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Case Presentation

Herpes Zoster Induced Predominant Motor Lumbal Plexopathy in a Lung Cancer Patient

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Abstract

Herpes zoster is the reactivation of dormant varicella zoster (VZV) in the dorsal sensory root ganglia. Segmental limb paresis is rare and only a few cases have been reported in the literature. The pathophysiology of motor involvement is poorly understood. We present a case of herpes zoster lumbal plexopathy with predominat motor involvement both clinically and electro physiologically in a cancer patient.

Keywords: Herpes Zoster; Lumbar Plexopathy; Zoster-Associated Limb Paresis

Introduction

Varicella zoster virus belongs to the family of alpha herpes virus. After a first infection, usually in childhood, it causes varicella (chickenpox) and the virus becomes latent in the dorsal root ganglia of sensory and autonomic nerves [1]. Decline in virus-specific cell-mediated immune responses, which occur naturally as a result of aging or are induced by immunosuppression, may lead to the reactivation of the virus, resulting in local proliferation leading to herpes zoster (shingles), which is characterized by neuropathic pain and a vesicular rash in the affected dermatome [2]. Patients with neoplastic diseases (especially lymphoproliferative cancers), those receiving immunosuppressive drugs (including corticosteroids), and organ-transplant recipients are at increased risk for herpes zoster [3]. Because of high morbidity and severe complications, re-vaccination is recommended for this population [4]. The incidence of herpes zoster is approximately 3.6 per 1000 person-years in the USA [5]. A high rate of patients (about 60-70%) has to be hospitalized because of complications like encephalitis, transverse myelitis and pneumonitis. Herpes zoster is often complicated by chronic radicular pain (postherpetic neuralgia), which affects approximately 20% of all herpes zoster patients and about one third of patients > 80 years of age [5]. Herpes zoster mostly arises in the first branch of the trigeminal nerve and the sensory nerve roots of the thoracic and lumbar spinal nerve segments. Motor involvement occurring in the context of herpes zoster is rare and occurs in less than 5% of herpes zoster patients [6,7].

Case Presentation

A 75-year-old man was diagnosed of non-small cell lung (NSCLC) cancer diagnosed 4 months before referral to the neurology department. According to his nicotine abuse he additionally suffers from cerebrovascular disease, an obstructive pneumopathy and arterial hypertension. With a cancer stage IV disease he was symptomatically treated with focal radiotherapy of 39 Gray to the bulky tumor of the lung and sequentially received one cycle of a palliative chemotherapy with carboplatin and paclitaxel.

Two months after initiation of chemotherapy he noticed neuropathic pain at the inner site of his left thigh and lower back pain. A few days later a vesicular rash occurred in the affected region, which could be attributed to the left radicular segments L1-4. With the suspicion of localized herpes zoster, he was treated with oral valaciclovir and pregabaline as well as morphine against neuropathic pain.

His general condition declined as he had suffered from a fall. Beside a new arising lumbago, which was nociceptive and different from the former mentioned neuropathic pain, a few days later he noticed severe weakness and atrophy of his left thigh and was not able to climb the stairs or to properly stand up from a chair.

On neurological examination a few weeks later he showed hyper pigmented skin rash on the left side of his lower back and on the medial and ventral side of his thigh (Figure 1a/b). Beside an atrophy of the quadriceps muscle (Figure 1b) he had mild paresis of the left hip flexion, knee extension and leg adduction. Patellar- and adductor tendon reflexes were absent on the left side while the achilles tendon reflex was preserved. Moreover, he noticed only a slight hyperaesthesia in the area of the left femoral and obturatorial nerve.

Nerve conduction studies on the sural, tibial, femoral and peroneal nerve were normal without a side difference including F-waves, but electromyographic studies showed acute denervation of the adductor magnus-, iliopsoatic-and medial vastus muscle, whereas paraspinal electromyography on the level of the according lumbal segments was normal.

Therefore, medical history, clinical examination and electrophysiological examination revealed the pattern of a predominant, postganglionic motor neuropathy of the upper left lumbar plexus due to local herpes zoster infection. Later on MRI scan of the spine revealed a fracture of the vertebra L4 without compression of the cauda equine and no signs of leptomeningeal dissemination which was the serious differential diagnosis.

