

LEPTOSPIROSIS: A RARE INFECTIOUS CAUSE OF MYOCARDITIS**Sanae Boulaamayl*, Houda Mouad, Nawal Doghmi, Mohammed Cherti**

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center, Cardiology B
Rabat-Morocco.**SUMMARY**

Myocarditis is described in few cases of leptospirosis. Its pathophysiological mechanisms are justified by a myocardium's immuno-inflammatory state. It is generally asymptomatic, but can be revealed by heart failure or cardiogenic shock. We report a case of 29 years old man with leptospiral myocarditis, whose prognosis is good with antibiotics and appropriate cardiac care.

KEYWORDS: Leptospirosis, myocarditis, echocardiography, CMR.**INTRODUCTION**

Leptospirosis is an ubiquitous anthroponozoonose in tropical countries.

There are wide varieties of clinical presentations. Myocarditis is described in few cases of leptospirosis, despite progress in diagnostic modalities, prognosis remains variable and uncertain.

CASE REPORT

A 29 years old man, with no particular history or cardiovascular risk factors, is admitted for acute heart failure. The history goes back to 6 days before, by the installation of an acute influenza-like syndrome, flamboyant icterus with fever, chills and deterioration of general condition and several episodes of epistaxis. In addition, the patient reports the context of Moorish bath one week before symptoms.

Clinical examination shows a general ill health patient, icteric, diaphoretic and hemodynamically stable, a left gallop, edema of lower limbs with painful mild hepatomegaly. Pleuropulmonary examination shows bilateral lung crepitations.

Twelve-lead electrocardiogram shows sinus tachycardia at 140 c/m, with an incomplete right bundle branch block. [Figure1].

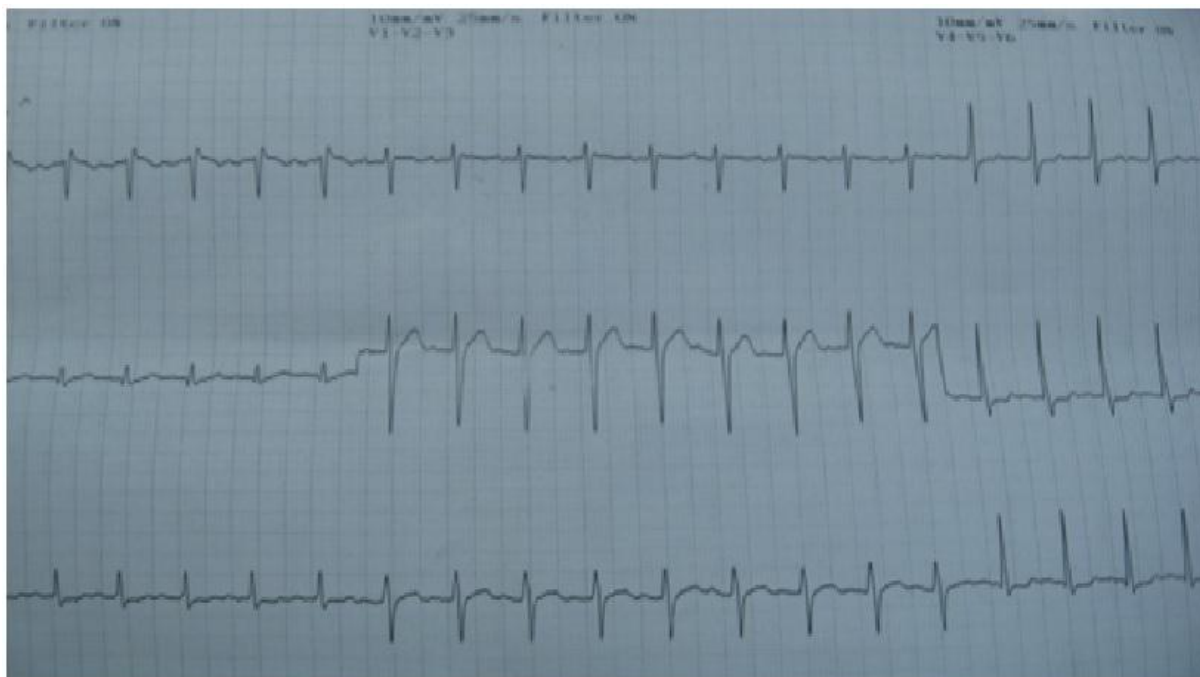


Figure 1: Electrocardiogram (ECG) shows sinus tachycardia at 140 c/min.

A chest radiograph shows cardiomegaly with cardiothoracic index at 0.57 with bilateral pulmonary overload. [Figure2].

