications, due to direct tissues invasion and/or autoimmune response(3-5). Within Mycoplasma pneumoniae infection, the incidence of cardiac involvement varies from 1% to 8% and is more common in adults than in children(5).

The tests that have shown greater diagnosis accuracy is the polymerase chain reaction (PCR) in combination with serology by ELISA technique, being the most valuable tools in the detection of M. pneumoniae (4-5).

Chest pain is the pivotal symptom that generally initiates the suspicion of AP, although to establish the diagnosis other causes of chest pain must be ruled out. The approach must be complemented with studies that shows the inflammatory involvement of the myocardium and pericardium, such as the electrocardiogram (EKG). or preferably echocardiography (ECC), a non-invasive tool that can establish the diagnosis in 90%, ruling out other cardiac lesions and also helps in the monitoring and surveillance of probable complications such as cardiac tamponade(6). Chest X rays , and biomarkers such as C -reactive protein (CRP) and troponins are useful in the diagnosis too (6).

Nonsteroidal anti inflammatory drugs (NSAIDs) and colchicine constitute the mainstay of treatment (7-9).

Case Presentation

The case of a 9 year old male, with no significant history is described . He went to the hospital for 1 month evolution chest pain, oppressive, progressive in intensity, without predominance of day variation, partially attenuated with acetaminophen. Four weeks after the onset of the symptoms, dyspnea with great exertion and fever of 38°C occurred, which did not respond to the use of antipyretics, for which he was hospitalized. During the initial questioning, he highlighted the history of uncomplicated pharyngitis, 2 months prior to the onset of the condition.

Upon admission to the hospital, he presented the following vital signs: HR 146 bpm RR 22rpm BP 90 / 60mmHg SatO2 98% Temperature 38.5°C. On physical examination: without relevant findings on the neurological examination. pale skin and mucous membranes, good state of hydration, hyperemic pharynx, not tonsuils exudate ,and neither retropharyngeal discharge. Patent external auditory canals, pearly, intact tympanic membranes. Neck with palpable nodes measuring 0.5 cm, softs, mobiles, not painful on palpation. Normal chest amplexion and amplexation movements, vesicular murmur, decreased at the left lower lobe . Hyperdynamic precordium, with perceptible pericardial rub when tilted 45° forward during exhalation, low intensity heart sounds, without presence of murmurs. Soft, depressible abdomen, pain on deep palpation of the right upper quadrant, peristalsis present. Integer extremities, symmetrical and synchronous pulses in all 4 extremities, capillary refill in 2 seconds. The laboratory studies on admission shoes elevated CRP , leukocytosis with neutrophilia (Table 1).

Table – 1 Laboratory Findings

PARAMETER	RESULT
Hematic Biometry	
Hemoglobin	10.2 gr/dL
Hematocrit	30.2 %
Platelets	310,000 µL
Leukocytes	17,890 µL
Neutrophils	15,920 µL
Lymphocytes	650 µL
Monocytes	0
Acute Phase Reactants	
CRP	85 mg/mL
Procalcitonin	<0.2 ng/mL
Blood Chemistry Test	
Glucose	106 mg/dL
Creatinine	0.40 mg/dL

Urea	14.8 mg/dL
Blood urea nitrogen	6.5 mg/dL
Liver Function Profile	
Aspartate Aminotransferase	24 Ui/L
Alanine aminotransferase	11 Ui/L
Alkaline phospatase	124 Ui/L
Gamma-glutamyl transferase	9 Ui/L
Lactic dehydrogenase	174 Ui/L
Total Bilirubin	0.90 mg/dL
Indirect BIlirrubin	0.30 mg/dL
Direct Bilirubin	0.60 mg/dL
Cardiac Profile	
Total creatine kinase	38 Ui/L
Myocardial creatine kinase	1 Ui/L

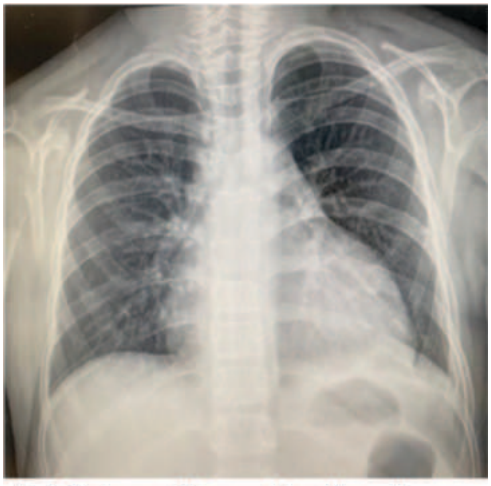

Fig 1. Chest x-ray with increased size of the cardiac silhouette.

In the images: , the chest x-ray showed an increase in the size of the cardiac silhouette (Figure 1). Echocardiogram revealed pericardial effusion and minimal left pleural effusion (Figure 2). The electrocardiogram showed no alterations.

