Effects of Bed Rest on Bone Metabolism in Patients with Femoral Neck Fracture

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The effect of continuous 1 week bed rest during traction on bone metabolism were studied in patients with femoral neck fracture. Bone demineralization was assessed by measuring serum calcium (Ca), phosphorus (P), and alkaline phosphatase (AlkP) before, during, and after 1 week bed rest with skeletal traction. In addition, 24-hour urinary excretion of calcium and phosphorus were measured. Serum Ca did not change significantly before, during, and after bed rest, whereas serum P increased after 1 week bed rest and postoperatively remained significantly elevated. Serum AlkP did not increase with bed rest, but became elevated postoperatively. Urinary Ca excretion increased from 105.8±15.2 to 138.0±15.4 mg/24h after 1 week of bed rest, and remained elevated postoperatively. In contrast, urinary P excretion initially increased 495.1±65.0 to 610.8±87.7 mg/24h, and subsequently decreased to a level significantly less than the admission level. These results suggest that 1 week bed rest for the patients with femoral neck fracture resulted in bone atrophy and demineralization.


Key words: Bone metabolism, Bed rest, Femoral neck fracture, Fluid shift, Demineralization

INTRODUCTION

Bed rest during preoperative skeletal traction for leg fracture provides pain relief and is therefore frequently utilized. However, maintenance of bone mineralization depends on the longitudinal mechanical forces applied to the bone in a normal gravity environment. Therefore, long-term bed rest induces disuse bone atrophy and demineralization. Previous studies of healthy young males have shown that that long-term bed rest produces increased urinary Ca (hypercalcuria), negative calcium balance, and bone demineralization (Griffis, 1971; Whedon 1984; Donaldson et al., 1970). In addition, prolonged bed rest induces fluid shifts, which may facilitate bone demineralization (Tavassoli, 1986; Thornton et al., 1977). There have been few studies in geriatric patients of the effects of bed rest and skeletal traction on bone metabolism. Since it is very important for aged patients with leg fractures to minimize bed rest induced osteoporosis, countermeasures to prevent demineralization are required.

The current study was designed to assess the effects of preoperative bed rest with skeletal traction on bone metabolism, and to evaluate bone demineralization in geriatric patients with femoral neck fracture. In addition, prophylaxis for bone demineralization during bed rest is discussed.

MATERIALS AND METHODS

Subjects: Twenty patients with femoral neck fracture were assessed for bone demineralization during 1 week bed rest. All the patients had normal
renal function. Serum BUN and creatinine were normal. Age range was 61 to 86 years with a mean of 73.1 years. Bed rest consisted of a continuous supine position with one pillow under the head.

Methods: Skeletal traction was performed on these patients with femoral neck fracture at a weight of 3 to 5 kg. Duration of bed rest during skeletal traction was 1 week.

Serum calcium (Ca), phosphorus (P) and alkaline phosphatase (AlkP) were measured before and during bed rest for 1 week. In addition, these values were measured postoperatively at 1 and 4 weeks. Corrected serum Ca was calculated as follows:

Corrected serum Ca (mg/dl) =

serum Ca (mg/dl) − serum albumin (g/dl) + 4.0

(Payne et al., 1973)

Twenty-four hour urine collection was performed for all patients. Urinary Ca and P excretion were measured for 1 day and 1 week at bed rest, as well as 1 and 4 week post-bed rest.

Statistical analysis. Results are expressed as the means ± standard error. Paired measurements were compared using Student's t-test.

RESULTS

The mean volume of 24-hour urine collections increased significantly (p < 0.01) from 947.5 ± 86.9 ml/24h after 1 day bed rest, to 1326.5 ± 130.1 ml/24h after 1 week of bed rest. After 1 week postoperatively, the volume decreased to 1228.0 ± 121.8 ml/24h which was significantly greater than that after 1 day bed rest (p < 0.01). Four weeks postoperatively, the volume further decreased to 1078.0 ± 83.7 ml/24h (Fig. 1).

Urinary Ca excretion increased significantly (p < 0.01) increased from 105.8 ± 15.2 mg/24h to 138.0 ± 15.4 mg/24h after 1 week bed rest (Fig. 2). With further observation, urinary Ca excretion remained elevated postoperatively.

