Lower back pain with sciatic disorder following L5 dermatome caused by herpes zoster infection

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Abstract

We report the case of a 62-year-old patient with lower back pain radiating into the right leg accompanied by numbness. The pain had an acute onset and was resistant to conservative pain treatment. A magnetic resonance imaging (MRI) scan of the lumbar spine showed no degenerative discovertebral lesions, but a swelling of the nerve root supplying the affected dermatome. For pain treatment the patient received lumbar epidural infiltrations. During this treatment the patient suddenly developed a skin rash with grouped vesicular blisters on an erythematous ground. After the diagnosis of a lumbar herpes zoster and an acyclovir treatment, the patient could be discharged in an ameliorated condition. This case demonstrates the importance to consider rare causes of lumbosciatic pain and disorders and to acknowledge unspecific changes in a MRI scan.

Introduction

Lower back pain with radiation into the legs is a common clinical picture in orthopedics. The pain may be accompanied by numbness or weakness of muscles. Most often these disorders are caused by degenerative discovertebral lesions such as herniated discs, spondylolithesis or hypertrophic spondylarthrosis generating a spinal stenosis. For verification of the diagnosis and further treatment options, especially if the complaints are persistent, an imaging procedure will usually be performed. The method of choice is the magnet resonance imaging (MRI). If such an imaging procedure does not show typical degenerative discovertebral lesions one should keep in mind rare differential diagnosis of lumbosciatic pain and be cautious to note unspecific alterations in the MRI. These unspecific findings may be the first and sometime only hint for a non-discovertebral disease such as herpes zoster.

Case Report

A 62-year-old man was admitted to our orthopedic hospital presenting severe lower back pain and a sciatic disorder following the L5 dermatome. The patient described the pain starting 6 days ago with a sudden onset. Initially, the patient took several non-steroidal anti-inflammatory drugs, such as diclofenac 150 mg and metamizol 1000 mg per day, without having any pain relief. After 3 days the patient consulted his general practitioner, where he received a local pain relief injection with a corticosteroid. Having no pain relief again and with a progressive symptomatology in the right leg accompanied by numbness, the patient was admitted to our hospital.

At admission the patient showed severe lumbar spine pain with radiation alongside the dermatome L5 on the right side till the first toe persisting for 6 days and suggesting a nucleus pulposus prolapse. Since 2 days the patient had a hypoesthesia in the same dermatome with a maximum in the lower leg and foot. The femoral nerve stretch test was positive on the right side and the straight leg raise test was early positive at 20° elevation of the right leg while these tests were negative on the left side. The Achilles tendon reflex and the patella tendon reflex could be provoked on both sides similarly. There was no motoric deficit in both legs, no bladder or bowel dysfunction, and no saddle anesthesia. Importantly, the skin had no abnormal findings. The medical history was unremarkable except of arterial hypertension and a nucleotomy of a cervical disc. For ambulant non-controllable pain the patient was admitted to our hospital. Under intravenous pain therapy the medical condition ameliorated slightly. For differential diagnosis the patient underwent a MRI scan of the lower back. This showed no herniated disc but a slight spinal stenosis between L4 and L5, as well as, an unspecific swelling of the spinal nerve root L5 on the right side (Figure 1). Based on these findings a conservative treatment including oral analgesia, physiotherapy and a series of lumbar epidural corticosteroid infiltrations was initiated. After the first lumbar epidural infiltration the patient developed a stain on the skin of his lower back. A closer examination of this mark showed an erythema with a group of small red blisters which had been partially dried out (Figure 2). There was no sign for a bacterial skin infection induced by the infiltration therapy. Also, the laboratory infection parameters including the leucocyte count and the C-reactive protein showed normal values.

The epidural infiltration therapy was stopped immediately and the patient was sent to a dermatological specialist who diagnosed a herpes zoster infection with neuralgic pain and hypoesthesia in the dermatome L5. After a pain therapy with ibuprofen 600 mg 3 times a day combined with a systemic antiviral treatment with acyclovir 800 mg oral 5 times a day for a period of 7 days the patient could be discharged soon from our hospital in a considerable ameliorated condition.

