Neonatal Cholestasis. A Rare Complication of Fetal Tachycardia

To the Editor:

Fetal tachycardia in an infrequent problem (approximate prevalence of 1/10,000 pregnancies)\(^1\) that causes important morbidity and mortality. The diagnosis can be made prenatally incidentally, although the most frequent form of presentation is heart failure and hydrops foetalis.\(^2\) In order to avoid complications of a sustained foetal tachycardia, an intrauterine diagnosis can be made using foetal echocardiography and administering adequate treatment by the transplacental route.\(^1,3,4\) We present a case of a newborn that, in spite of prenatal diagnosis and treatment, developed a cholestasis secondary to foetal tachycardia, a complication which, to date, has rarely been described.\(^5\)

In the ultrasound at 33 weeks gestation, foetal tachycardia of 240 beats/min was observed, with 1:1 atrio-ventricular conduction and pericardial effusion (Figure 1). Transplacental foetal treatment was started by administering digoxin to the mother, resulting in a decrease in the foetal heart rate to 140 beats/min. No toxicity was found in the mother. Ten days after starting treatment, the foetal tachycardia reappeared (223 beats/min); flecainide was added to the treatment, with a good response, but 5 days later, foetal tachycardia was again observed, and delivery was induced. During the 2 hours before birth, a normal foetal heart rate was observed. The baby was born with spontaneous cries, but progressive respiratory depression ensued and therefore the baby had to be examining in the delivery room (Apgar 3/8). When exploring the newborn, a macrosomia, an edematous aspect and a systolic murmur III/VI in the mesocardium stood

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out. A cardiologic study was performed and a Wolff-Parkinson-White (WPW) type pre-excitation was found in the electrocardiogram (Figure 2). No pericardial effusion was found on echocardiography, but a moderate to severe tricuspid insufficiency and mild myocardial dysfunction (ejection fraction, 55%; shortening fraction, 27%) were observed. Neonatal treatment was initiated for the tachycardiomyopathy with furosemide and digoxin. The patient remained stable, with a normal heart rate, at all times, and there was an improvement in the functioning of the left ventricle.

On the fourth day of life, the newborn presented a green ictericia and coluria, without acolia nor other symptoms. Hyperbilirubinemia (20.2 mg/dL; direct, 4.9 mg/dL) was confirmed in the blood work. Direct bilirubin levels >2 mg/dL in newborns and lactating babies is considered indicative of cholestasis, and therefore complementary tests were requested to complete the study. Alterations were not found in the hepatic enzymes (alkaline phosphatase, AST, ALT) nor in its function, except for elevated GGT and LDH (773 and 1255 U/L, respectively). In the abdominal ultrasound no anomalies were found in the biliary path and the rest of the study for neonatal cholestasis made it possible to exclude infectious or metabolic causes. The parameters for cholestasis were progressively decreasing without treatment until they reached normal levels at 18 days of life, and the baby was discharged with digoxin as its only treatment.

After 2 months of life, the baby was reviewed in a visit; it presented a good ponderal weight gain and no symptoms. Its ventricular function appeared completely normal on echocardiogram. As the cardiomyopathy diagnosed at birth had disappeared, treatment with digoxin was suspended and a prophylactic treatment was initiated with amiodarone that was also suspended at the 1 year review. Currently (18 months old), the baby continues to be symptom-free and has not presented any episodes of supraventricular tachycardia.

Foetal tachycardia and atrio-ventricular block (AVB) can produce cholestasis in the neonatal period; the pathophysiological mechanism is not clear, although it seems that it is due to congestion or ischemia. Hepatic alterations can be transitory, as in the case of our patient, but cases have been described that presented irreversible damage (specifically in those with AVB). Therefore, follow-up on liver function should be carried out on all newborns with a history of alterations of the intrauterine rhythm.

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Traumatic Aortic Rupture

To the Editor:

A severe thoracic trauma may affect the heart and/or large blood vessels, usually by a rapid deceleration or crushing. Mortality is increased in the short term, and an early diagnosis is fundamental.

We present a case of a 33-year-old male that suffered a severe thoracic trauma after being run over. He needed mechanical ventilation and inotropic drugs. He presented a murmur that indicated aortic insufficiency. A thoracic computerised tomography (CT) was performed, that showed a pneumothorax and bilateral rib fractures, without signs of aortic dissection. The transthoracic echocardiogram showed severe aortic insufficiency with mild dilation of the aortic root. In the transoesophageal echocardiogram (TEE), a thread-like image was observed, that corresponded to broken right coronary fibers that protruded towards the outflow tract of the left ventricle and severe aortic insufficiency. In the anterior wall of the proximal ascending aorta, there was an image of pseudoaneurysm with rupture of the intimal and medial layers and a medio-intimal flap was observed protruding towards the lumen (Figures 1 and 2). Emergency surgery was performed to replace the aortic valve and implant a Dacron patch in the proximal ascending aorta, with favourable postoperative evolution.

The traumatic rupture of the aorta is a serious pathology, with an early mortality of up to 80%-90%; 32% die in the first 24 hours and 74% in the first 2 weeks. Early surgery improves the vital prognosis.

Aortic lesions from closed trauma happen more frequently in the descending thoracic aorta, at the aortic isthmus level, due to the increase in tension of the wall. Only 5% are found in the ascending aorta. Traditionally, aortography was the technique of reference to diagnose (anterior view).