

Can Hashimoto Thyroiditis Cause Massive Pericardial Effusion?

The Editor,

Sir,

Cardiac tamponade and pericardial effusion are relatively frequent complications of untreated severe hypothyroidism in adults. This is an uncommon presentation or complication of hypothyroidism in children (1, 2). A six-year old boy was admitted to hospital with fatigue, swelling all over the body and breathlessness for over a year and difficulty in walking and getting up from a sitting position for four months. On physical examination, pulse rate was 108 beats/minute, respiratory rate was 36/minute and blood pressure was 90/50 mmHg. He had facial oedema, dry skin and nonpitting oedema of the lower extremities. Respiratory sounds in the base of the right hemithorax were silent. Heart sounds were distant and muffled. The liver was palpable 4 cm below the costal margin. Laboratory evaluations were normal. The chest radiograph showed cardiomegaly (cardiothoracic index 0.72). An electrocardiogram revealed sinus bradycardia, with a low p wave and QRS complexes. Echocardiogram showed massive pericardial effusion of more than 3 cm (Figure). The patient underwent pericardiocentesis, yielding

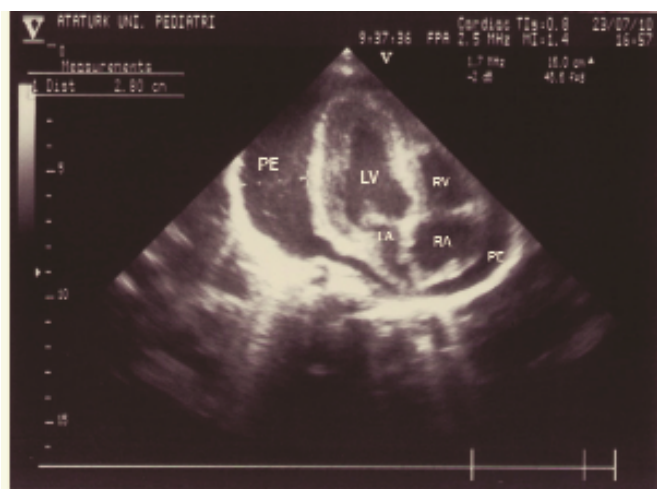


Figure: Pericardial effusion appearance in the four-chamber view in two-dimensional echocardiography. Diameters of pericardial effusion (PE): right ventricle (RV) 33 mm, left ventricle (LV) 22 mm. LA = left atrium, RA = right atrium

approximately 400 ml of clear fluid. Pericardial fluid was transudative. The fluid showed no organisms on culture. Pericardiocentesis was repeated after three days. All other

causes of massive pericardial effusion (infection, neoplasm, trauma, uraemia *etc*) except hypothyroidism were ruled out. Thyroid function results were consistent with the diagnosis of Hashimoto's disease: thyroid stimulating hormone (TSH) was > 100 uIU/mL (0.27–4.2 uIU/mL), free thyroxine 0.2 ng/dL (0.93–1.7 ng/dL), free triiodothyronine 0.8 ng/dL (1.8–4.6 ng/dL), antithyroid peroxidase antibodies 776 IU/mL (0–150 IU/mL) and antithyroglobulin antibody 971 IU/mL (0–150 IU/mL). He was started on 25 µg per day of levothyroxine orally. The dose was increased by 25 µg every week up to 75 µg. The patient responded well to the commencement of oral thyroxine treatment. After thyroid replacement therapy, the pericardial effusion was undetectable on repeat echocardiogram three weeks later.

Cardiac tamponade and massive pericardial effusion are uncommon complications of acquired hypothyroidism in children (1, 3). The systemic hypometabolism that is associated with hypothyroidism results in a decrease in cardiac output that is mediated by reductions in heart rate and contractility (1, 2, 4–6). In our case, diagnosis of cardiac tamponade was based on clinical presentation and findings on echocardiography. Hypothyroidism, as the cause of the pericardial effusion and tamponade, was diagnosed by exclusion criteria, because other afflictions are the most frequent causes of nontraumatic pericardial effusion (1, 4–6).

Treatment of hypothyroidism is always mandatory following tamponade drainage, because, generally, a residue of the effusate following pericardiocentesis disappears after appropriate therapy (5, 7). Hypothyroidism should be ruled out as a hidden underlying cause of pericardial effusion. Pericardial effusion can be easily reversed with thyroid replacement but pericardiocentesis is necessary when tamponade develops.

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Widespread Idiopathic Calcinosis Cutis: A Case Report

The Editor,

Sir,

A three-month-old female patient presented with subcutaneous hardening of the skin on the back and lower left abdominal quadrant. The lesions had been present since birth and were spreading. She had no history of chronic disease, drug use or hospitalization. There was also no family history of similar complaint. On physical examination, body weight, height and head circumference measurements were normal. Her whole body was covered with plaquelike, immobile subcutaneous nodules, that had infiltrated the skin and were particularly apparent on the trunk and back (Fig. 1). Other

