OBSERVATIONS

Immunosuppression Therapy Posttransplantation Can Be Associated With a Different Clinical Phenotype for Diabetic Charcot Foot Neuroarthropathy

Proinflammatory changes in the immune phenotype of circulating blood monocytes in acute Charcot in diabetes have recently been described (1), but the effects of immunosuppression have not been investigated. This report describes a modified clinical phenotype for Charcot in three subjects on immunosupression therapy for previous transplantation—two live-related renal and one simultaneous pancreas kidney transplant. Charcot presented without typical features such that the early acute phase was difficult to define clinically. In the two in whom immobilization was not initially undertaken, deformity developed. All had previous proliferative retinopathy, neuropathy, and nephropathy resulting in end-stage renal failure.

A 42-year-old woman with type 1 diabetes, duration 34 years, presented with 3 months of intermittent right foot and ankle swelling. Immunosuppression therapy was tacrolimus, mycophenylate, and prednisolone. Plain X-ray was normal and magnetic resonance imaging (MRI) demonstrated midfoot bone marrow edema. On repeated clinical examination over 6 months, there was no swelling, erythema, or increased temperature in the right foot, and plain X-rays remained normal. Immobilization was not undertaken. A further 6 months later, she still reported intermittent symptoms, but increased temperature was detected, and MRI demonstrated more bone marrow edema and malalignment in the midfoot.

A 53-year-old man with known type 2 diabetes for 8 years presented with 8 months of intermittent left foot pain and swelling after walking for an hour. Immunosuppression therapy was tacrolimus. Clinically, there was no increased temperature or foot swelling, plain X-ray demonstrated normal alignment, and MRI demonstrated midfoot bone marrow edema. Immobilization was not applied. On repeated clinical examination over 5 months, there were no clinical signs, and plain X-rays were unchanged. At 6 months, the left foot was slightly warmer with mild swelling over the dorsum. An MRI demonstrated more extensive midfoot bone marrow edema and collapse of the intermediate and medial cuneiform bones.

A 48-year-old woman with type 1 diabetes, duration 41 years, presented with 3 days of right foot discomfort and swelling. Immunosuppression therapy was tacrolimus, mycophenylate, and prednisolone. On examination there were no clinical signs. Plain X-ray was unremarkable, and MRI demonstrated mild midfoot bone marrow edema. She was reviewed five times over the ensuing 2 months without signs to suggest active Charcot. Immobilization was not applied until further MRI of both feet demonstrated more extensive bone marrow edema in the right than the left midfoot.

In order to avoid deformity, immobilization must be applied early in acute Charcot while plain X-ray is still normal. MRI is often used at this stage to confirm suspected clinical diagnoses. However, MRI demonstrated bone marrow edema in midfoot and hindfoot areas in 30% of subjects with diabetic neuropathic ulceration, did not predict future Charcot or osteomyelitis, and was more common in end-stage renal disease (2). Although all three had midfoot bone marrow edema on MRI, clinical signs were lacking so that immobilization was not initially applied. Clinicians should be aware that Charcot can present posttransplantation without the cardinal clinical signs but can still lead to deformity.

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