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Discussion

Herpes zoster is a viral disease with reactivation of the varicella zoster virus (VZV). After a primary infection most often during childhood the virus persists in the spinal ganglion. In case of decreasing immunity the VZV can be reactivated and travels anterograde to the skin causing an inflammation of nerves and zoster.[1] The rate of reactivation is stated with 20%.[2] Most often the localization is thoracic with 50-56% and at the head with 20%.[2] Rarely the virus affects lower nerval segments and causes symptoms of pain, burning and dysesthesia mimicking lumbosciatica as seen in our case. There is no exact incidence of lumbar herpes zoster stated in literature. Usually the trigger for viral reactivation and inflammation is an immunosuppression, such as a malignant tumor, an immunosuppressive treatment or a chronic immunosuppressive disease.[1-3] Our patient did not show a relevant medical history. However, trauma, stress or an age ≥50 years are also described as triggering factors.[4]

In case of doubt of a discovertebral origin for lumbosciatica and the suspicion of a herpes

zoster neuralgia the diagnostics should be carried out soon, since an early treatment is inevitable due to the risk of persisting symptoms.[3] The therapy of herpes zoster includes pain treatment and an antiviral treatment. Pain treatment may be performed according to the WHO analgesic ladder and co-analgesics, such as amitryptiline. Local infiltrations for pain relief in patients with herpes zoster including selective nerve root block and epidural infiltrations as initially performed in our case are described.[5-7] However, due to the use of corticosteroids and the risk of a viral reactivation because of the infiltration itself, these procedures remain controversial.[8,9] An antiviral therapy should be started within 48-72 hours after the onset of symptoms.[2,3] The antiviral therapy should be given for at least 7 days and can be conducted oral or intravenous. A common antiviral agent is acyclovir which can be given both oral (800 mg 5 times per day) and in severe cases intravenous (5-7.5 mg/kg 3 times per day).[2] Alternatives for oral treatment are valacyclovir and famciclovir, for example. Indications for an antiviral therapy include an age beyond 50 years, an immunosuppression, severe zoster of the trunk of extremities, zoster of the head, neck or sacral area, and severe dermatitis.[2] Complications of herpes zoster in case of a late or missing diagnosis may be post-herpetic neuralgia or postzosteric neuropathies. The manifestation rate of post-herpetic neuralgia increases with the age of >55 years and amounts to 73% in patients older than 70 years.[2] Postzosteric neuropathies as numbness and paresis will only recover in 50%.[2] The diagnostic may be prolonged if the skin rash appears late, as in our case, or if there is no skin rash at all (zoster sine herpete). In 80% of herpes zoster the skin rash appears after 3-5 days of prodromal pain and paresthesia.[2] Yet, the prodromal stage may be longer or the cutaneous affection can be absent at all. In these cases inconspicuous diagnostic signs become more important. In our patient a discovertebral origin of the complaints could be ruled out via MRI. However, the patient showed a nerve root enlargement of the affected segment. Retrospectively, this nerve enlargement is to be seen as a vasculitis and inflammation of the nerve in the context of a reactivated VZV. MRI findings signaling a herpes zoster are unspecific and inconsistent. Whereas several cases report no pathologic findings in a MRI,[10-13] there is proof that a MRI can show nerve enlargement or enhancement.[13-15] One study showed the variation of imaging abnormalities based on the electrodiagnostic localization of the lesions.[16] In patients with preganglionic electrodiagnostic lesions the MRI showed no abnormalities. In patients with root lesions the MRI of the spine did not show abnormalities whereas the MRI of the plexus showed abnormalities in 50%. In patients with electodiagnostically defined postganglionic lesions 64% showed alteration of the plexus or nerves in the MRI of the plexus or nerves whereas only 9% showed spinal cord or root abnormalities in a MRI of the spine. In case of doubt or lacking skin rash laboratory diagnostic may be helpful. The VZV polymerase chain reaction and the detection of VZV in cell cultures are well established methods.[2] Furthermore IgM- and IgA-anti VZV antibodies may be examined in blood.[2] If the typical skin rash with grouped papulovesicular lesions is present, a clinical diagnosis, as in our case, can be sufficient and an antiviral treatment can be initiated immediately.

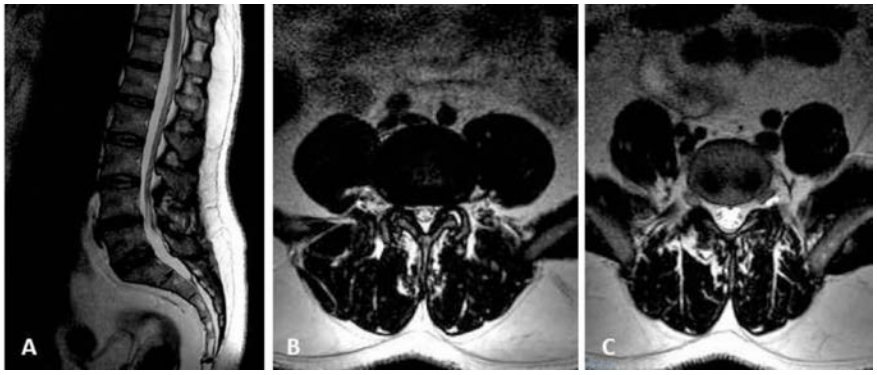

Figure 1. Sagittal magnetic resonance imaging (MRI) of the lumbar spine in T2 (A), transversal MRI of the lumbar spine in T2 showing a protrusion of the disc between L4 and 5 causing a slight spinal stenosis (B) and a moderate swelling of the right spinal nerve root L5 (C).