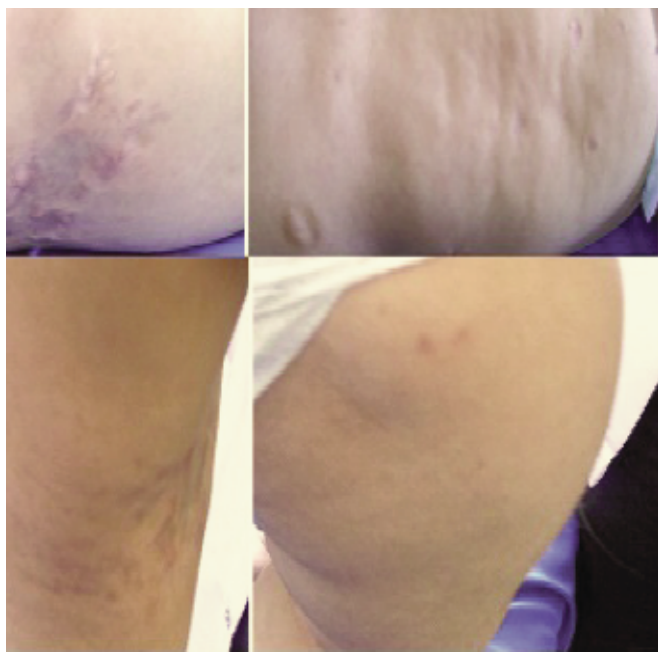


Fig. 1: Calcified lesions spread all over the body.

systemic examinations were normal. At laboratory examination, serum calcium, phosphorus, alkaline phosphatase, parathormone, 25-OH vitamin D levels and measurement of 24-hour calcium excretion in urine were within normal range. Complete blood count, erythrocyte sedimentation rate, urine test, glucose, renal function tests, liver function tests, thyroid function tests, antinuclear antibody, anti-DNA, anti-SM, anti-neutrophil cytoplasmic antibodies and serum C3, C4 levels were also normal. Malignancy or pathologic signs were not found at radiological examination. Calcified nodules localized in the subcutaneous fat tissue were identified by ultrasound. Incisional biopsy was performed. The skin tissue was stained with haematoxylin and eosin (H&E) for pathological investigation. On light-microscopic examination, calcified nodules were seen in subcutaneous fat and dermis. The lesions consisted of granules of calcified material and/or amorphous masses of calcium. There was also a small amount of inflammatory infiltrate around the calcified nodule. Pathological investigation was compatible with calcinosis cutis (Fig. 2). Idiopathic calcinosis cutis was

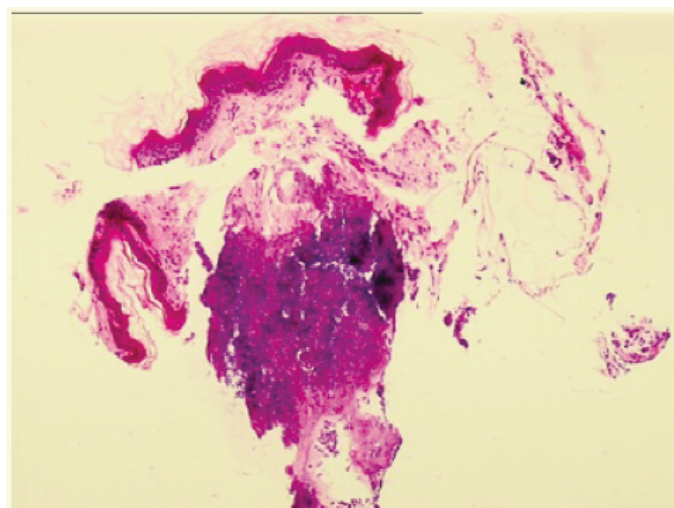


Fig. 2: Subepidermal calcified nodule.

diagnosed on the basis of clinical, radiological and pathological results.

Calcinosis cutis is a rare condition in childhood. Cutaneous lesions are generally in the form of nodules or plaques on the abdomen, thigh and hip regions (1). The pathogenesis of this condition is unknown and probably multifactorial. Calcinosis cutis may be classified as metastatic, dystrophic, iatrogenic and idiopathic (1, 2). Idiopathic calcinosis cutis is a rare form and occurs in apparently normal tissue in the absence of known tissue injury, systemic metabolic effect or collagen vascular disease and percutaneous exposure to calcium. Clinical and laboratory findings are normal and there are no systemic diseases. Calcium deposits are present in subcutaneous tissues. The reason for

calcium and phosphate collection is unclear. Calcium and phosphate excretion may be normal (1, 3). Serum calcium and phosphorus levels and calcium and phosphate expulsion were within normal limits in the index patient. Metastatic calcinosis cutis was excluded radiologically and by laboratory tests. There was no infection and connective tissue disease that might cause dystrophic calcification, and laboratory parameters that might suggest collagen tissue disease were negative. Thus, our patient was diagnosed with idiopathic calcinosis cutis. Calcinosis cutis is generally localized in one area, although rarely, as in our case, it can be spread over the entire body. Treatment for calcinosis cutis consists of the correction of the underlying pathology and treatments emerging complications. Spontaneous resolution of lesions has also been reported in idiopathic cases. Various treatments are used, such as bisphosphonates, a low calcium diet, intralesional corticosteroids, diltiazem, aluminum hydroxide, colchicine, warfarin, probenecid, salicylate, and minocycline, but are inadequate in the treatment of developed calcinosis cutis. Total excision has been reported to be superior to medical treatment in idiopathic calcinosis cutis, but this is the least favourable treatment option (4, 5). No medical and surgical treatment was applied to the patient. Instead, dietary recommendations were given to her mother. The patient was followed-up for 10 months and there was no change of the calcinosis cutis.

In conclusion, when subcutaneous calcified mass is determined, differential diagnosis should be performed and calcinosis cutis should be considered.

Keywords: Calcinosis cutis, child, skin

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