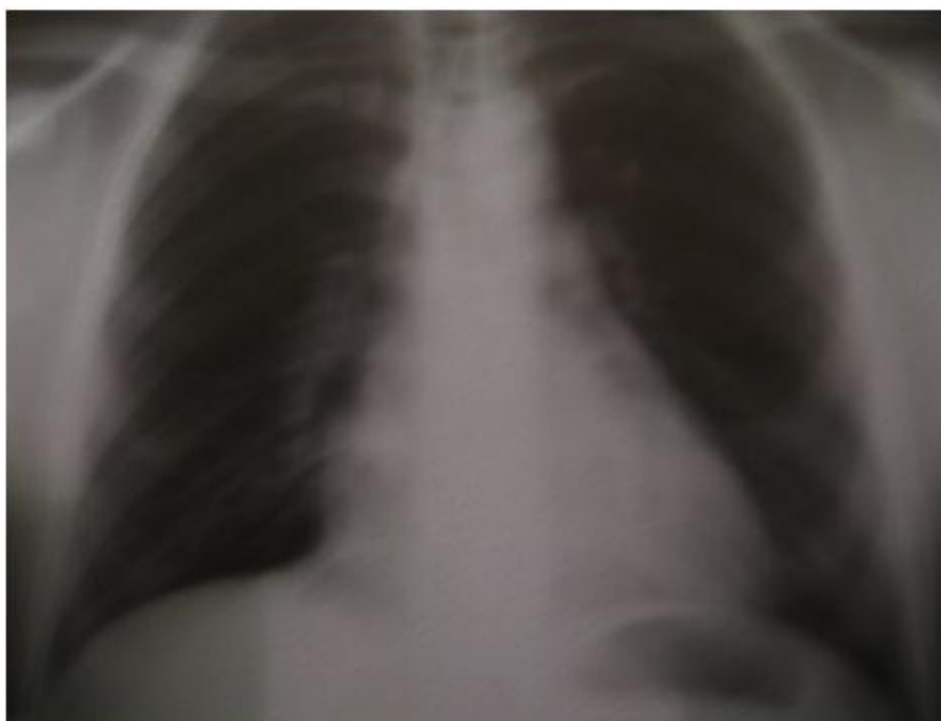


Figure 2: Chest radiograph shows cardiomegaly with cardiothoracic index at 0.57.

Blood tests demonstrate thrombocytopenia (36g/L), leukocytosis (18.7g/L), renal dysfunction with urea (2.53g/L), creatinine (72.2mg/L) and clearance at 18ml/min. The patient has also elevated the CRP (266mg/L), sedimentation rate (83mm) in the first hour, fibrinogen (9.3g/L), hepatic insufficiency cell with AST (267IU/L), ALT (86IU/L), direct bilirubin (207mg/L) and indirect bilirubin (79mg/L), lipase (209IU/L), amylase (213IU/L), creatine kinase (386IU/L), lactate dehydrogenase (374IU/L) and positive troponin (3,57ng/L).

Transthoracic echocardiogram shows no dilated left ventricle (LVEDD=54mm, LVESD=41mm), with distorted systolic function (EF=40%) and global hypokinesis, a dilated right ventricle (RVEDD=40mm), limit systolic function (S velocity=10cm/s). [Figure3].



Figure 3: Transthoracic echocardiogram shows distorted systolic function (EF=40%).

Cardiac MRI performed few days after index admission, demonstrates myocardial late gadolinium enhancement located in the subepicardial to mid-basal anterolateral segment. [Figure4].

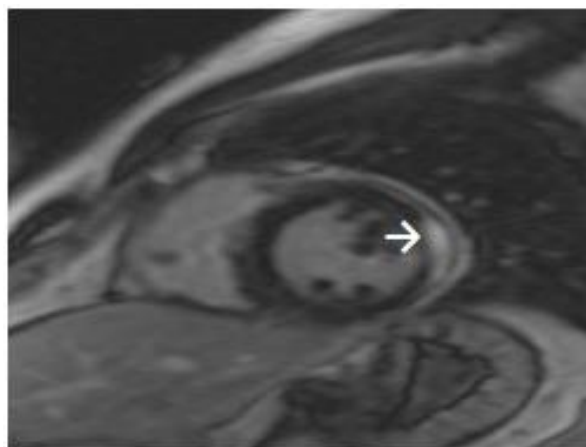


Figure 4: Cardiac MRI showing Epicardial delayed enhancement (arrow) in mid-basal anterolateral segment in short axis.

Due to strong clinical and biological suspicion of leptospirosis, the serology of Martin and Petit is done and leptospira IgM result is found to be positive. The treatment is initiated by antibiotics with cephalosporin (2g/24h during 10days) associated to parenteral rehydration.

The outcome is marked by a clinical improvement with disappearance of icterus, asthenia with normalization of renal and hepatic functions, inflammatory syndrome and troponin's negativation. One month later, echocardiography shows improvement of left and right ventricular systolic function and kinetics [Figure5].

