CASE REPORT CARPAL TUNNEL SYNDROME COMPLICATED BY REFLEX SYMPATHETIC DYSTROPHY SYNDROME

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SUMMARY

This report describes three patients with reflex sympathetic dystrophy complicating carpal tunnel syndrome. Pain disproportionate and more diffuse than that commonly associated with carpal tunnel syndrome suggested the associated diagnosis in each patient and facilitated prompt management.

KEY WORDS: Entrapment neuropathy, Shoulder-hand syndrome.

REFLEX sympathetic dystrophy syndrome (RSDS) is characterized by extremity pain and autonomic nervous dysfunction. Most events are precipitated by trauma or surgery but central nervous system abnormalities, peripheral neuropathy, cervical osteoarthritis and myocardial infarction may be underlying disorders [1]. RSDS has been reported as a complication of surgical carpal tunnel release in 5% of patients [2, 3], although it is not commonly recognized in untreated carpal tunnel syndrome (CTS). We describe three patients with recent onset CTS, who then evolved into the clinical syndrome of RSDS in the absence of surgical intervention. CTS was associated with polymyalgia rheumatica (PMR) in one patient, followed excessive wrist activity in another, and was the initial symptom of a seronegative inflammatory polyarthritis in the third.

CASE REPORTS

Case 1

A 60-year-old woman was diagnosed and treated for PMR with low dose corticosteroids for 18 months. She remained well for more than 2 years off steroid but then had a relapse of typical PMR. She did not seek medical advice until 9 months later when she developed paraesthesiae and numbness of the thumbs and second and third fingers of both hands. Initially the paraesthesiac were worse at night but subsequently persisted throughout the day. Bilateral median nerve dysfunction at the level of the carpal tunnel was confirmed on nerve conduction studies. Two months later, she experienced severe bilateral diffuse hand pain, swelling, and a blue mottling of the hands and arms. Her hands were puffy and swollen (Fig. 1). There was marked tenderness of the soft tissues of the hands and forearms with associated hyper aesthesia. The shoulders were stiff and limited as were the hips. She had clinical evidence of persistent bilateral carpal tunnel syndrome. The sedimentation rate was elevated to 45 mm/h (Wintrobe) she was not anaemic. Rheumatoid factor was negative. Liver function tests were normal. The technetium bone scan with flow studies showed increased uptake of both, the right and the left hands, wrists, elbows and shoulders consistent with a diagnosis of reflex sympathetic dystrophy syndrome.

She was treated with prednisone 30 mg/day and within 2 weeks had marked improvement of symptoms of RSDS and CTS. The ESR returned to normal within 10 weeks and she was maintained on prednisone 7.5 mg/day for control of the PMR. CTS was the presenting feature of recurrent PMR In this patient and RSDS occurred: months after onset of nerve root entrapment symptoms. Her response to steroid treatment was rapid (Fig.2).

Case 2

A 66-year-old man, previously in good health and unaccustomed to prolonged vigorous exercise, spent a few days sawing wood, A few days later he developed persistent paraesthesiac of the right hand in the median nerve distribution. One month later, he experienced diffuse pain in the right hand and painful right shoulder with a reduced range of motion. The shoulder pain improved somewhat following a steroid injection. The right hand was swollen and mottled and he was unable to make a fist. There was tenderness and hyperaesthesiae over the whole of the right hand, wrist and forearm.

FIG. 1.-Hands of patient 1 at presentation showing diffuse swelling.

Figures will be added in future versions of this media

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He had a positive wrist flexion test and a decreased light touch in the median nerve distribution consistent with a clinical diagnosis of CTS. The CBC and sedimentation rate were normal. Tests for rheumatoid factor and antinuclear antibody were negative and protein electrophoresis was normal. Chest X-ray was normal. He did not respond to therapy with non-steroidal anti-inflammatory agents and a week later was treated with prednisone 40 mg/day. Within 2 weeks he had a marked improvement in the pain and swelling of the hand and less dysaesthesiae in median nerve distribution. Prednisone was gradually reduced and discontinued over the following 3 months. The symptoms of carpal tunnel syndrome subsided spontaneously, although he was left with residual numbness of the inner aspect of the right third finger. This patient experienced clinical carpal tunnel syndrome following excessive wrist activity and RSDS developed 1 month later. Both the carpal tunnel syndrome and the RSDS responded promptly to systemic corticosteroids.

