lesson of the month (2)

Herpes zoster brachial plexopathy with predominant radial nerve palsy

Herpes zoster or shingles is the reactivation of dormant varicella zoster virus (VZV) in the dorsal root ganglia. Segmental motor paresis is rare and only few cases of brachial plexitis have been reported in the literature. This case reports herpes zoster resulting in unilateral brachial plexitis with predominant radial nerve palsy. The patient was treated successfully with aciclovir, gabapentin and physiotherapy with good recovery. Radial neuritis secondary to active herpes zoster has been rarely reported in the past.

Introduction

Herpes zoster or shingles is the reactivation of dormant varicella zoster virus (VZV) in the dorsal root ganglia. The vesicular skin lesion arises in specific dermatome of the sensory nerve. The commonly involved nerve segments are thoracic, lumbar and trigeminal. Motor paresis occurs in less than 5% of herpes zoster patients.[1] Brachial plexus involvement is a rare occurrence. Radial nerve involvement in herpes zoster lesion of the brachial plexus is very rare. This case reports unilateral radial nerve paralysis due to shingles.

Lesson

An 83-year-old female patient was admitted to hospital complaining of right-hand weakness and arm pain for one-week duration. The patient was diagnosed to have shingles, by the general practitioner, with history of vesicular rash on posterior aspect of right arm for one week. On examination of the right upper limb there were vesicular rashes over the posterior aspect along C8/T1 dermatomal distribution. Marked decrease in active wrist and finger extension, reduced power in wrist and finger extensors and reduced thumb abduction were noted. There was also decreased sensation in C7/C8 dermatomes. The rest of the peripheral and central neurological examinations were normal. Routine blood tests were normal. Diagnosis of brachial plexopathy was confirmed by a consultant neurologist. Conservative treatment with aciclovir 800 mg five times daily was continued along with gabapentin, for pain relief, and physiotherapy. Nerve conduction study confirmed severe right-sided brachial plexus lesion affecting the distribution of the radial nerve. The extensor digitorum and first dorsal interosseus muscles markedly affected with mild weakness of abductor pollicis brevis. The patient was discharged and followed-up in the outpatient clinic showing progressive improvement in hand functions. There was no postherpetic neuralgia or any other complications.

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Discussion

Herpes zoster, commonly known as shingles, is the reactivation of dormant VZV of the dorsal root ganglia. The virus then moves along and presents as unilateral painful vesicular rash in dermatome supplied by the sensory nerve. Shingles occur more commonly in the elderly population and are approximately 8 in 1,000 patient years in those over 70 years of age.[2] Cell-mediated immunity plays an important role of preventing the reactivation of the virus.[3] Elderly population, trauma, lymphoma and immunosuppressant state are the main predisposing factors for the development of shingles. Prodromal dermatomal pain and paraesthesia starts two to three days before the onset of vesicular rash in the region of skin supplied by the affected sensory nerve. The vesicles progress into pustules (4 to 6 days), scab (7 to 10 days) and finally the healing process (2 to 4 weeks).[4] The important neurological complication of herpes zoster is the postherpetic neuralgia; other complications are motor neuropathy, cranial nerve palsy, transverse myelitis, encephalitis, herpes zoster opthalmicus, pneumonitis and hepatitis.[5]

Motor neuropathy occurs in 0.5 to 5% of herpes zoster patients.[1] Cranial nerve paralysis is the most common motor paralysis accounting for 80% of all motor paresis in herpes zoster cases.[6] Segmental limb paresis is rare with preponderance to cervical segments and thoracic segments are the least common. Upper limb motor paresis involving the proximal muscles has been reported more frequently than in the lower extremity.[7] Few case reports of brachial plexus neuritis causing upper limb paresis secondary to herpes zoster have been reported in the past.[8–10] In all these case reports the paresis was in the superior or middle trunks of the brachial plexus. Ulnar nerve involvement was reported by Athwal *et al.*[11] There were no reports of predominant radial nerve involvement in the literature. Our case clearly demonstrates the predominant radial nerve palsy associated with cutaneous vesicular lesions. The brachial plexus involvement in herpes zoster is due to distal extension of the inflammation in the sensory ganglion.[8]

The mainstay of treatment of motor paresis or brachial plexus neuritis due to shingles is antivirals, pain relief, physiotherapy and occupational therapy. The commonly used effective

antiviral is aciclovir. Some studies have suggested the use of corticosteroids in combination with antiviral to reduce pain and hasten the healing process.[12] Our experience of reduced severity of postherpetic neuralgia is consistent with documented evidence.[13] In patients with segmental paresis, complete or partial recovery is about 76% after two years.[6]

Physicians and orthopaedic surgeons need to be aware of herpes motor paresis as a cause for radial nerve palsy especially when associated with skin lesions. Neurological examinations and physiological nerve conduction study will confirm the diagnosis. Multidisciplinary input and prolonged follow-up is needed in treating these patients. The combination of aciclovir and gabapentin, along with physiotherapy, hastens the recovery. In conclusion, herpes zoster causing radial nerve palsy is a rare occurrence but nevertheless needs to be kept in mind as a differential diagnosis for unilateral radial nerve damage.

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book reviews

A short history of medical genetics

By Peter S Harper. Oxford University Press, Oxford 2008. 576 pp. £31.99

I very much enjoyed this book. It provides a thoughtful overview of the development of medical genetics, tracing the evolution of ideas from over 300 years ago to the present day. The different aspects of the discipline are discussed in separate sections, with helpful notes and recommended sources provided at the end of each chapter. It describes how certain key geneticists contributed to the development of these ideas, and provides tantalising, but brief, insights into their personalities and relationships. The only drawback to the chapter/subject approach is that the same players may reappear at different stages in the narrative, when the main discussion about the individual (using a short biography inset) occurs once, giving a curiously time-skipping effect as in some books where earlier and later events are inter-digitated.

The chapter 'Before Mendel' describes some of the early ideas of genetic disorders, with intriguing examples, such as the work of Joseph Adams (1756–1818), who not only distinguished between hereditary congenital disorders and 'dispositions' (where the disorder develops gradually over a lifetime), but also categorised 'predispositions', where an external factor is also needed for the disorder to become apparent in an individual. Further, he opposed celibacy in the family members of individuals affected by 'madness', as a measure to reduce the likelihood of increasing the burden of the disorder, since there had been no observed increase in the frequency of the disorder, and such restraint would only be heeded 'by the most amiable and best disposed' an idea that preceded those of the anti-eugenicists in the mid-1990s. Interesting quotes, such as a poem written by Erasmus Darwin entitled the *Temple of nature*, illustrate the descriptions of the early thinkers in this subject, starting from the 1750s, and it is fascinating to see the evolution of ideas developing from 'the continuity of the germplasm' to our modern understanding of inheritance.

Phenylketonuria (PKU) is used as an example of a condition with autosomal recessive inheritance with the potential for treatment, and the potential for eugenic issues in prevention. Penrose explored PKU as a paradigm for carrier detection, dietary treatment of affected individuals, and approaches to prevention. He examined the argument regarding contemporary promotion of the prevention of recessive diseases by restraint of procreation in carriers and commented:

> *to eliminate the gene from the racial stock would involve sterilising one percent of the population if carriers could be identified. Only a lunatic would advocate the procedure to prevent the occurrence of a handful of harmless imbeciles.*