Risedronate reduces BMD loss after hip replacement

Periprosthetic bone resorption is a complication of cementless total hip arthroplasty (THA). Part of the stress that would normally be experienced by the femur is borne by the implant, leading to bone remodeling, and this resorption can lead to loosening of the implant. Bisphosphonate therapy has been shown to protect against bone resorption after THA. As no data so far exist on the effect of risedronate in this setting, Yamasaki *et al.* conducted a randomized, placebo-controlled study of the effect of risedronate on periprosthetic bone resorption after cementless THA.

Participants were randomly allocated to placebo ($n = 21$) or risedronate (2.5 mg/day; $n = 22$) for 6 months after undergoing cementless THA. Three patients in the risedronate group withdrew because of dyspepsia. BMD in the proximal femur (which was divided into seven zones) was measured 3 weeks and 6 months postoperatively by dual-energy X-ray absorptiometry. There was no significant difference in BMD between the groups at 3 weeks; these values were used as baseline figures. At 6 months, BMD decrease was significantly less in the risedronate group than in the placebo group in the five most proximal zones; BMD ranged from 79.8% to 91.1% of baseline in the placebo group, and from 88.1% to 100.9% of baseline in the risedronate group. There was no significant difference in BMD loss in the two most distal zones.

The authors conclude that risedronate helps to protect against periprosthetic bone resorption after cementless THA.

Original article Yamasaki S *et al.* (2007) Risedronate reduces postoperative bone resorption after cementless total hip arthroplasty. *Osteoporos Int* **18:** 1009–1015

Positive association of Addison's disease with celiac disease

Addison's disease (primary adrenal insufficiency) is commonly caused by an autoimmune reaction. Several studies have suggested an increased risk of celiac disease in patients with Addison's disease. These studies were, however, limited in size and the risk of Addison's disease in celiac disease had not been estimated.

A study by Elfström *et al*. examined 14,366 individuals with celiac disease (identified through the national inpatient register of the Swedish National Board of Health) as well as a control group of 70,095 individuals without celiac disease. None of the patients had Addison's disease at, or 1 year after, study entry. Subjects with celiac disease were at an 11-fold increased risk of subsequent Addison's disease. The incidence rate for Addison's disease was 15 per 191,780 person-years in individuals with celiac disease and nine per 971,639 person-years in reference individuals. Subjects who developed Addison's disease were at an eight-fold increased risk of subsequent celiac disease. The positive association between the two disorders was seen both before and after the diagnosis of celiac disease.

The authors conclude that the risk of developing Addison's disease is highly increased in patients with celiac disease and suggest that these findings might be explained by the presence of shared genetic traits. They recommend screening of individuals with Addison's disease for celiac disease, as well as being increasingly aware of Addison's disease in subjects with celiac disease.

Original article Elfström P *et al.* (2007) Risk of primary adrenal insufficiency in patients with celiac disease. *J Clin Endocrinol Metab* **92:** 3595–3598

Patients on sunitinib should be monitored for hypothyroidism

A recent study by Mannavola *et al.* evaluated the effect of the oral tyrosine kinase inhibitor sunitinib on thyroid function in patients treated for a gastrointestinal stromal tumor (GIST). The study was initiated following the death of a patient, in whom a myxedematous coma was observed while being treated with sunitinib for a GIST.

Twenty-four patients (age range 40–75 years) treated with sunitinib were prospectively evaluated in this study. They were treated according to a 6-week cycle scheme, during which the drug was administered daily for 4 weeks ('ON period'), followed by 2 weeks of withdrawal ('OFF period'). Only one patient had a past history of thyroid disease.

Ten patients developed hypothyroidism. In these patients, TSH levels were initially raised