Disodium etidronate has been claimed to delay and partially prevent postoperative recurrence of heterotopic ossification.⁷

The immature bone, cellular proliferation and zoning characteristic of myositis ossificans were absent.⁸ In the histological differential diagnosis we considered a neoplasm of the xiphoid with which the lesion appears to be in continuity. However, the lack of immature or atypical elements was more in keeping with a metaplastic phenomenon. An osteochondroma consists of bone and cartilage, but typically has a cartilaginous cap with bone beneath.⁹ In our patient, the cartilage and bone were mixed

together, with no cap-like structure. Bizarre parosteal osteochondromatous proliferation was also considered, but this tends to occur in the hands and feet, and no bizarre chondrocytes were present.¹⁰

We have here described a case of metaplastic bone formation with marrow spaces and cartilage in an extraskeletal site. The condition must be considered in swellings affecting mid-line abdominal scars.

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High-altitude illness induced by tooth root infection

J Finsterer

Summary

High-altitude illness may occur after recent pulmonary infection, but high-altitude illness after root canal therapy has not been described previously. A 44-year-old man is presented who skied to a 3333 m high peak in the Eastern Alps one day after he had undergone root canal therapy because of a tooth root infection. After 4 hours above 3000 m severe symptoms of high-altitude illness, including pulmonary oedema, developed. His condition improved after immediate descent. The next day he presented with local and general signs of infection which were successfully treated with gingival incisions and antibiotics. In conclusion, acute tooth root infection and root canal therapy may induce high-altitude illness at an altitude just above 3000 m.

Keywords: high-altitude illness; pulmonary oedema; root canal therapy; tooth root infection

Usually, high-altitude illness occurs at elevations >2500 m and includes mountain sickness, high-altitude pulmonary oedema, cerebral oedema and retinal haemorrhage.¹

Whether or not recent infection predisposes to high-altitude illness is controversial.^{2,3} High-altitude illness associated with tooth root infection, as in the following case, has not been reported previously.

Case report

Four weeks before a skiing trip, a previously fit 44-year-old man experienced increasing fatigue and declining fitness. There was no indication of an infectious disease, although root canal therapy for a granuloma of the first left upper incisor had been proposed by the treating dentist 2 month earlier but declined by the patient. Two weeks before the trip, the patient experienced aching of the first left upper incisor with an associated gingival inflammation over its root column. Radiography was indicative of tooth canal infection, and the patient underwent root canal therapy, leaving the root canal open, one day before starting his skiing trip.

After slowly ascending to 1720 m by bus, he took a cable car to reach 3170 m within one hour. He had ascended to altitudes >3000 m within one day without major problems several times before. This time he observed unusual

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breathlessness and fatigue when ascending to 3333 m, prompting him to take a rest every few steps. Despite taking an unusual 2 hours to reach the summit and return, and despite persisting symptoms, he set out to climb another nearby peak, but dyspnoea and tiredness forced him to abandon this attempt. Though he was trained as a skiing teacher, he moved like a beginner, could hardly stay on the lift, and had to take several rests before descending to 2900 m. When skiing further down, the frequency of pauses increased and his breathing failed to return to normal. Disorientation and nausea occurred. After trying to descend further, he fell and could not get up. He started to lose consciousness and recognised paresthesias in both arms and legs. He managed to attract the attention of a passing physician to whom he presented with drowsiness, severe dyspnoea, tachypnoea, coughing, cyanosis and tachycardia. Though auscultation was not possible in this acute situation, the physician suspected high-altitude oedema and organised an immediate transfer to an altitude of 2300 m with a snow-tending machine. There the patient's condition markedly improved, but he was not able to walk unaided before reaching 1720 m.

During the following night, a gingival abscess developed. Blood chemistry the next day revealed a leucocytosis of 11.9 g/l (normal 4–10 g/l), C-reactive protein 9.0 mg/l (0–5 mg/l) and serum creatine kinase 300 U/l (0–72 g/l) with normal MB-fraction. Blood culture was negative and chest X-ray and electrocardiogram were normal. The tooth infection was successfully treated by repeated gingival incisions and clindamycin for 8 days.

Discussion

The pathophysiological background of high-altitude pulmonary oedema is poorly understood. One theory proposes primary cerebral hypoxia causing cerebral vasodilatation, elevated cerebral capillary pressure and increased sympathetic response.⁴ Another theory is based on the assumption that, due to the low pO_2 at high altitude, liquid may penetrate into

the alveolar cave. Respiratory infection, impairing oxygen uptake and reducing oxygen delivery at the tissue level, may have an additional effect.¹ The release of vasoactive, inflammatory mediators during infection may directly aggravate high-altitude illness.¹ There are limited and controversial data on the relationship between recent infection and the occurrence of high-altitude illness.¹ Pneumonia and mediastinitis have been reported to predispose to high-altitude illness,^{1,2,5} although upper respiratory tract infection does not.⁵ Whether or not predisposition to high-altitude illness increases with the severity of infection or is dependent on the type of infection, remain to be determined.

