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Myocardial infarction Type II° in young patient with congenital thrombotic thrombocytopenia purpura (TTP)

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Abstract

A 33-year-old female presented to our Emergency Department (ED) because of hypertensive crisis with Headache and blurred vision. She had been having progressive shortness of breath over 6 Months that worsened acutely on the admission day with no significant past medical history. This patient was not known, that she has TTP, she came to our emercency department, and she was in the same clinis 3 months ago with depression at the psychiatry. She hast wo childs, and she didnot lost any child in the pregnancy.

Keywords: TTP; myocardial infarction typ II°; ADAMTS13; Trponin.

Introduction

The Myocardial infarction is defined as a presence of the myocardial injury with an elevated of the heart biomarkers [1]. The detection of an elevated troponin more than 99th percentile upper rate limit (URL) is defined as myocardial injury [2]. Myocardial infarction has five types, the type II° will be discussed in our case. The Myocardial Infarction type II is due to insufficient blood flow to the ischemic myocardium to meet the increased myocardial oxygen demand of the stressor [3,4]. In our case we introduce a myocardial infarction type II° by congenital TTP. Congenital thrombotic thrombocytopenic purpura caused by the absence of a functional protease (ADAMTS13) that processes von Willebrand factor multimers into smaller fragments [5]. The multimers bind to platelets and initiate abnormal clotting, thrombosis, and hemolysis [6].

Case report (history/examination):

A 33-year-old female presented to our Emergency Department (ED) because of hypertensive crisis with Headache and blurred vision. She had been having progressive shortness of breath over 6 Months that worsened acutely on the admission day with no significant past medical history. The patient was as she arrived the ED, aggressive and that is she directly be intubated. She was afebrile. An electrocardiogram was performed, which showed sinus rhythm with a left ventricle hypertrophy, Sokolow index was 43 mm. This Patient has a hs-TNI at 33490 ng/dl, 4 hours after the admission (normal 2.3-11.6 ng/l), at the admission was only 420 ng/l Coronary angiogram was without a significant epicardial coronary artery disease. For further evaluation we measured the microcirculatory resistance (IMR=72) and coronary flow reverse (CFR =1.2) which were pathologic.

An arterial blood gas showed the following results: pH 7.4 (normal 7.35-7.45), pO2 328 (normal 71-104 mmHg) pCO2 43 (normal 37-43 mmHg), bicarbonate 25.9 (normal 22-26 mmol/L), lactate 1.4 (normal 0.5-2.5 mmol/L), sodium 133 (normal 134-144 mmol/L), potassium 2.7 (normal: 3.5-5.5 mmol/L). Laboratory evaluation revealed markedly elevated creatinine level at 3.5 (normal: 0.7-1.1 mg/dl), the Hemoglobin was 111 g/l (normal 120-160 g/l) with Thrombocytopenia (43 /nl (normal 150-400 /nl), the haptoglobin was low <0,20 (normal 0,30-2,0 g/l), elevated LDH 702 (normal rang <250 U/l).

Considering the above we have laboratory values to suspect TTP (thrombotic thrombocytopenic purpura), in laboratory diagnostics we saw low haptoglobin and increased LDH, for further diagnostics we determined functional protease (ADAMTS13), this was low. SLE could be ruled out with laboratory tests.

In the first cMRI showed a suspected PRES from the brainstem to the medulla oblongata with multiple marrow medullary lesions, from those was some are morphologically associated with chron. inflammatory CNS disease. Suspected supratentorial cerebral edema. No other lesions in the cervicothoracic spinal cord. In the second cMRI showed In comparison to the

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preliminary examination, declining diffusion restriction on the centrum semiovale bilaterally and in the splenium corpus callosum, most likely transient reversible cytotoxic edema. Using CT/thorax and abdomen, we did not see any definable focus of infection in the thoracoabdominal region. Suspicion of increased mediastinal lymph nodes without clearly pathological enlargement. The Echocardiographic showed the picture of a hypertrophic non-obstructive cardiomyopathy.

Discussion

The cardiovascular complications have been reported with TTP. The TTP has a lot of manifestation like chronic renal failure, heart failure, multiple I- Stroke, depression, etc…. [7]. Even if we try to treat the TTP and its manifestation, it is stay as chronic disease [8]. The cause of the manifestations is due Blood clots can form in veins and arteries. Typical locations are in kidney and brain, less common are in the abdominal organs or heart [9]. To diagnose TTP, you will ask about medical and family history. you will ask about symptoms and do a physical exam to look for signs of TTP [10]. We must order one or more blood tests, ADAMTS13 assay, bilirubin test, blood smear, Coombs test, kidney function tests and urine tests and lactate dehydrogenase (LDH) test [11].

TTP can cause life-threatening complications if it is not treated right away. Plasma treatments and medicines are the most common ways to treat TTP. Plasma Treatments are [12,13,14]:

1. Therapeutic plasma exchange (plasmapheresis) is used to treat acquired TTP.

2. Plasma infusion is used to treat inherited TTP

Plasma treatments usually continue until blood tests results and symptoms improve. This can take days or weeks, depending on patient`s condition [15].

Conclusion

The congenital thrombotic thrombocytopenia purpura (TTP) can cause manifestation of cardiac and brain Symptoms in a systematic way, the damage can be reversible, hoe every can be life threatening.

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