Urinary P excretion increased significantly (p < 0.05) from 495.1 ± 65.0 mg/24h to 610.8 ± 87.7 mg/24h after 1 week bed rest (Fig. 3). However, after 1 week.
postoperatively, urinary P excretion decreased to 438.5±78.1 mg/24h. Four weeks postoperatively, urinary P excretion further decreased significantly (p<0.01) to 323.8±68.6 mg/24h, while urinary Ca excretion remained elevated.

Serum Ca concentrations did not change significantly during the period of study (Fig. 4). In contrast, serum P concentration increased significantly (p<0.05) after 1 week of bed rest. Furthermore, serum P concentrations postoperatively maintained higher values than that before bed rest (Fig. 5).

AlkP concentrations did not change significantly after bed rest for 1 week. However, the levels after 1 and 4 weeks postoperatively significantly increased to 174.9±14.1 and 188.1±12.7 IU/l, respectively (Fig. 6).

**DISCUSSION**

The major findings in this study were as follows: 1) The volume of 24-hour urine collections increased significantly after 1 week bed rest; 2) Urinary Ca excretion increased significantly after 1 week of bed rest and remained elevated postoperatively, whereas urinary P excretion decreased postoperatively after an initial increase at 1 week of bed rest; and 3) Serum P increased significantly after 1 week bed rest and subsequently remained elevated, AlkP increased postoperatively, while the serum Ca levels did not change.

The increase in volume of 24-hour urine collections may be due to fluid shift during bed rest (Tavassoli, 1986; Thornton et al., 1977; Pace, 1977). Fluid shift is defined as the cephalad shift of blood and tissue fluid from the lower extremities to the torso during bed rest (Pace, 1977). This shift increases the central blood volume, which stimulates cardiopulmonary vasculature stretch receptors. This leads to decreased release of antidiuretic hormone from pituitary gland, and urine excretion increases.

The mechanism of increased urinary calcium excretion after bed rest is probably accelerated...
bone demineralization during prolonged bed rest (disuse bone atrophy). Furthermore, rise in urinary Ca excretion may result from a decrease in Ca reabsorption in renal proximal tubules. Urinary calcium excretion is a function of the glomerular filtration rate, the plasma ionic calcium concentration, and the tubular reabsorption of calcium. The major portion of calcium reabsorption occurs in the proximal tubules, and this process determines the magnitude of urinary calcium excretion (Griffith, 1971). Previous studies have suggested that prolonged recumbency is associated with hyperaldosteronism and sodium retention (Griffith, 1971; Claus-Walker et al., 1969; Lutwak et al., 1969). These fluid and electrolyte shifts during bed rest may facilitate an expansion of extracellular fluid, which may induce decreased Ca reabsorption in proximal tubules, resulting in hypercalciuria.

Serum P after bed rest increased significantly than that before bed rest. Furthermore, the postoperative levels maintained higher values significantly than that before bed rest. Serum P is one of the bone resorption parameters. Bone resorption during reparative phase of femoral neck fracture may induce the persistent rise in serum P concentration.

AlkP increased significantly postoperatively than did preoperatively. AlkP exists in osteoblast and correlates with the magnitude of osteoblastic activity. Acceleration of bone formation during reparative phase of femoral neck fracture may cause a significant rise in AlkP concentration.

Our results indicate that prophylactic measures are necessary to prevent bone demineralization in elderly patients with fracture during bed rest. Administration of oral phosphate supplements may play a role in the prevention of bed rest-induced demineralization disorders. Hormonal therapy such as synthetic salmon calcitonin intramuscularly may prevent demineralization, since calcitonin is known to inhibit bone resorption. Schneider et al. (1984) have shown that supplemental calcium and/or phosphorus, etidronate, and calcitonin were not effective in preventing bed rest induced hypercalciuria. In contrast, Hulley et al. (1971) showed that potassium phosphate supplements reduced urinary calcium excretion, but this treatment did not prevent bone demineralization of the calcaneus during bed rest.

Electrical low-frequency stimulation may improve disuse atrophy of both muscle and bone. Issekutz et al. (1966) have suggested that bed rest induced bone demineralization is due to the absence of longitudinal pressure (weight bearing) on the bones rather than on physical activity. Therefore, muscle contraction induced by electrical low-frequency stimulation may prevent bed rest-induced bone atrophy.

In conclusion, the elderly patients with leg fracture require prophylactic measures against bed rest induced bone atrophy and demineralization.

REFERENCES
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