acid excretion and creatinine clearance was low, being 290 mg/day and 37 mL/min respectively. Additional abnormal investigations included a haematocrit of 27.3%, urea of 30 mg/dl, ESR of 78 mm/h. Serum triglyceride and total lipid were elevated being 291 mg/dl and 1281 mg/dl respectively. On USG bilateral soft tissue swelling in the axillar region were found to be compatible with lipomatosis. Urolithiasis was not observed. With these findings and family history she was suspected to have hereditary familial hyperuricemic nephropathy.

After resolution of the acute attack with indomethacin, allopurinol treatment was started but had to be discontinued upon occurrence of widespread erythematous rash. She was hospitalised and desensitisation regime lasting 10 days was commenced. After desensitisation, she was free of symptoms with 300 mg/day allopurinol and 0.5 mg/day colchicine. Four months later, she developed oral ulcers. Since there was a doubtful genital ulcer history she was examined for Behçet's disease. Pathergy test was negative and unexpectedly she was HLA B27 positive but no other signs suggesting spondyloarthritis was present. Oral ulcers disappeared after allopurinol dosage has been lowered to 150 mg/day, the rash or ulcers has not recurred. The tophi were dissolved, renal functions and blood pressure have not deteriorated over time, there has been dramatic improvement of her symptoms with normalisation of serum urate levels, she is now Steinbrocker class II.

Results

Conclusion Hyperuricemia or gout appearing at an early age in a female is usually associated with progressive renal failure. Early recognition of this disorder is important since allopurinol therapy may ameliorate the progression of the renal lesion. In cases of hypersensitivity to allopurinol oral desensitisation is a feasible approach.

AB0156 THE EFFICACY OF ALENDRONATE IN POSTMENOPAUSAL OSTEOPOROSIS

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Background This is a prospective study of bone mineral density in postmenopausal women treated with alendronate.

Objectives To evaluate the efficacy of alendronate in postmenopausal osteoporotic women for 1, 2 and 3 years.

Methods In a prospective study, we included 98 women diagnosed of postmenopausal osteoporosis attended from 1997 to 2000. The mean age was 59.07 (SD: 6.68). Patients received alendronate 10 mg daily, they were allowed to take calcium and vitamin D supplementation. The subjects were assessed at baseline, 12, 24 and 36 months for bone mineral density (BMD) measurements and X-ray and every 6 months blood and urinary biochemistries and markers of bone turnover were done.

Results Sixteen patients (16%) have dropped out because of adverse effects, the most common was gastrointestinal events. 34 (34.6%) patients had previous fractures (17 had spine fractures, 12 Colles's fractures and one hip fracture). Three patients on treatment with alendronate had new fractures (1 spine, 1 scaphoid and one Colles's fracture). 70, 24 and 7 subjects have completed one, two and three years of treatment, respectively. The results were analysed by Student's t-test. Patients treated with alendronate for 1 year had significantly greater improvement in BMD at spine. The mean BMD at baseline was 0.373 vs

0.773 ($p < 0.005$) and at hip 0.665 vs 0.643 at baseline ($p < 0.005$). Patients who completed two years had similar values of BMD as the year before, the mean BMD was 0.745 at spine and 0.646 at hip.

Conclusion Patients treated with alendronate for one year had significantly greater improvement in BMD at both the spine and hip.

AB0157 PARANEOPLASIC HYPERCALCEMIA COMPLICATING A B CELL NON-HODGKIN LYMPHOMA (NHL)

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Background Hypercalcemia is an unfrequent manifestation of B cell NHL. We describe a case report of hypercalcemia secondary to PTH-rp production, inaugurating a B-cell NHL.

Objectives A 77 year old woman was admitted with dorsalgia. Physical examination was negative except neuropsychological hyperexcitability.

Biological data revealed an increased calcemia (4.2 mmol/l) with decreased phosphoremia (0.45 mmol/l) suggesting a hyperparathyroidism. However, PTH(RIA) was low (3.5 pg/ml, N = 11.0–62.0) and 1.25(OH)2D3 level was in the normal range (40 yg/l, N = 15–80). PTH-rp was moderately increased (2.1 ymol/l, N < 1.5). Bone marrow aspiration was normal. No sign for osteolysis was present on bone X-ray, CT scan and MRI. The abdominal CT scan and abdominal MRI revealed a retroperitoneal mass. A needle biopsy was diagnostic for NHL B type. LDH were elevated to 3290U/l (n < 470). Other lymphoid tissue involvement was limited to the spleen. IV farnidronate was successfully administered for treatment of hypercalcemia.

Methods

Results Malignant hypercalcemia occurs in 14p.cent of leukaemia and lymphoma. Among NHL, T cell lymphoma positive for HTLV-1, are more frequently associated with hypercalcemia mediated by production of 1–25(OH)2D3 or lymphotoxin/OAF. Less than 3p.cent of B cell NHL are associated with a paraneoplastic hypercalcemia. Two hypercalcemic mediators have been characterised: PTH-rp and/or OAF

Conclusion Humoral hypercalcemia mediated by PTH-rp is a rare complication of B cell-NHL. Hypophosphatemia with a decreased PTH plasma level must alert for this mechanism.

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OP0011 CONSTRUCTION OF A PHYSICAL MAP AND IDENTIFICATION OF CANDIDATE GENES FOR FAMILIAL CHONDROCALCINOSIS AT CHROMOSOME 5P15

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Background A gene for familial chondrocalcinosis was previously mapped to a locus (CCAL2) on chromosome 5p.¹ The purpose of the study was to identify this gene and investigate its role in