Right-sided endocarditis is exceptional in non-drug add- dict patients without previous heart disease. Few cases have been published, and its diagnosis sometimes pre- senta a significant clinical challenge. We describe a 57- year-old patient with no history of parenteral drug addic- tion or vascular catheter use, who had tricuspid valve endocarditis in a morphologically normal valve. The clini- cal debut was characterized by acute febrile syndrome, purpura (petechia) on the legs, and oligoarthritis. This entity usually has a good prognosis and responds well to treatment, and presents certain common clinical features (persistent fever, pulmonary lesions, anemia and micros- copic hematuria) that can lead the clinician to suspect the diagnosis. However, diagnosis should be based on micro- biological studies (S. aureus is the organism isolated most often) and on echocardiographic findings.

Key words: Endocarditis. Tricuspid. Nonaddict.

INTRODUCTION

Right-sided infectious endocarditis (IE) is common in intravenous drug abusers and accounts for 35%-60% of endocarditis cases in this population.1,2 Prior to the increase in intravenous drug addiction in recent decades, right-sided endocarditis was rare and oc- curred almost exclusively in patients with cardiac mal- formations.3,4 According to current estimates from large series, only 5%-10% of right-sided IE appear in non-addicted patients.5,6 Definite diagnoses of right- sided IE are exceptional in non-addicted patients with- out predisposing heart disease. Very few of such cases have been reported in the literature and their diagnosis can pose a significant clinical challenge.7

We describe the case of a man with no prior history of venipuncture, recently diagnosed in our department with tricuspid valve endocarditis in a morphologically normal valve.

CLINICAL CASE STUDY

A 57-year-old man with a history remarkable only for a duodenal ulcer that had been inactive for several years, was admitted for a febrile syndrome (up to 40°C) of seven days’ duration, severe generalized joint and muscle pain with significant prostration, reddish dot-like skin lesions on both legs, and inflammation of
the left shoulder and elbow. He showed no clinical infectious focus of the respiratory, abdominal, genitourinary or otorhinolaryngological tract. He denied the use of injection drugs or intravenous catheters. He had no alcohol habit, risk behavior for sexually transmitted disease or recent dental procedures. He had no history of rheumatic fever. The patients’ clinical history included a small superficial lesion on the left elbow that had occurred 5 weeks before and took several days to heal.

Physical examination showed a temperature of 38°C, fine end-inspiratory crackles in the lower third of both lung fields, a significant limitation in mobility due to intense joint and muscle pain, signs of inflammation in the left acromioclavicular and sternoclavicular joints and to a lesser degree in the left elbow, and multiple dot-like purpuric-petechial lesions on both legs, which were negative on diascopy. All other aspects of the comprehensive physical examination were normal. There were no skin or mucosal stigmata of endocarditis, venipuncture marks, heart murmurs, lymphadenopathy, signs of meningism, or neurological symptoms.

Laboratory analyses disclosed normocytic anemia (10.5 g/dL of hemoglobin), leukocytosis (23 000/µL) with neutrophilia (85%), liver panel alterations with moderate cholestasis (bilirubin, 2.0 mg/dL; alkaline phosphatase, 416 U/L; GGT, 75 U/L), and microscopic hematuria (20 RBCs per field). Bilateral basal parenchymal involvement was seen in the chest x-ray. Serology for human immunodeficiency virus was negative. Oxacillin-sensitive Staphylococcus aureus grew in all four blood cultures and transesophageal echocardiography showed a mobile, 16x3 mm, filiform vegetation in the left leaflet of the tricuspid valve, which was morphologically normal apart from this feature (Figure 1). There were no signs of vegetation in the mitral, aortic or pulmonary valves, which were all normal. These findings were corroborated in the follow-up transesophageal study performed 24 days later.

Chest computed tomography (CT) (Figure 2) showed multiple peripheral pulmonary nodules, suggesting septic emboli. No alterations were observed in x-rays of the shoulders and elbows. The diagnosis of native tricuspid valve IE with no clear predisposing condition was established. The portal of entry was not precisely identified, although the left elbow skin lesion described by the patient was considered, even though there was no significant evidence of inflammation. The endocarditis was complicated by asymptomatic pulmonary involvement, anemia, vasculitic purpura, and oligoarthritis, probably due to an immunologic mechanism. Skin biopsy was not performed since the lesions were regressing, and the synovial fluid was not studied because of the small amount.

Therapy with parenteral cloxacillin at a dose of 2 g/4 h was started. This treatment was changed to cefazolin after findings consistent with immunooallergic interstitial nephritis were observed (deterioration of renal function with significant peripheral eosinophilia and eosinophiluria, all of which resolved with the change in antibiotic), although there was no histological confirmation. Subsequent challenge study was not considered appropriate. After 6 weeks of specific intravenous antibiotic therapy, blood cultures were nega-
The absence of peripheral stigmata of endocarditis, according to the description of Nandakumar and Raju, signs that constitute the "tricuspid syndrome," is usually not detected at the time that the signs and symptoms present, as occurred in the case described. Among the systemic manifestations, the joint, muscle and skin signs that dominate in the picture presented by our patient are not included in the typical clinical data described for nonaddicted patients with native tricuspid valve IE in the main series. This fact makes the case described, in which left heart involvement was ruled out by 2 transesophageal echocardiograms in an interval of more than 3 weeks, even more unusual.

As in any case of IE, the clinical picture, positive findings on blood culture and echocardiography (the gold standard) are the main diagnostic tools in native tricuspid valve IE. In general the prognosis is excellent with medical treatment (specific antibiotic therapy and support have a high success rate) and the development of heart failure is uncommon. Only 25% of cases require valve replacement or surgery. The indications for the latter option are the same as in any patient with IE: failure of antibiotic therapy with persistent fever, or right heart failure. Mortality associated with native tricuspid valve IE is lower than that described for endocarditis in patients with a predisposing condition.

REFERENCES

12. Clifford SP, Eykym SJ, Oakley CM. Shaphylloccocal tricuspid valve endocarditis in patients with structurally nor-