Why does post-transplant osteonecrosis develop?

Sir,

We read with interest the article by Ekmekci et al. [1], on the association of thrombophilia and osteonecrosis (ON) of the femoral head in renal transplant recipients. We previously reported a case with diffuse ON and severe osteoporosis which was unusual in its presentation in the early post-transplant period, focusing on pre-transplant hormonal changes [2]. Following our report, Dr Weinstein drew our attention to their research supporting glucocorticoid (GC)-induced ON involved in osteocyte apoptosis, by personal communication. They demonstrated that osteoblasts and osteocytes were the direct targets of GC action in vivo, and that excess levels of steroid hormone directly induced apoptosis of these cell types.

Ekmekci et al. [1] reported that factor V Leiden and prothrombin gene mutations might be an important risk factor for the development of ON of the femoral head. They observed no difference in the cumulative doses of GCs and ciclosporin A between the ON and control groups, whereas Celik et al. [3] found that the 3, 6 and 12 month treatments with cumulative GC doses were significantly higher in the ON group and that there was no correlation between ON and genetic mutations of factor V Leiden, prothrombin and 5,10-methylenetetrahydrofolate reductase (MTHFR). Our case had MTHFR C677T heterozygous mutation but not factor V Leiden G1691A and prothrombin G20210A mutations. In the non-transplant population, there are different reports on the relation of genetic mutations predisposing to coagulation and ON [3,4].

A recent hypothesis emphasizes the fact that vascular thrombosis is the major pathogenic event leading to osteocyte necrosis and eventual collapse of the femoral head in ON [5]. Exogenous and endogenous factors lead to endothelial dysfunction, thrombus formation and ischaemia, finally inducing apoptosis in osteocytes and osteoblasts. Thrombophilia, particularly impaired fibrinolysis, can play a potential role in thrombus formation. Decreased fibrinolytic activity through decreased plasminogen activator inhibitor-1 gene, hypofibrinolysis, and osteonecrosis.

The state of bones in the pre-transplant period, the effect of uraemic milieu, post-transplant medications and other cofactors on haemostatic alterations, and individual genetic differences can determine the outcome. More evidence is needed to better comprehend the role of these parameters for future prevention and treatment of ON.

Conflict of interest statement. None declared.

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Increasing incidence of severe encapsulating peritoneal sclerosis after kidney transplantation

Sir,

Encapsulating peritoneal sclerosis (EPS) is a life-threatening complication of peritoneal dialysis (PD). Since the first report in 1980 [1], the reported overall prevalence of EPS has

Advance Access publication 18 April 2007

Advance Access publication 29 March 2007