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Acute pericarditis associated to onset of diabetes mellitus☆

Pericarditis aguda asociada a debut de diabetes mellitus

Acute pericarditis, an inflammatory process involving the pericardium, is a common condition occurring in association with other diseases and may be the first sign of an underlying systemic disease. In the United States, acute pericarditis occurs in approximately 1 out of each 1000 hospital admissions and in 1% of all patients admitted to the emergency room with chest pain and elevated ST in the electrocardiogram (ECG).[1] A number of infectious and non-infectious causes, including systemic diseases, may be responsible for this condition.[2] Acute pericarditis is more common in males than in females, and in adults as compared to children.[3] The case of a patient diagnosed with acute pericarditis at the onset of diabetes mellitus is reported below.

A 31-year-old male patient with an unremarkable medical history reported polydipsia, polyuria, and asthenia over the preceeding 20 days. In the previous week he had also experienced palpitations and stabbing chest pains unaffected by changes in posture and of increasing severity, which led him to attend the emergency room. Physical examination revealed blood pressure levels of 120/75 mmHg and a heart rate of 85 beats per minute. Cardiopulmonary auscultation revealed no cardiac murmurs or pericardial rub. Laboratory tests showed leukocytosis without neutrophilia (WBCs 14,000, with 68% neutrophils). Venous glucose level was 480 mg/dL, and the results of all other laboratory tests were as follows: creatinine 1.3 mg/dL, creatinine kinase (CK) 4020 IU/L (normal range 55–170 IU/L), CK-MB 2.42 ng/mL (normal value less than 3.6 ng/mL), and troponin I < 0.012 ng/mL (cut-off value for myocardial infarction < 0.12 ng/mL). Venous blood gas test results included: pH 6.93, pCO_2 29 mmHg, pO_2 19 mmHg, and bicarbonate 6.1 mEq/L (normal range 20–24 mEq/L). Urine examination revealed ketone bodies. Based on a diagnosis of onset of diabetes mellitus with ketoacidosis, treatment was started with continuous insulin infusion and intravenous hydration, which improved blood glucose levels and metabolic acidosis. ECG showed sinus rhythm with left axis deviation and diffuse concave ST segment elevation. Chest X-rays showed no changes in pulmonary fields or the mediastinum. Because of the findings of elevated CK in laboratory tests and ECG changes, an echocardiogram was performed, which showed a non-dilated left ventricle with a 60% ejection fraction. No pericardial effusion was found, but pericardial refringence was seen. The patient was diagnosed with acute pericarditis associated with diabetic ketoacidosis and, after evaluation by the cardiology department, treatment was started with colchicine and ibuprofen, which led to a clinical improvement. A gradual improvement was seen in kidney function, and the creatinine level at discharge was 0.7 mg/dL. The results of other biochemical tests performed were glycosylated hemoglobin 10.7% and negative insulin, tyrosine phosphatase IA2 and Langerhans cell antibodies. Thyroid function was normal. The patient was treated with a basal-bolus insulin scheme, and once stable was discharged on this same insulin scheme and ibuprofen 800 mg every eight hours for one week, in a tapering scheme, and colchicine 1 mg daily for three months. In the first outpatient visit after admission, an improvement was seen in blood glucose level, and rapid action insulin was therefore discontinued. A repeat echocardiogram showed no changes, and an ECG revealed sinus rhythm with 60° axis, normal PR interval, narrow QRS, and early repolarization (dolphin back elevation in the inferior aspect, normal precordial leads). Acute pericarditis was considered to be resolved, and colchicine treatment was therefore discontinued. To date, the patient has not re-experienced cardiac symptoms.

Acute pericarditis may be associated with a number of systemic diseases or may occur as an isolated condition. Its most common etiologies include viruses (adenovirus, enterovirus, cytomegalovirus, and influenza, hepatitis B, and herpes simplex viruses), tuberculosis, uremia, tumors, and autoimmunity.

The most common clinical symptoms and signs of pericarditis include chest pain, pericardial rub, ECG changes (ST segment elevation in all leads or shortening of the PR interval), and pericardial effusion. It is generally considered that at least two of these symptoms or signs should be present before acute pericarditis is diagnosed.[4]

In 1971, Bennet and Blake[5] first reported seven cases of pericarditis associated with diabetic ketoacidosis. Few cases have been reported in the literature since then. None of the patients reported by Bennet had chest pains, unlike the patient reported here, who had complained of chest pains over the previous week.

