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SCIENTIFIC LETTERS

Pituitary apoplexy after myocardial perfusion scintigraphy☆

Apoplejía hipofisaria tras gammagrafía de perfusión miocárdica

Pituitary apoplexy (PA) is an uncommon condition caused by infarction or hemorrhage in the pituitary gland, usually inside an adenoma. Acute damage leads to pituitary gland expansion and secondary compression of perisellar structures, causing neurological and endocrinological deficiencies of different severity that may become permanent and even life-threatening. The classical syndrome consists of sudden headache associated with vomiting, visual deficiency, ophthalmoplegia, and impaired consciousness.[1]

A case of pituitary apoplexy occurring after myocardial perfusion scintigraphy (SPECT) is reported here. This occurred in a 74-year-old female patient with cardiovascular risk factors who had an implanted pacemaker due to sinus disease and a history of ischemic heart disease starting 17 years previously with unstable angina. The patient had undergone percutaneous revascularization for a single coronary lesion in the anterior descending artery. Her history included a non-functioning pituitary macroadenoma diagnosed seven years before. Previous laboratory tests showed impaired pituitary function, with TSH levels of 2.6 μIU/mL, T4 levels of 0.7 ng/dL, and T3 levels of 2.1 pg/mL three years earlier, but the patient was undergoing irregular monitoring at another center and did not receive replacement therapy. The patient reported an intolerance to acetylsalicylic acid and was being treated with ticlopidine and lorazepam. She was admitted to hospital for instable angina work-up. Treatment was started with low molecular weight heparin at a dose of 1 mg/kg/kg twice daily, clopidogrel, carvedilol, nitrates, and atorvastatin, and the patient remained stable from the cardiovascular viewpoint from the start of treatment. Myocardial perfusion SPECT with Tc^{99} was performed at rest and after stimulus with dipyridamole on the fourth day of admission. On that day, the patient experienced malaise and nausea, and high blood pressure levels were found at the end of the test. No other changes were seen. In the ward, the patient had a BP of 160/99 mmHg. In the following 24 h she continued to have a feeling of nausea and vomiting and experienced high blood pressure, headache, and right ophthalmoplegia consisting of incomplete paralysis of the third cranial nerve with ptosis and mydriasis. Since magnetic resonance imaging could not be performed as the patient had an implanted pacemaker, an urgent CT scan of the head was requested to rule out bleeding complications. The scan showed a pituitary macroadenoma with no evidence of bleeding inside the tumor (Fig. 1). Laboratory tests which were performed because of a clinical suspicion of PA confirmed panhypopituitarism, showing the following values: TSH 0.61 μIU/mL, T4 0.6 ng/dL, T3 1.6 pg/mL, cortisol 3.2 μg/dL, prolactin 1 ng/mL, FSH 1.7 IU/mL, and LH 0.6 mIU/mL. Hormone replacement therapy was

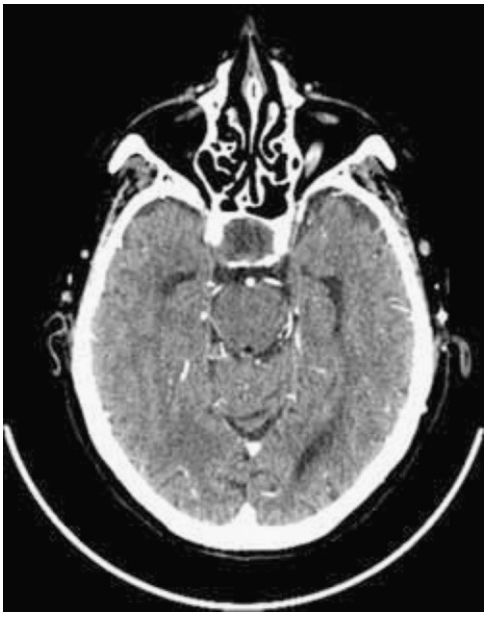

Figure 1 Pituitary macroadenoma extending to chiasmatic cisterns, with no evidence of bleeding inside the tumor.

