**SELF ASSESSMENT ANSWERS**

**Q1: What is the diagnosis and differential diagnosis?**

The radiograph shows a lucent area with osteolysis of the humeral head associated with expansion of bone and thinning of cortex, soft tissue swelling, and soft tissue calcification. The differential diagnoses are hydatid disease, tuberculosis, actinomycosis, fungal osteomyelitis, chronic bacterial osteomyelitis, chondrosarcoma, malignant fibrous histiocytoma, myeloma, metastatic disease, giant cell tumour, and aneurysmal bone cyst. The diagnosis of bone hydatidosis is difficult and easily overlooked unless there is a strong element of suspicion. However, presence of lucent areas, thinning of cortices, expansion of bone, and associated with soft tissue calcification is highly suggestive of hydatid disease or echinococcosis.

**Q2: What are the other sites of involvement?**

Hydatid cysts may develop in almost any part of the body. The liver is the most commonly affected organ, followed by the lung, and the remainder of the body. Bone involvement is rare, possibly because of mechanical resistance, and accounts for only 0.5%–2.5% of human hydatidosis. Skeletal sites are vertebral (30%–40%), long bones, especially lower limbs, pelvis, ribs, scapula, and rarely in calvarium and phalanges. The cysts may remain dormant in the bone for as long as 10–20 years.

**Q3: What is the treatment?**

Surgical treatment combined with chemotherapy (albendazole or mebendazole) is recommended. Chemotherapy should be given for periods of up to two years after surgery. Echinococcus larvae behave in a different manner in bones compared with soft tissues with no fibrous membrane around the osseous cysts. The behaviour of osseous hydatid cysts resembles that of locally malignant lesions. Curettage removes only macroscopic cysts and the local recurrence rate is 70%–80%. The only promising strategy for bone manifestations of hydatid disease is the combination of radical surgery and antihelminthic drugs over a period of up to two years.

**References**


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**IMAGES IN MEDICINE**

**Transient scrotal hair growth in infancy**

A male infant presented at 15 weeks of age having developed a number of dark coarse scrotal hairs during the preceding week (fig 1). There were no other signs of androgenisation and testes were of normal infant size. Investigations showed a testosterone concentration within the normal range at 3.2 nmol/l (age related reference range 0.03–6.14 nmol/l), and a normal urinary androgen profile. Serum cortisol, 17 hydroxyprogesterone, FSH, LH, thyroxine, and TSH levels were unremarkable. Subsequent linear growth was normal and by 7 months of age, the scrotal hair had disappeared.

Transient isolated scrotal hair development starting at a few months of age has been reported in a total of 10 infants from various centres in the USA. Our knowledge this has not been reported from the UK. In seven cases spontaneous disappearance of scrotal hair occurred at between 8–15 months of age. In two cases it was “diminished” by 13 months and 21 months of age, and in the remaining case time of resolution was not stated. All were thoroughly investigated and no underlying pathological cause was found.

The appearance of hair in the pubic area in a male infant usually suggests a true excess of circulating androgens, for example in congenital adrenal hyperplasia. However, the development of isolated scrotal hair alone, without other signs of androgenisation, may be a benign phenomenon explained by an increased sensitivity of scrotal hair follicles in affected infants to the normal “physiological” high concentrations of testosterone seen in early infancy. As concentrations decrease later in infancy the scrotal hair disappears or diminishes. Although this seems to be a benign condition, it should be regarded only as a diagnosis of exclusion—all infants with signs of androgenisation need urgent full investigation.

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**REFERENCES**
