Secondary oxalosis and sperm granuloma of the epididymis

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Abstract

A 30 year old man with a 20 year history of chronic renal failure who presented with a testicular lesion is described. The lesional pathology, secondary oxalosis, and associated sperm granuloma of the epididymis was clinically considered to be an intrascrotal tumour. The oxalate crystal deposition was present within the rete testis, the ductuli efferentes, and the epididymis along with sperm granulomata. This seems to be a rare complication of secondary oxalosis associated

with chronic renal failure and having both clinical and pathological implications.

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Case report

A 30 year old man with a 20 year history of chronic renal failure (due to membranoproliferative glomerulonephritis) presented with intermittent haemospermia. A firm lump in the posterior region of the left testis was palpable and, because an ultrasound scan showed an ill defined area of altered echogenicity in the left testis suggestive of tumour, an orchidectomy was performed.

Pathology

The testis was small and measured $4 \times 2 \times 2$ cm. A small hydrocele was present and the tunica vaginalis was thickened. The head of the epididymis was enlarged and measured 2.1 cm in its maximum dimension. Most of the epididymis, particularly the body and tail, had a distended, tortuous, yellow tubular appearance. Microscopical examination showed mild dilatation of the rete testis, which was lined by normal cuboidal epithelium. Abundant, irregular, laminated, tubular, and in places fan-shaped crystalline material typical of calcium oxalate (and showing strong positivity to the silver nitrate-rubeanic acid stain) was present within the interstitium and tubules of the epididymis and within the ductuli efferentes and its adjacent connective tissue (fig 1). Smaller quantities were present within the interstitium and channels of the rete testis. The interstitial deposits were associated with fibrosis, a lymphohistiocytic reaction, and a florid giant cell reaction, and a giant cell reaction to the intratubular crystals was present. Within the epididymis, numerous dilated tubules were present, some of which were filled with spermatozoa. Several foci of extravasated interstitial spermatozoa surrounded by histiocytes forming sperm granulomata were present (fig 2). An occasional granuloma containing a mixture of spermatozoa and calcium oxalate crystals at its centre was also present. A few dilated, focally ruptured epididymal ducts were identified.

The epithelium of the ductuli efferentes showed additional features including focal clear cell change, abundant lipofuscin deposition, and eosinophilic granular change of the

Figure 1 Birefringent calcium oxalate crystals within epididymal tubules.

Figure 2 Sperm granuloma of epididymis.

cytoplasm. In places the interstitial fibrosis projected into the tubules of the ductuli efferentes testis and epididymis as broad obstructing papillary structures. Haemosiderin deposition was not a feature.

The testis showed extensive, focal tubular atrophy and fibrosis with the rest of the tubules showing mild hypospermatogenesis. The body and tail of the epididymis showed extremely dilated epididymal tubules containing proteinaceous material and occasional spermatozoa.

Discussion

Chronic renal failure may produce hyperoxalaemia and secondary oxalosis due to decreased oxalate excretion with the tissue deposition (oxalosis) typically occurring within the kidney, although other organs may also be involved.¹⁻³ Furthermore, deposition of calcium oxalate crystals within the rete testis and ductuli efferentes of patients with chronic renal failure has been described under the descriptive notation of acquired cystic transformation of the rete testis.⁴ Although showing cystic dilatation of the rete testis, this case differs in several respects

including the clinical presentation with a palpable, intrascrotal lesion, haemospermia (which was cured by surgery), and a small associated hydrocele. Also, the histological involvement was more extensive producing prominent epididymal oxalosis and epididymal spermatic granuloma formation. Direct damage to the epididymal tubular epithelium and basement membrane, with or without tubular obstruction and distension caused by interstitial fibrosis, are mechanisms for spermatic granuloma formation. To the best of our knowledge, the combination of calcium oxalosis and spermatic granulomas of the epididymis has not been reported before.

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Polycythaemia rubra vera transforming to acute lymphoblastic leukaemia with a common immunophenotype

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Abstract

Lymphoblastic transformation of polycythaemia rubra vera is an extremely rare phenomenon. A case of a 76 year old man with polycythaemia rubra vera who developed acute lymphoblastic leukaemia (ALL) 16 years after his initial diagnosis is reported. Membrane markers showed a CD10 positive (common ALL) immunophenotype. To our knowledge this association has not been previously recorded. The rare occurrence of ALL in polycythaemia rubra vera may indicate that in a minority of patients clonal expansion of an abnormal pluripotent haemopoietic stem cell is responsible for the polycythaemia rubra vera disease phenotype.

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The transformation of polycythaemia rubra vera to myelofibrosis or acute myeloblastic leukaemia is well recognised but the transfor-

mation of polycythaemia rubra vera to acute lymphoblastic leukaemia (ALL) is an extremely rare phenomenon. We report a case of polycythaemia rubra vera who transformed into ALL with a CD10 positive immunophenotype, to our knowledge, an association not previously recorded.

Case report

A 76 year old man presented with polycythaemia rubra vera in 1974. His haemoglobin concentration was 230 g/l, white cell count $11.2 \times 10^9/l$, and platelet count $260 \times 10^9/l$. Splenomegaly (3 cm) was present and his red cell mass was raised at 60.6 ml/kg. He was initially treated with venesection and aspirin. Intermittent low dose busulphan treatment was started in 1979 for increasing splenomegaly. Treatment with intermittent venesection and low dose busulphan was continued until the development of deteriorating pancytopenia (haemoglobin concentration 72 g/l, white cell count $2.5 \times 10^9/l$, platelet count

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