Discussion

To the best of our knowledge this is the first report of a cancer patient with a predominant motor lumbal plexopathy due to herpes

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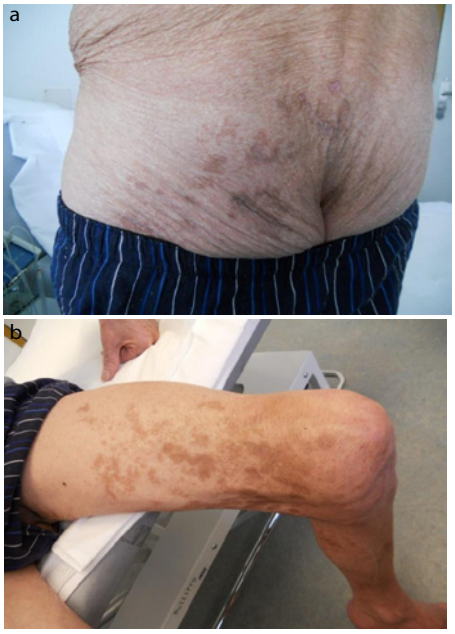

Figure 1: Herpes zoster vesicular rash in the dermatoms L1-L4 (a and b) and severe atrophy of the quadriceps muscle on the left (b).

zoster infection. Symptomatic varicella zoster reactivation is a frequent phenomenon in older and immunocompromised patients [3]. Like in our patient cancer and chemotherapy either alone or in combination are strong risk factors for the development of herpes zoster. This condition mostly leads to severe patient discomfort and lowers quality of life [8].

In our patient the predominant sign of herpes zoster associated lumbal plexopathy was motor weakness in the affected limb. Beside a neuropathic pain syndrome sensory symptoms were almost absent. As our patient suffers from a stage IV metastasized carcinoma other differential diagnosis has to be excluded. Direct infiltration of the lumbal plexus was unlikely due to the corresponding vesicular rash in the affected limb and the recoverage of the symptoms weeks after antiviral therapy. Moreover, leptomeningeal disease also would not recover without adequate therapy and MRI of the lumbal spine later on was unsuspicious despite a L4 vertebra fracture, which cannot be responsible for the symptoms, as multiple and more cranial lumbal segments were involved clinically and electrophysiologically. An involvement of the dorsal rami of the lumbal radices were not suspected on electrophysiological examination. Therefore, the lesion was localized to the lumbal plexus as paraspinal muscles were not affected.

Weakness associated with herpes zoster is rare and can affect bulbar, limb and truncal muscles. Motor paresis almost always follows the appearance of typical vesicular rash by an average of 2-3 weeks and, in greater than 90% of cases, occurs in the same segmental distribution as the rash [9]. Cranial nerve paralysis is the most common motor paralysis accounting for 80% of all motor paresis in herpes zoster cases [10]. Just a few reports have detailed the limb paresis; mostly involving the proximal muscles of upper extremities [11,12]. The pathophysiology of herpes zoster is well characterized, but the precise mechanism of motor abnormalities is poorly understood. The wide variety of neurological complications of herpes zoster may be attributed to the ability of varizella zoster virus to infect many cell types [13]. Most zoster paresis clinical series have postulated that the root or anterior horn cell, and less commonly the more distal structures, are the sites of motor injury [9,14,15]. Fabian et al demonstrated in a postmortem case the diffuse inflammatory demyelinating process of all trunks in a brachial plexus neuritis, which could explain the good recovery observed in many patients [16]. In our case, the electrophysiological findings suggest however an axonal neuropathy. According to the latency from first symptoms (pain) to the motor weakness a secondary inflammatory process involving the axons of the motor fibres is the most likely cause in our patient. Direct involvement of the anterior horn cells seems to be unlikely as absence of acute denervation in the ramus dorsalis of the affected myotoms suggests a more distal pathology at the level of the plexus.

As the differential diagnosis of motor plexopathy in cancer patients are numerous, a profound history taking, neurological examination and electrophysiology are most important to yield the correct diagnosis and initiate an appropriate therapy. Furthermore, close interdisciplinary clinical interaction between the oncologists and the neurologists preserved further examinations in this old patient being in a supportive care situation due to cancer stage IV.

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