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CASE REPORT

Hypercalcemic crisis and primary hyperparathyroidism: Cause of an unusual electrical storm☆

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Hyperparathyroidism;
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Abstract Hypercalcemia is a known cause of heart rhythm disorders, but it is rarely associated with ventricular arrhythmias. The authors present the case of a 53-year-old man with ischemic and alcoholic dilated cardiomyopathy and severely reduced ejection fraction, with a cardiac resynchronization therapy-cardioverter-defibrillator (ICD) device, who was admitted to the emergency department due to an electrical storm with multiple appropriate ICD shocks, refractory to antiarrhythmic therapy. Etiological investigation documented severe hypercalcemia secondary to previously undiagnosed primary hyperparathyroidism. The episodes of ventricular tachycardia only ceased after serum calcium levels were reduced.

PALAVRAS-CHAVE
Hiperparatiroidismo;
Hipercalcemia;
Taquicardia ventricular

Crise hipercalcémica e hiperparatiroidismo primário: causa de tempestade arrítmica invulgar

Resumo A hipercalcemia é uma causa conhecida de perturbações do ritmo cardíaco, contudo, a sua associação a arritmias ventriculares é rara. Os autores apresentam o caso clínico de um doente de 53 anos de idade, com cardiomiopatia dilatada de etiologia isquémica e etanólica, e grave compromisso da função sistólica global, portador de sistema de ressincronização cardíaca (CRT) com cardioversor desfibrilhador (CDI), admitido no serviço de urgência por tempestade

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arrítmica, com múltiplos choques de CDI apropriados, refratária a terapêutica antiarrítmica. Na investigação etiológica foi documentada hipercalcemia grave secundária a hiperparatiroidismo primário até então desconhecido. Somente após redução da calcemia se observou cessação dos episódios de taquicardia ventricular.

Case report

A 53-year-old Caucasian man with several cardiovascular risk factors (hypertension and heavy smoking and alcohol consumption) had a history of non-ST-elevation acute coronary syndrome at the age of 49 years and severe disease of the mid and distal right coronary artery, which were treated by angioplasty and implantation of two bare-metal stents. Because of persistent severe left ventricular dysfunction (biplane left ventricular ejection fraction of 15%) even on optimal medical therapy, an atrioventricular cardiac resynchronization therapy (CRT)/implantable cardioverter-defibrillator (ICD) system was implanted.

Three years later, he experienced an episode of monomorphic ventricular tachycardia (VT) with a pattern of complete right bundle branch block and superior axis prompting an ICD shock followed by chest pain and elevation of troponin I levels (peak 1.69 mg/l, cutoff <0.07). He underwent repeat coronary angiography that showed in-stent occlusion, treated by angioplasty and placement of three drug-eluting stents. Among the patient's other known personal history were smoking-related chronic obstructive pulmonary disease, erosive gastritis and peptic ulcer. He was medicated as an outpatient with losartan, bisoprolol, spirolactone, furosemide, aspirin, clopidogrel, amiodarone, simvastatin and pantograph. He remained in New York Heart Association class II from that time.

The patient was readmitted to our hospital one year after the previous hospitalization due to multiple ICD shocks, preceded by syncope, with transient recovery of consciousness following each shock, beginning 24 hours previously and increasing in frequency in the hour before admission. He had discontinued his medication in the previous five days due to financial difficulties, since when he had complained of anorexia, asthenia, constipation and dizziness. On admission he was conscious, oriented, and sweating but with no signs of peripheral hypoperfusion. The electrocardiogram (ECG) documented monomorphic VT with complete left bundle branch block and a rate of 300 bpm (Figure 1). A 300-mg bolus of amiodarone was administered followed by intravenous (IV) perfusion at 4 μg/kg/min, but recurrence of VT (with constant morphology on telemetry) was observed; two boluses of IV lidocaine (total 150 mg) were administered but also had no effect. The patient received multiple ICD shocks immediately after arrival at the hospital, resulting in the rapid exhaustion of the generator before the cardiologist was able to inhibit the therapies with the aid of a magnet. During periods of sinus rhythm, the ECG documented QRS with complete right bundle branch block, PR interval at the upper normal level, and corrected QT interval of 405 ms, with no signs of acute ischemia (Figure 2). Transthoracic echocardiography showed a severely dilated left ventricle, with diffuse hypokinesia and severely impaired global systolic function. Interrogation of the CRT-ICD revealed various episodes of VT in the previous 24 hours, with a total of 48 appropriate shocks (Figure 3), and the generator at the end of battery life. Given the patient's worsening heart failure and persistence of the arrhythmic storm, he was intubated and given propofol and midazolam for sedation and analgesia. Laboratory tests at admission showed acute kidney injury (Acute Kidney Injury Network stage 1) with creatinine 1.5 mg/dl and urea 101 mg/dl; mild hypokalemia (potassium 3.0 mmol/l, normal range 3.5-5.1); severe hypercalcemia (calcium 18.5 mg/dl, normal range 8.6-10; Ca^{2+} 2.46 mmol/l, normal range 1.13-1.32); and slight elevation of troponin I (peak 1.5 mg/dl, cutoff <0.07). The hypokalemia was immediately corrected.

Since the patient's monomorphic VT was not associated with pain or anginal equivalents, and there were no signs of acute ischemia on the baseline ECG, the cause of the arrhythmia was assumed to be the presence of severe hypercalcemia. With the support of internal medicine and endocrinology specialists, pharmacological measures were instituted to correct hypercalcemia with pamidronate, zolendronic acid, furosemide and hydrocortisone. However, since the hypercalcemia was severe and refractory to the above treatment, it was decided to begin continuous venovenous hemodiafiltration (CVVHDF); this resulted in progressive reduction in the frequency and duration of TV episodes, which were no longer sustained and ceased completely when calcemia reached 15.8 mg/dl. The patient was extubated and CVVHDF was discontinued after 48 hours, but furosemide (60 mg/day) was continued to maintain normocalcemia. The possibility of ablation to treat the VT was discussed but was not performed since the arrhythmia did not recur after correction of hypercalcemia.

Etiological investigation of hypercalcemia showed marked elevation of parathyroid hormone (PTH) (1020 pg/ml, normal range 14-72), and normal albuminemia (3.5 g/dl, normal range 3.2-4.9), suggesting primary hyperparathyroidism (PHPT). Cervical ultrasound revealed a well-defined hypoechogenic homogeneous solid mass topographically slightly posterior and inferior to the left lobe of the thyroid gland suggestive of a parathyroid adenoma, and scintigraphy of the parathyroid glands documented a functional lesion of the left inferior parathyroid. These