Rapidly destructive coxarthropathy, characterized by severe joint destruction occurring within a few months, is ascribed to subchondral insufficiency fracture resulting from osteoporosis and ischemic hip disease. Corticosteroids can cause avascular osteonecrosis of the femoral head, osteoporosis, or both. Most cases of corticosteroid-induced osteonecrosis are caused by exogenous corticosteroid administered for the treatment of various diseases. A few cases of osteonecrosis caused by endogenous corticosteroid, such as in Cushing’s syndrome, have been reported previously.

This article presents a case of rapidly destructive coxarthropathy with osteonecrosis and osteoporosis caused by Cushing’s syndrome.

CASE REPORT

A 55-year-old woman presented with intense right hip pain and a limp. No history of trauma, excessive alcohol consumption, smoking, or corticosteroid medication was reported.

Plain radiographs revealed diffuse osteoporosis and sclerosis in the femoral head, and magnetic resonance imaging (MRI) showed a low-intensity band on T1-weighted images (Figure 1). The patient was diagnosed with Ficat stage II avascular osteonecrosis of the femoral head. The patient was morbidly obese (height, 155 cm; weight, 70 kg; body mass index, 29.1); her body weight had increased by 14 kg over 2 years, and she had a moon face. Edema in the lower extremities, easy bruising, and mild acne were also noted. An endocrinologist was consulted.

Serum examinations revealed high levels of serum and urine cortisol (serum cortisol, 21.2 µg/dL, normal range 4.0-18.3 µg/dL; urine cortisol, 164 µg/day, normal range 30-100 µg/day), a low serum adrenocorticotropic hormone level (<5 pg/mL, normal range 9-52 pg/mL) and hyperlipidemia (total cholesterol, 257 mg/dL, normal range 130-230 mg/dL). Bone mineral density, evaluated with the T-score (ie, values relative to the mean value for young adults), was reduced in the lumbar spine (-3.06 standard deviation [SD]), femoral neck (-2.47 SD), and forearm (-3.00 SD).

Based on the endocrinological examination, a diagnosis of Cushing’s syndrome from

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adrenal adenoma was made, and laparoscopic tumor resection was performed. Total hip arthroplasty was later performed due to severe joint destruction of the femoral head 4 months after the initial examination (Figure 2).

**DISCUSSION**

Few cases of osteonecrosis of the femoral head caused by endogenous corticosteroid, such as in Cushing’s syndrome, have been reported; however, corticosteroid has been reported as one of the factors causing osteonecrosis of the femoral head. Endogenous hypercortisolism caused by pituitary adenoma is defined as Cushing’s disease, and similar conditions due to other causes are defined as Cushing’s syndrome. We report a case of osteonecrosis of the femoral head due to Cushing’s syndrome caused by adrenal adenoma. Only 15 cases of endogenous corticosteroid-induced osteonecrosis of the femoral head have been noted since the first report by Madell and Freeman.

Phillips et al reported that the inclusion of adrenergic substances in exogenous corticosteroid to enhance the effect of corticosteroid could be a reason for the large number of reports of femoral head osteonecrosis caused by exogenous hypercortisolism. They also reported that the durations from diagnosis of Cushing’s disease to onset of osteonecrosis in four patients varied from 8 months to 11 years.

This large range between diagnosis of Cushing’s disease and osteonecrosis is believed to be due to endogenous corticosteroid increasing in a relatively short period in some cases and acting gradually in other cases. Because it is difficult to convert serum cortisol concentration into an amount of exogenously administered corticosteroid, it is not possible to determine the correlation between duration of Cushing’s disease and onset of femoral head osteonecrosis.

On the other hand, Cerletty et al reported the possibility that some patients with idiopathic osteonecrosis of the femoral head have endogenous hypercortisolism although its existence has not been recognized. This suggests that the incidence of femoral head osteonecrosis caused by endogenous corticosteroid may be higher than reported. In the present case, no subjective symptoms were noted before osteonecrosis occurred other than rapid increase in body weight. The lack of symptoms associated with idiopathic femoral head osteonecrosis may result from asymptomatic Cushing’s syndrome. Thus, care must be taken when a diagnosis of “idiopathic” femoral head osteonecrosis is made.

Moreover, rapidly destructive coxarthropathy occurred 4 months after the initial examination. The pathophysiology is not clearly understood. However, Yamamoto and Bullough reported that a subchondral insufficiency fracture resulting from osteoporosis may contribute to the condition. Laroche et al described that some cases of rapidly destructive coxarthropathy may be attributed to ischemic hip disease.

**REFERENCES**


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**Most cases of corticosteroid-induced osteonecrosis are caused by exogenous corticosteroid administered for the treatment of various diseases.**


