Algoneurodystrophy following herpes zoster

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Summary: Algoneurodystrophy frequently follows an identifiable triggering event. It is not widely recognized that herpes zoster can precipitate algoneurodystrophy and three such cases are described here. In one, the affected dermatome did not correspond to the limb involved by the algoneurodystrophy.

Introduction

Algoneurodystrophy (reflex sympathetic dystrophy syndrome) is a poorly understood phenomenon embracing several overlapping syndromes including Sudeck's atrophy and 'shoulder hand syndrome'.^{1,2} In the latter, the patient initially presents with a red, painful, oedematous hand and forearm with a stiff painful shoulder. Gradually, pain diminishes and oedema may subside. The skin becomes cold, pale and shiny and the muscles atrophy. Radiographs show increasing osteoporosis and, if untreated, the patient is eventually left with a cyanotic, functionally useless hand with severe osteoporosis.

Algoneurodystrophy can occur at any age and there is an equal sex incidence.³ Between 15 and 50% of cases are idiopathic; the remainder are precipitated by events such as trauma, neurological damage (especially root or peripheral nerve lesions) and ischaemic heart disease. Although herpes zoster was identified by Sudeck⁴ as a precipitant of algoneurodystrophy and this is mentioned in a recent European monograph,⁵ it is seldom referred to in English language publications.

The last large series of cases published 18 years ago⁶ identified precipitating factors in 85% of cases but failed to mention herpes zoster as a cause. We therefore report three cases, one of whom had a classical 'shoulder hand syndrome' following herpes zoster of a lower thoracic dermatome. Such an association has not been previously described.

Case report

Case 1

A 66 year old Caucasian woman developed extensive herpes zoster affecting most of the right arm with a

patch of vesicles on the right leg. She was not given specific treatment and, in the weeks following resolution of the skin lesions, she developed severe pain in the right arm associated with oedema of the arm and fingers and the picture of right shoulder capsulitis. The skin of the affected limb was red and shiny and she noted excessive sweating.

The capsulitis gradually resolved over a period of 6 months with mobilization and physiotherapy and 15 months later, she was pain free. However, she had a residual 'intrinsic plus' deformity of the right hand with flexion of the metacarpophalangeal joints and hyperextension of the proximal interphalangeal joints. Nerve conduction studies showed substantial delay in the sensory action potential of both the median and ulnar nerves on the right. Thermography and blood flow measurements were compatible with algoneurodystrophy in the hypervascular phase and hand radiographs showed patchy osteoporosis. Despite intensive physiotherapy, the deformity persisted although the oedema and vasomotor abnormalities resolved.

Case 2

A 66 year old Caucasian female developed low lumbar pain following trauma and, a few days later, developed severe right buttock pain accompanied by vomiting. The typical rash of herpes zoster appeared, extending down the lateral aspect of the right lower leg. Within a few days, diffuse swelling of the right foot and distal third of the lower leg developed with severe burning pain and tenderness. The overlying skin was red and shiny. The patient noted numbness of the first and second toes and inability to dorsiflex the hallux. There was no evidence of deep vein thrombosis or arthropathy.

Three months later, the right foot remained oedematous with shiny red skin and only slight weakness of extensor hallucis longus and diminution

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of pin prick in an L5 distribution. Neurological examination was otherwise normal. Radiographs showed degenerative changes in the lumbar spine and patchy osteoporosis of the right foot and ankle. A diagnosis of algoneurodystrophy secondary to L5 herpes zoster was made. She was treated with simple analgesics and physiotherapy and made a slow recovery.

Case 3

A 72 year old Caucasian woman presented with a history of herpes zoster affecting the left T12–L1 dermatomes. Before the rash had completely resolved, her shoulder stiffened with pain radiating down the left arm to the hand. Six weeks after the onset of symptoms, both active and passive movements of the left shoulder were grossly restricted and painful, and the left hand was oedematous with shiny skin. There were no other articular findings or neurological deficit.

Radiological examination revealed gross osteoporosis of the left hand, particularly over the phalanges and metacarpals (Figure 1). X-ray of the left shoulder was normal but her technetium radio-isotope scan showed increased uptake in the left shoulder and hand. She was treated with intensive physiotherapy, simple analgesics and transcutaneous nerve stimulation, but shoulder pain persisted. She was later given three, fortnightly, 40 unit ACTH injections with temporary benefit. Shoulder and hand function steadily improved over the next 10 months.

Figure 1 Severe osteoporosis affecting the left hand in case 3.

Discussion

Algoneurodystrophy may be initially devastating and the long term prognosis is variable even if the treatment is initiated in its early stages. Some workers advocate systemic steroid treatment,² the rationale being the mild inflammatory changes found in synovial tissue of these patients.⁷ Steinbrocker and Argyros in 1958² found systemic steroids and stellate ganglion block to be equally effective but use of the latter treatment has declined in popularity since then. Currently, the mainstay of treatment is mobilization and physiotherapy. Although the pathogenesis of algoneurodystrophy is unclear, decreased mobility of the affected limb *per se* may be important. Finsterbush and Friedman⁸ demonstrated synovial changes and vascular proliferation in the immobilized knees of rabbits similar to that demonstrated in humans with algoneurodystrophy.^{2,7}

While the therapeutic effectiveness of stellate ganglion block^{1,2} confirms a major role for the sympathetic system in the pathogenesis of the syndrome, the role of pain afferents in the establishment of a 'reflex arc' is largely speculative.^{2,6} Sensory input causing hyperactivity in segmental interneurons has been proposed but cannot explain case 3 where the dermatome affected by herpes zoster was 12 cord segments removed from the involved limb. Motor involvement in cutaneous herpes zoster may complicate involvement of the dorsal root ganglia and sensory horn of the spinal grey matter and involves perhaps 5% of all cases.⁹ This is thought to be due to direct invasion of the anterior horn by virus. However, Thomas and Howard⁹ found that 10% of cutaneous zoster related motor deficits occurred several cord segments away from the skin lesion. Such 'dissociated' motor deficits may have a pathogenetic mechanism in common with the case we describe.

The plethora of descriptive terms and partial forms of algoneurodystrophy, as well as our poor understanding of its pathogenesis, contribute to the lack of clinical awareness of this condition and may explain why the diagnosis is often missed. It can mimic articular, vascular and neurological disease but the distinctive symptom complex of algoneurodystrophy should readily be recognized and clinicians are reminded that it can be precipitated by a wide variety of clinical events.

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