Images in Nephrology (Section Editor: G. H. Neild)



Back pain in chronic renal failure

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Keywords: back pain; chronic renal failure; fluorosis

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Patient SK, a 40-yr-old female, resident of Bhagalpur village in Bihar, India, was operated for gallstones 3 years previously. On pre-operative checkup, mild renal dysfunction was detected. She was asymptomatic for renal disease with serum creatinine of 159 µmol/l (1.8 mg/dl), bland urinary sediment and small echogenic kidneys on ultrasound. She was on conservative management for chronic renal failure (CRF) and was doing well till 1 year ago. Since then she developed back pain, which increased on walking. Pain was dull in character without any radiation. There were no systemic complaints. In the past month when her pain increased substantially, causing discomfort in day-to-day activities. On investigation she was found to have moderate renal failure with blood urea nitrogen (BUN) of 11.8 mmol/l and serum creatinine of $309 \,\mu\text{mol/l}$ (3.5 mg/dl). Other labs showed serum calcium of 2.4 mmol/l, phosphorus 1.1 mmol/l, alkaline phosphatase 243 U/l, uric acid 381 µmol/l and intact parathyroid hormone (iPTH) of 182.0 pg/ml. X-rays of bilateral forearms, pelvis and spine were taken (Figures 1–3, respectively), which revealed generalized increase in bone density, degenerative changes with osteophytes in lumber vertebra, calcification of bilateral iliolumbar and sacrospinous ligaments and interosseous membrane calcification in forearms. Definitive diagnosis was reached with estimation of fluoride levels in blood and urine, which were 0.291 mg/l and 0.962 mg/l (15.3 and $50.6 \mu \text{mol/l}$), respectively. Her drinking water source, ground water from a tubewell, was found to contain 3.910 mg/l (205.9 µmol/l) of fluoride. She was diagnosed to have fluorosis with moderate CRF and was advised to use

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Fig. 1. Plain film of both forearms showing interosseous membrane calcification.

domestic reverse-osmosis-treated water for cooking and drinking.

Main sources of fluoride include food and water. About 50–70% of fluoride is excreted by the kidneys [1]. Individuals with kidney disease have decreased ability to excrete fluoride in urine and are at risk of developing fluorosis even at normal recommended limit of 0.7 to 1.2 mg/l (37–63 μ mol/l) of fluoride in drinking water [2]. In fluorosis with normal renal function, urine fluoride rises above 0.5-4.48 mg/l $(26-236 \, \mu mol/l)$ and may reach $1.5-13.0 \, mg/l$ [3]. Serum fluoride rises to $(79-685 \, \mu mol/l)$ $0.04-0.28 \,\text{mg/l}$ (2.1-14.7 µmol/l) in such patients [1]. Fluoride is bone-seeking due to its high affinity for calcium phosphate and therefore accumulates in bone. Radiological changes can be quite similar to changes of renal osteodystrophy, and therefore the diagnosis may be missed unless specifically investigated.

Conflict of interest statement. None declared.

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Fig. 2. Plain film of pelvis showing calcification of bilateral iliolumbar (black arrow) and sacrospinous ligaments (white arrow).



Fig. 3. Plain film lateral view of lumbar spine showing increase in bone density with osteophytes.

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Received for publication: 6.3.06 Accepted in revised form: 10.4.06