The present case is interesting because of the relatively low altitude at which high-altitude illness developed and the rapidity with which the symptoms occurred. Slight symptoms occurred immediately after reaching 3170 m and progressed to pulmonary oedema after only 4 hours above 3000 m. Usually, there is a constant delay between arrival at high altitude and the onset of symptoms of high-altitude illness.¹ In our patient, the occurrence of high-altitude illness at a relatively low altitude and the quick development of pulmonary oedema were assumed to be due to inflammatory mediators of the tooth root infection, the effect of the root canal therapy, or the incipient gingival abscess. An unrecognised pre-existing illness as a possible explanation for the altitude illness is rather unlikely, since the patient completely recovered after the gingival incisions and clindamycin therapy and follow-up clinical and laboratory investigations were all normal. A further argument for the infection as the most probable cause of the high-altitude illness is the fact that he had not experienced any similar problems during previous trips to >3000 m. Disregarding the cause of high-altitude illness, the treatment of choice remains the immediate descent to lower altitudes.⁶

In conclusion, tooth root infection and root canal therapy may predispose to high-altitude oedema. Such patients should not climb mountains >3000 m before full recovery.

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Gynaecomastia as a presenting feature of thyrotoxicosis

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Summary

The association between gynaecomastia and thyrotoxicosis is well recognised. However, the reported frequency of the association is variable, partly depending upon the defining criteria used. Here we report two patients with thyrotoxicosis in whom gynaecomastia was the presenting feature. Both patients had other contributing factors, which are assumed to have predisposed to gynaecomastia. In both patients, the gynaecomastia resolved with successful treatment of the thyrotoxicosis. When gynaecomastia is a presenting, or prominent feature of thyrotoxicosis, the possibility of additional underlying pathology should be considered.

Keywords: gynaecomastia; thyrotoxicosis; sex hormone

Gynaecomastia has long been known to be associated with thyrotoxicosis but does not usually occur as a presenting feature of the condition. Its reported frequency was as high as 40% in two early series.^{1,2} However, recent reviews suggest a much lower frequency than previously thought.^{3,4} Differences in reported frequency depend in part upon the criteria used for defining the presence of gynaecomastia.

Here we report two patients with thyrotoxicosis in whom gynaecomastia was the initial presenting feature. Subsequent investigations showed that there were other co-existing factors in both patients to explain the development of gynaecomastia.

Case reports

Case 1

A 30-year-old Chinese man presented to his general practitioner because of left-sided gynaecomastia with galactorrhoea, associated with decreased libido and sexual potency for 6 months. He was referred for further investigation. His wife was pregnant at 8 weeks gestation. There was no history of drug intake. There were no symptoms of thyrotoxicosis. Physical examination confirmed left-sided gynaecomastia and galactorrhoea. Secondary sexual characteristics, testis, fundi and visual fields were all normal. A small soft goitre was palpable. Initial investigations showed that he was biochemically thyrotoxic with serum sensitive thyroid stimulating hormone (sTSH) < 0.03 mIU/l (normal range: 0.3–4 mIU/l), and free thyroxine (T_4) 22.9 pmol/l (10.2–19.6).

Anti thyroglobulin and anti thyroid microsomal antibodies were negative. The prolactin concentration was elevated at 1320 mIU/l (<480). Follicle-stimulating hormone (FSH) was 2.4 IU/l (<12), luteinising hormone (LH) 3.5 IU/l (<15), and testosterone 5.9 nmol/l (12.0–34.0), indicating hypogonadotrophic hypogonadism. Oestradiol concentration was 112 pmol/l (<361). Sex hormone binding globulin (SHBG) was 15 nmol/l (10–62).

Dynamic pituitary function tests revealed an adequate cortisol and growth hormone (GH) response to glucagon stimulation with peak cortisol and GH concentrations of 680 nmol/l and 20.0 mIU/l, respectively. A LH-releasing hormone (LHRH) stimulation test showed an impaired FSH response (baseline concentration 2.3 IU/l and peak concentration 4.0 IU/l). Thyrotropin-releasing hormone (TRH) test was also performed and sTSH was persistently less than 0.03 mIU/l, consistent with thyrotoxicosis. Magnetic resonance imaging of the hypothalamic–pituitary region was normal.

He was treated with carbimazole using a dose-titration regime. Free T_4 returned to normal after 2 months treatment and has remained in the normal range. The gynaecomastia showed gradual improvement after 3 months' treatment with carbimazole. However, the galactorrhoea persisted despite carbimazole, and the prolactin concentration remained in the range 1400–1800 mIU/l. Testosterone also remained low, range 2.3–8.7 nmol/l. His sexual drive and impotence showed no improvement. Bromocriptine was therefore added to control the hyperprolactinaemia, whereupon the prolactin concentration fell to 80 mIU/l and testosterone increased to 13.8 nmol/l. His galactorrhoea resolved with restoration of normal sexual function.

Case 2

A 35-year-old Caucasian man presented with right-sided gynaecomastia for 3 months. He gave a history of lymphocytic lymphoma at the age of 16 years, which was treated by radiotherapy and surgery in Australia. The radiotherapy field included the groin region. Chemotherapy was not administered. Since that time, there had been no recurrence of the lymphoma. However, he had had bilateral hydroceles as a consequence of radiotherapy. Sexual drive and potency were normal. Physical examination showed the right testis to be diminished in size and there was marked lymphoedema of the left leg. Significant gynae-

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