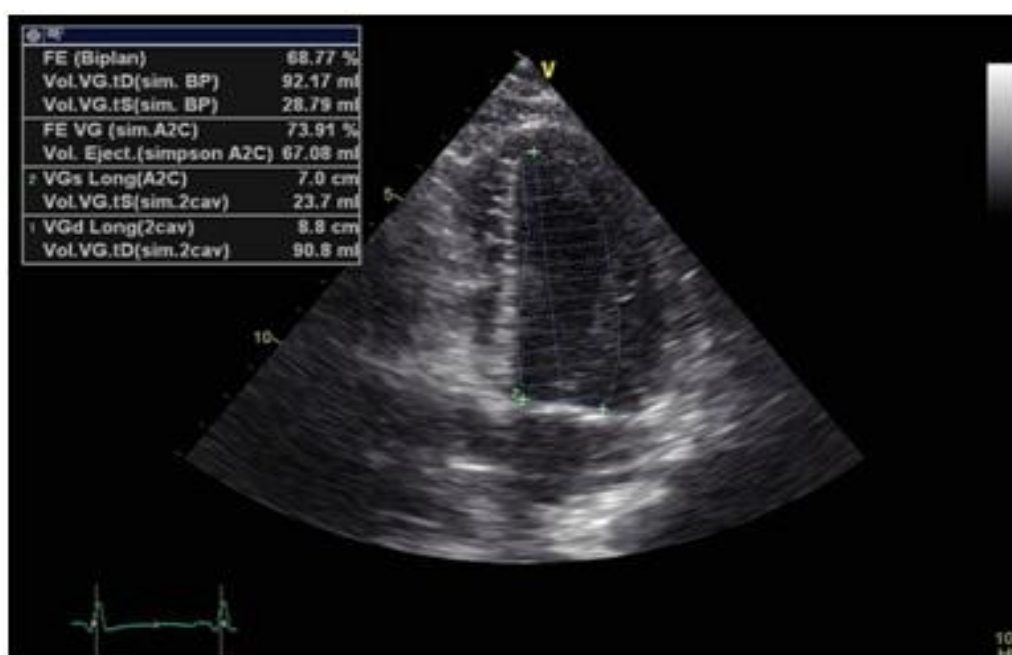


Figure 5: Transthoracic echocardiogram shows the normalization of the Ejection Fonction Left Ventricular.

DISCUSSION

Leptospirosis is an anthroponozoonosis related to extracellular bacteria of spirochete *Leptospira interrogans* whose icterohaemorrhagiae serovar is the most common. It is an endemic disease of tropical countries and in hot and humid regions worldwide. Human infection results from exposure of urine, contaminated water and soil wetted by urine or infected animal tissues. The transmission is either direct, rat bite for example, or most often indirect, by using contaminated water as occurred in our case.^[1]

Clinical presentation of leptospirosis in humans is highly polymorphic and non-specific. There are degrees of severity varying from asymptomatic to severe or even fatal forms. Incubation is usually silent, it takes 5 to 14 days, and the onset of symptoms is manifested by a sudden deterioration of the general condition and a flu-like syndrome. The transition to the illness starting phase could happen after a short period of remission, it is generally combined to severe infectious syndrome associated to multiple organ failure.

Biological diagnosis is difficult, based on research of *Leptospira* in blood and CSF in the first five days and in urine after the 12th day by direct examination and culture. Systematic PCR in the first week of disease should allow faster, more sensitive and specific diagnoses (blood, CSF, urine) and should replace Martin and Petit serology or ELISA antibody screening, which underestimates disease incidence.^[2]

Cardiac involvements of leptospirosis are often underrated. It occurs between the 6th and 12th day of illness but usually remains subclinical, like the case of our patient who presented symptoms one week after contamination, it may be hemodynamic disorders including congestive heart failure, irreversible shock, tachycardia, dyspnea and exceptionally chest pain. Biology seems to contribute by nonspecific increasing of CPK-MB which is usually related to myocard damage and troponin is the specific marker of myocardial injury. In electrical terms, we find rhythm and conduction disorders or repolarization abnormalities. Type I atrioventricular block is the most common trouble.^[3]

Leptospirosis affects the three tunics of the heart and causes pericarditis, endocarditis and less often myocarditis. Echocardiography provides evidence for cardiac disease.^[4] Myocardial damage due to *Leptospira* can cause a localized or diffused systolic dysfunction, manifested by non-specific disorders of myocardial kinetics.^[5]

The development of magnetic resonance imaging should allow an interesting noninvasive approach to myocarditis and shows epicardial LGE positivity as a result of focal expansion of extracellular space, especially in the acute phase.^[6]

Establishing the diagnosis of myocarditis is traditionally dependent on endomyocardial biopsy (EMB) as the gold standard. However, reasons limiting its routine use include sampling error in focal disease and increased risk of procedural complications. Cardiac MRI show good correlations with histological sampling by EMB in diagnosing myocarditis and in patients with biochemical evidence of myocyte damage.^[7]

Leptospirosis is often benign and its evolution is mostly favorable with antibiotics.^[8] Cardiac involvement is a factor of poor prognosis; it is part of the multiple organ failure. And in some series the mortality rate rises to 54% if myocarditis is associated.^[9] Shock and rhythm disorders are the causes of death.^[1-2] However, some studies show that these clinical, electrical and echocardiographic abnormalities may disappear if the treatment is early and properly conducted, as occurred in our case where the evolution was favorable under suitable treatment.

CONCLUSION

Leptospiral myocarditis is rarely described in leptospirosis cases but it is increasingly prevalent due to the use of cardiac MRI. Myocardial biopsy remains the gold standard for the diagnosis of leptospiral myocarditis. Early identification and treatment are recommended to prevent potentially fatal consequences.

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