Based on the findings of the physical examination and imaging studies, the diagnosis of AP was made. A battery of studies was carried out in search of the etiology of the disease, within which a TORCH screen, serologies for Epstein Barr Virus (EBV) and Mycoplasma Pneumoniae, as well as a rheumatological profile were requested. The results of the studies are summarized in table 2.

Treatment was started with non-steroidal anti-inflammatory drugs (NSAIDs) based on ibuprofen at a dose of 8 mg kg, human gamma globulin and a systemic steroid. Upon verification of positive serology for M. pneumoniae, treatment was given with clarithromycin (15mgkg/day) for 7 days, the symptoms subsided and there was no recurrence until the time of writing this manuscript.

DISCUSSION

AP is a disease caused by inflammation of the pericardium, usually benign and self-limiting, which can present as an isolated entity or as a manifestation of a systemic disease (1). However, it can also have a chronic behavior and even evolve to cardiac tamponade, which can lead to the death of the patient (1-6).

Table 2, Serological And Antibodies Results.

Laboratory study	Result	Interpretation
Mycoplasma pneumoniae		
Anti-Mycoplasma pneumoniae IgG	39 UI/mL	Positive: Greater than 30.00
Anti-Mycoplasma pneumoniae IgM	25.6 UI/mL	Positive: Greater than 17.00
Virus Epstein Barr		
Anti-Epstein Barr IgG	503 UI/mL	Positive: Greater than 20
Anti-Epstein Barr IgM	10.9 UI/mL	Negative: Less than 20.0
TORCH Profile		
Anti-cytomegalovirus IgG	98.62 UI/mL	Positive: Greater than 15.00
Anti-cytomegalovirus IgM	0.24 S/CO	Negative: Less than 0.85
Anti-Toxoplasma IgG	0.74 INDEX	Negative: Less than 1.60
Anti-Toxoplasma IgM	0.12 INDEX	Negative: Less than 0.50
Anti-Rubeola IgG	52.51 S/CO	Positive: Greater than 10.00
Anti-Rubeola IgM	0.07 S/CO	Negative: Less than 0.75
Anti-Herpes I IgG	0.65 INDEX	Negative: Less than 0.90
Anti-Herpes I IgM	1.15 INDEX	Positive: Greater than 1.00
Anti Herpes II IgG	9.71 INDEX	Positive: Greater than 1.00
Anti Herpes II IgM	1.19 INDEX	Positive: Greater than 1.00
Rheumatological profile		
c-ANCA/PR3	6.20 Ratio	Negativo: Less than 20
p-ANCA/AMPO	4.20 Ratio	Negativo: Less than 20
Anti DS-DNA Antibodies	6 IU/ml	Negativo: Less than 20
Anti-Smith Antibodies	1.8 Ratio	Negativo: Less than 20

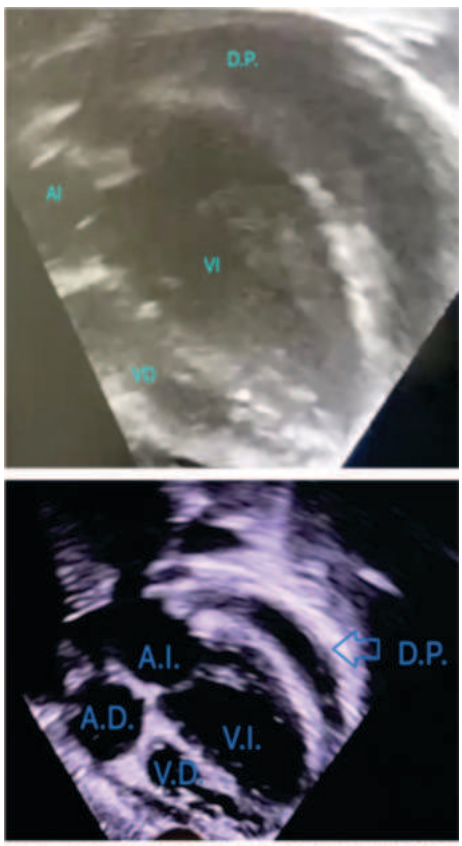

Fig 2. Echocardiogram demonstrating global pericardial effusion of posterior predominance. AI (Left Atrium), VI (Left Ventricle), VD (Right Ventricle), DP (Pericardial Effusion).

In relation to the classification of this entity, the European Society of Cardiology Guidelines for the Diagnosis and Management of Pericardial Diseases (ESCDMPD) of 2016 propose a dichotomous perspective: for infectious and non-infectious causes, however it can also be classified according to its evolution over time as acute, incessant, chronic and recurrent pericarditis. Acute pericarditis is defined by a duration < 4 weeks; Incessant pericarditis is a condition that lasts 4 to 6 weeks without remission. Recurrent pericarditis is called the appearance of new signs and symptoms of pericardial inflammation after an asymptomatic interval of 4 to 6 weeks and, finally, pericarditis with a duration of more than 3 months is called chronic (10).

