Sudeck's Atrophy of the Left Tibiotarsal Joint in a Renal Transplant Patient: Effects of Medical and Physical Therapy

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Dear Sir,

In view of the diversity of osteoarticular entities affecting the transplanted patient, special attention must be paid to Sudeck's atrophy [1-3]. To our knowledge, no such cases have been reported up to now. Sudeck's atrophy manifests as pain restricted to a bone segment, palpable edema, vasomotor skin instability, and focal bone demineralization.

A 49-year-old woman with a cadaver donor renal transplant since June 1982 complained of acute pain in the left ankle with hypothermia of the same leg in March 1992. The graft function had always been normal, and treatment included azathioprine 100 mg and prednisone 10 mg daily. Both X-ray and Doppler sonography of the left tibiotarsal joint were negative. After 15 days during which pain continued, the ankle swelled, and walking became difficult (fig. 1a). A repeat X-ray revealed traces of mottling and an irregularity in frontal cortex of the left astragalus, with swelling of the soft periarticular tissues.

Bone technetium scintigraphy showed accumulation of the marker substance at left tibiotarsal joint and homolateral knee, while the iliofemoral-popliteal axis appeared normal. Nuclear magnetic resonance imaging and computerized tomography of the tibiotarsal joint excluded any focal bone lesions of the segment examined, only visualizing the

Fig. 1. Ankle swelling (a) regressed after 5 months of therapy (b).

swollen soft periarticular tissues. Creatinine was 1.2 mg%, calcium 9.9, phosphorus 3.7, and uric acid 10.8 mg%. All tests for inflammatory reactions were negative. Treatment was started with nonsteroidal anti-inflammatory agents, calcitonin, and vitamin D, with no immediate clinical benefit. Another X-ray 1 month later showed pronounced patches of decalcification on distal

epiphysis of the tibia, heel, astragalus, and tarsal bones (fig. 2b). The diagnosis of Sudeck's atrophy was made.

Physiotherapy was started with calcium iontophoresis, hydrogalvanotherapy, and therapy combined with calcitonin, vitamin D, and anti-inflammatory drugs. The immunosuppressive therapy remained unchanged.

After 5 months the pain had completely receded and the clinical picture completely normalized with restoration of the joint function (fig. 1b). An X-ray at that time revealed a normal bone structure.

In February 1994 a recurrence of the disease took place in the same bone segment. The X-ray findings suggested a quick treatment according to the previous protocol. Within 1 month complete recovery was achieved.

Sudeck's atrophy is rare, usually occurring at around 50 years of age in males and females [1]. It generally affects the distal extremities and particularly the feet [2]. In 50% of the cases the contralateral joint is also affected [1]. Its pathogenesis remains still unknown, even if an altered reflex response by the sympathetic nervous system and somatic pain fibers after a trauma or some other unidentified stimulus seem to play a role [2].

After an acute phase with swelling, intense pain, and X-ray evidence of hyper-transparency with small mottled areas, giving the affected bone segment a 'leopard skin' appearance (fig. 2b), the chronic, atrophic phase is characterized by pale, thin, and dry skin, hypotrophy, and muscular retraction with stiffness in the joints. X-rays show high-degree osteoporosis with thin 'pencil line' epiphyseal cortices and practically nonexistent spongy tissue ('vitreous atrophy'). In other cases, the normal trabeculae are replaced by wide-spaced coarse bone trusses following the lines of force ('hypertrophic atrophy'). This patient did not reach the chronic atrophic stage, and the physical therapy allowed recovery of the normal joint function.

Sudeck's atrophy and a possible recurrence must, therefore, be carefully considered in view of the spectrum of posttransplant osteoarticular complications.

References

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