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therefore started with steroids at doses for acute adrenal insufficiency and levothyroxine 25 μg/d. Ophthalmological examination revealed a significant visual acuity (VA) deficiency with paracentral islets in campimetry of both eyes. The patient remained hemodynamically and neurologically stable after the start of replacement therapy, but experienced recurrent angina episodes in the following days despite medical treatment, which led to the prioritization of coronary angiography over neurosurgical surgery. Three-vessel coronary disease was found, and percutaneous revascularization was performed with three stents coated with monoclonal antibodies being implanted. Because of the need to maintain double antiaggregation therapy for at least one month and neurological stability, neurosurgical surgery was delayed. Three months later, the tumor was resected through an endonasal approach with no complications. Histopathological examination revealed tissue consistent with a completely infarcted pituitary adenoma, with blood remnants and no evidence of viable tumor. Repeat ophthalmological examination at two months showed a recovery from visual deficiency, with a VA of 0.8 in both eyes, similar to that found one year before, and the disappearance of ophthalmoplegia. The patient is currently stable on hormone replacement therapy with hydrocortisone and levothyroxine.

PA is an uncommon endocrinological emergency whose pathogenesis has not been fully elucidated yet. Various theories have been proposed, from insufficient vascular supply due to high metabolic requirements[2] to abnormal adenoma vascularization which makes it more susceptible to bleeding as compared to other central nervous system tumors.[1] According to the reported data, most adenomas are non-functioning,[3] so that PA may often be the form of presentation of a tumor not previously diagnosed. The actual incidence of PA is unknown, but it is estimated to occur in 0.6–21% of pituitary tumors.[3–5] An incidental finding of pituitary hemorrhage and/or infarct in imaging tests or histopathological samples is more common, but these cases are not true PAs. PA usually occurs spontaneously, but several factors possibly contributing to its development have been reported. Thus, it has been associated with head trauma, radiotherapy, arterial hypertension or hypotension, major surgery (particularly cardiac surgery, due to blood pressure changes and anticogulant treatment), hormone stimulation tests or drug treatment with GnRH analogues, estrogens, dopamine agonists, and antiplatelet or anticoagulant drugs. PA has also been related to various diagnostic or therapeutic techniques using heparin or contrast media,[6,7] but the pathophysiology of the process is unknown. This is, to our knowledge, the first case of PA reported after myocardial perfusion SPECT. The time sequence suggests that apoplexy was triggered by the procedure, maybe in relation to an unnoticed episode of arterial hypotension caused by dipyridamole. Blood pressure levels would have increased as a response to the stimulus or the stress situation, or simply due to fluctuation. A limitation of the reported case was that magnetic resonance imaging, the imaging test of choice, could not be performed.[3] However, although bleeding inside the tumor was not seen in the CT scan, which has a low sensitivity to the detection of hemorrhage,[3] histopathology showed the presence of blood remnants. Despite this, it would appear that a greater hemorrhagic component would have been expected in a patient on anticoagulant treatment such as the one reported, and it is a reasonable conclusion that the starting event was ischemic in nature. As regards treatment, it is generally agreed that it should initially be aimed at the control of water and electrolyte balance and the administration of hormone replacement therapy, particularly high-dose glucocorticoids. However, greater controversy exists as to which patients should undergo surgery and when. If impaired consciousness, a significant visual deficiency, or progressive symptoms exist, early surgical decompression in the first week is recommended,[3,8,9] but the criteria defining a significant neuro-ophthalmological deficiency are not clear.[9] In this regard, optical coherence tomograpy has emerged in recent years as a useful tool for visual diagnosis and prognosis in these patients. Visual recovery will depend on the timing of surgery, but may occur even when the surgical procedure has to be delayed.[10] The fact that this occurred in our patient, in whom surgery was indicated but had to be delayed because of her concomitant cardiac disease, supports this theory. By contrast, ophthalmoplegia has a better prognosis with both conservative and surgical treatment.[3] In other situations, conservative management may be adequate, but treatment should be individualized in each patient. Finally, our case report is not only intended to describe the eventual relationship of PA with the diagnostic test, but also to emphasize the importance of maintaining a high index of suspicion when making a diagnosis that continues to be essentially clinical.

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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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