Case 3

An 81-year old lady, who was previously in good health, presented with a 4 month history suggestive of left CTS and a 2 month history of a swollen painful left hand, her left shoulder and elbow were stiff. Median nerve compression at the level of the left carpal tunnel was confirmed by nerve conduction studies. She denied systemic symptoms, other peripheral joint complaints or trauma. In addition to clinical findings of CTS she had diffuse swelling and tenderness of the whole of the left hand and was unable to make a fist. The hand was cold and mottled there was reduced and painful range of motion of the left shoulder and elbow. She was not 'anaemic and the ERS was 'slightly elevated 'it 30 mm/h ('Wintrobe). The rheumatoid factor and antinuclear antibody were negative and the blood glucose was normal. X-rays of the hands showed osteoarthritis only. She partially responded to prednisone 40 mg/day, but subsequently required a surgical carpal tunnel release. The signs of RSDS improved gradually and prednisone was discontinued after 4 months. She was left with a residual median nerve sensory deficit. One year' later she developed a low grade seronegative inflammatory polyarthritis of the opposite hand. The initial symptom of polyarthritis in this patient was of left CTS complicated by RSDS. Steroids improved symptoms of RSDS, but surgical decompression for the carpal tunnel was required.

DISCUSSION

Precipitating factors commonly associated with RSDS include trauma, surgical procedures, infection, myocardial infarction 'and neurological disease [1]. This syndrome has been described as complicating surgical carpal tunnel decompression in about 5% of patients [2, 3], but to our knowledge has not been described as a complication of CTS prior to surgery. We have described three patients who developed classical symptoms of CTS and within a 1-2 month period developed signs and symptoms of RSDS. Our patients were all seen early in the course of their disease and all had the characteristic findings of diffuse hand and forearm swelling, extreme tenderness of soft tissues as well as hyperaesthesiae which was nor limited to the distribution of the median nerve. The neurological symptoms in the median nerve distribution suggested the primary diagnosis although the most prominent clinical findings in all three patients were those of the RSDS.

Each of our patients had a clearly recognizable precipitating cause for carpal tunnel syndrome [4]. Patient I had PMR, which has previously been associated with carpal tunnel syndrome [5], the second patient had wrist trauma following sawing wood and the third patient had limited seronegative polyarthritis. The first two patients had a rapid and successful response to treatment with prednisone most likely due to the short duration of symptoms. Patient 3 had a

more prolonged course, required carpal tunnel decompression and has subsequently demonstrated low grade polyarthritis of the opposite hand.

These patients illustrate another precipitating cause of RSDS. Although peripheral nerve injuries are a common cause of RSDS, this syndrome is most commonly seen when there has been severance or a significant traumatic insult to a peripheral nerve [1]. The rapid and complete response to treatment in two of our patients suggests that prompt treatment may prevent a more prolonged and disabling course of the disease [6].

The diagnosis of RSDS should be suspected in any patient presenting with symptoms out of proportion to that commonly observed with a particular condition such 'is carpal tunnel syndrome Recommendations concerning treatment for RSDS arc varied but it is generally accepted that early mobilization and steroid treatment ire successful in the majority of patients [1].

Fig. 2.- Same patient 6 months later showing complete resolution of swelling.

Figures will be added in future versions of this media

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REFERENCES

1. Schwartzman RJ - McLellan TL. Reflex sympathetic dystrophy. A review. Arch Neurol 1987;44:555-61

2. MacDonald RI. Lichtman DM, Hanlon II, Wilson JN. Complications of surgical release for carpal tunnel syndrome. J Hand Surg [Am] 1978:1:706.

3. Lichtman DM, Florio RL. Mack GE. Carpal tunnel release under local anaesthesia: evaluation of the outpatient procedure. J Hand Surg 1979;4:544-6.

4. Gelberman RH. Rydevik BL. Pess GM. Szabo RM. Lindborg G. Carpal tunnel syndrome a scientific basis for clinical care. Orthop Clin North Am 1988; 19: 115-24.

5. Wysenbeek AJ, Calabrese LH, Scherbel AL. Reflex sympathetic dystrophy syndrome complicating polymyalgia rheumatica. Arthritis Rheum 1981; 24: 863-4

6. Goldner JL. Causes and prevention of reflex sympathetic dystrophy (letter). J. Hand Surg [Am] 1980; 3:295-6.

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