ECG changes seen in diabetic ketoacidosis include ST depression, prolongation of the QT interval, T wave changes, and prominent U waves. The reason for these changes has not been fully elucidated, but they are thought to be secondary to metabolic changes and changes in plasma potassium levels,[6,7] which cause dehydration of the pericardial layers.[8]

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Medical treatment of acute pericarditis associated with the onset of diabetes mellitus includes water and electrolyte replacement, insulin treatment, and analgesics. Once the internal environment is stable, ECG changes persist for 48–72 h, and this type of pericarditis is therefore considered to be benign in nature.[3]

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Hyperthyroidism due to Graves-Basedow disease in a woman refractory to thyroid hormones☆

Hipertiroidismo por enfermedad de Graves Basedow en mujer con resistencia a las hormonas tiroideas

Sir,

Resistance to thyroid hormone (RTH) is an uncommon syndrome in which there is a decreased sensitivity to thyroid hormones. It is characterized by elevated free T3 and T4 levels with normal or slightly increased TSH levels. RTH may coexist with autoimmune thyroid disease, and an association with Hashimoto thyroiditis has more frequently been documented.

We report the case of a female patient with RTH who developed hyperthyroidism due to Graves-Basedow disease (GBD) and methimazole-induced toxic hepatitis.

This was a 39-year-old woman referred for high TSH and free T4 levels. No symptoms suggesting thyroid dysfunction were found in a problem-oriented medical history. The patient did not complain of headache or visual changes. She was not aware of any personal history of interest, and physical examination revealed 49.7 kg of weight, 1.48 m of height, a heart rate of 87 beats per minute, normal blood pressure, and palpable grade Ib-II goiter. Laboratory test results were as follows: free T4 2.81 ng/dL (0.9–1.9), TSH 6.81 μU/mL (0.3–4.5), and total T3 2.28 ng/mL (0.8–2). Thyroglobulin and peroxidase antibodies were positive (455 IU/mL (0–115) and 274 IU/mL (0–32) respectively). The results of alpha subunits of glycoprotein hormones, cortisol, estradiol, gonadotropins, and prolactin were normal. Measurements of T3 and T4 antibodies provided normal results. Ultrasound images were consistent with multinodular goiter. The biggest nodules were 1 cm and 1.3 cm in size and had a benign appearance in ultrasonography. Based on these data, resistance to thyroid hormone was suspected. The patient was asked to provide laboratory tests from first-degree relatives. Tests from her brother, who was also symptom-free, also showed high free T4 and TSH levels (3.98 ng/dL and 6.72 μU/mL respectively). Finally, a genetic study was performed by sequencing exons 3–10 of the THRB (thyroid hormone receptor beta) gene, and a heterozygous mutation was found in exon 10, consisting of c.1357C>T;p.Pro453Ser.[1]

In subsequent visits, TSH levels ranged from 6.6 to 8.1, and free T4 levels from 2.8 to 3.2. Low TSH levels were found three years after diagnosis, and repeated tests showed TSH <0.014, free T4 >7.77, and TSI 21.12 IU/L (positive >1.5). The patient reported a moderate weight decrease (2 kg in four months), a slight increase in nervousness, palpitations, and occasional tremor. No ophthalmopathy was found. A thyroid scan (Tc^{99}) showed a thyroid gland of normal location and shape with a diffusely increased uptake.

GBD was diagnosed, and treatment was started with methimazole 30 mg daily in descending doses. High transaminase levels were found three months later (see Table 1), and treatment was therefore discontinued after showing improvement. Propylthiouracil was instead administered, but markedly high transaminase levels again occurred, associated with normal bilirubin levels, normal liver ultrasound examination, negative serologic tests for hepatitis B and C, and negative LKM, ANA, AMA antibodies. Propylthiouracil was discontinued based on the diagnosis of thionamide-induced toxic hepatitis, and the patient was referred for radioiodine treatment.

She was administered 12 mCi of radioiodine six months after the start of antithyroid medication. One month later, hormone levels consistent with hypothyroidism (TSH 50.63

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