The diagnosis of AP is established when a patient meets at least 2 of the following 4 criteria: chest pain, pericardial rub, electrocardiographic changes, and pericardial effusion (8,10). Likewise, the 2016 ESC guidelines define the following diagnostic criteria that can be applied for clinical and/or epidemiological purposes: inflammatory markers (elevated CRP, high erythrocyte sedimentation rate, leukocytosis and imaging findings (evidence of inflammation of the pericardium in ECC, computed tomography (CT) or cardiac magnetic resonance (CMR)) (10).

Regarding the etiology of AP, the most common is idiopathic, accounting for 80%-90% of all cases. Viruses are the most frequently confirmed cause in cases of pericarditis with clarified etiology. Related pathogens are: Coxsackie virus, echovirus, EBV, cytomegalovirus (CMV), rubella, herpes zoster, parvovirus B 19, human immunodeficiency virus (HIV), adenovirus, influenza, hepatitis B and C(9). Bacterial etiology is much less common, but has a mortality rate of up to 40% (12). This occurs as a complication of a distant infection and spreads contiguity or hematogenously (12). Less common is direct or iatrogenic inoculation during cardiac surgery. Since the introduction of broad-spectrum antibiotics and the application of vaccines for Haemophilus influenzae and Streptococcus pneumoniae, the incidence of bacterial pericarditis is much lower, estimated at between 5%-10% of all cases, with young children being the most common. most affected(10). Tuberculosis is a rare cause in developed countries, but not in other parts of the world, and should be considered the main etiology in cases of chronic and/or restrictive pericarditis. In endemic regions, infection with tuberculosis and HIV is common (12).

Was en 1979, when Dr. Pönkä performed the first review of heart disease due to Mycoplasma pneumoniae (13). Studied 560 patients with M. pneumoniae infection confirmed by serology, with cardiac lesions observed in 25 patients (4.5%), demonstrating that it is a rare complication, suggesting that an EKG or echocardiogram be performed as a routine study for M. pneumoniae infection (13).

It is important to recognize M. pneumoniae as a common cause of upper and lower respiratory infections in people 5 to 20 years of age. Extrapulmonary manifestations may accompany respiratory symptoms or, much less frequently, be present without it and may occur as a result of direct invasion and/or an autoimmune response in association with M. pneumoniae infection (14). Specifically, cardiac manifestations (myocarditis, pericarditis and myopericarditis) are rare manifestations with an incidence ranging from 1-8.5% in people with serological evidence of infection. Due to the explained above, the pathogenesis of cardiac involvement associated with Mycoplasma Pneumoniae has made it necessary to generate some hypotheses to explain it; The first of them responds to direct invasion of the myocardium through the lymphatic or circulatory system or from the lower respiratory tract due to contamination; The second refers to autoimmune mechanisms as a consequence of a certain molecular mimicry between human and non-human antigens of the pathogen, and the third, to a greater tendency toward intravascular coagulation (14).

Respecting the treatment of AP, in cases where the etiology has been identified, therapy should focus on the underlying cause. Regardless of the cause, one of the cornerstones of the treatment of recurrent AP is the use of NSAIDs with the aim of relieving pain, inflammation and fever (8). Acetylsalicylic acid, ibuprofen and indomethacin are the most commonly prescribed and their administration should be continued until the symptoms resolve and the CRP normalizes, which indicates the resolution of the inflammation (8). The use of NSAIDs corresponds to a class I recommendation according to the ESCDMPD (10).

Systemic steroids are widely used, but their use is controversial. Despite the rapid symptomatic remission and the decrease in acute phase reactants, steroids have been related to an increase in the recurrence rate of pericarditis, so guidelines for the treatment of pericarditis in adults suggest avoiding their use. during the first episode of pericarditis (class III recommendation) (8-10).

Colchicine in low doses (0.5 mg once a day for patients weighing < 70 kg or 0.5 mg twice a day for patients > 70 kg) is a good adjunct to treatment in conjunction with NSAIDs or a corticosteroid in all patients with AP and, above all, in recurrent pericarditis. At low doses, it is well tolerated and can reduce the risk of recurrence by half in both the adult and pediatric populations(9). Biological therapies, such as the nonselective IL-1 antagonist (Anakinra), have been the subject of investigation. The AIRTRIP study evaluates its use in adult patients with recurrent pericarditis dependent on steroids and refractory to colchicine, observing a favorable response. However, studies in the pediatric population have been limited to case reports in which significant improvement has been demonstrated. Finally, and almost anecdotally, the use of human immunoglobulin and TNF-α blockers have been evaluated in the context of recurrent pericarditis with inconclusive results(9).

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