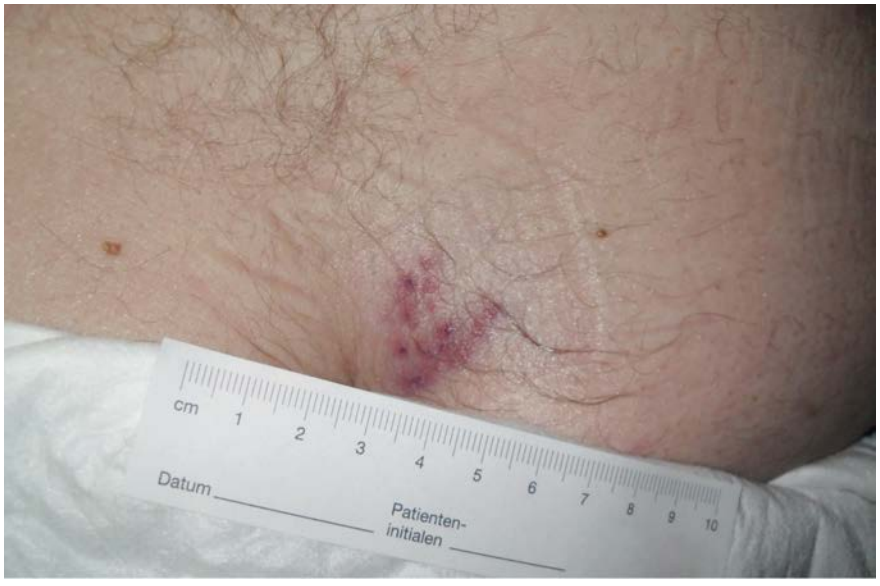

Figure 2. Local erythema with blisters above the intergluteal cleft.

Conclusions

A lower back pain with disorder of the L5 dermatome most often derives from a disc herniation or stenosis of the lumbar spine. Nevertheless, rare causes, such as an infection of the spinal nerve root with a herpes zoster virus, should also be taken into consideration. Especially if the pain occurs with a sudden onset, a MRI only shows slight modifications and a conservative pain therapy including analgesic drugs and corticoid infiltrations do not show any effect, a herpes zoster infection should never be excluded in the differential diagnosis. Also, it is important to pay attention to secondary findings, such as an erythema with blisters and, in case of doubt, refer for dermatologic evaluation.

References

1. Nagel MA, Gilden D. Neurological complications of varicella zoster virus reactivation. Curr Opin neurol 2014;27:356-60.
2. Gross G, Schofer H, Wassilew S, et al. Herpes zoster guideline of the German Dermatology Society (DDG). J Clin Virol 2003;26:277-89.
3. Bader MS. Herpes zoster: diagnostic, therapeutic, and preventive approaches. Postgrad Med 2013;125:78-91.
4. Hope-Simpson RE. The nature of herpes zoster: a long-term study and a new hypothesis. Proc R Soc Med 1965;58:9-20.
5. Conliffe TD, Dholakia M, Broyer Z. Herpes zoster radiculopathy treated with fluoroscopically-guided selective nerve root injection. Pain Physician 2009;12:851-3.
6. Shakir A, Kimbrough DA, Mehta B. Postherpetic neuralgia involving the right C5 dermatome treated with a cervical transforaminal epidural steroid injection: a case report. Arch Phys Med Rehabil 2007;88:255-8.
7. Hardy D. Relief of pain in acute herpes zoster by nerve blocks and possible prevention of post-herpetic neuralgia. Can J Anaesth 2005;52:186-90.
8. Parsons SJ and Hawboldt GS. Herpes zoster: a previously unrecognized complication of epidural steroids in the treatment of complex regional pain syndrome. J Pain Symptom Manage 2003;25:198-9.
9. Szokol JW, Gilbert HC. A herpes zoster outbreak temporarily associated with an epidural steroid injection. Reg Anesth Pain Med 1998;23:328.
10. Helfgott SM, Picard DA, Cook JS. Herpes zoster radiculopathy. Spine 1993;18:2523-4.
11. Koch P, Diedrich O, Pennekamp PH, Schmitz A. Rare differential diagnosis of a radicular spine syndrome: herpes zoster radiculitis. Z Orthop Ihre Grenzgeb 2006;144:583-6.
12. Kulcu DG, Naderi S. Differential diagnosis of intraspinal and extraspinal non-discogenic sciatica. J Clin Neurosci 2008;15:1246-52.
13. Tsai J, Bert RJ, Gilden D. Zoster paresis: asymptomatic MRI lesions far beyond the site of rash and focal weakness. J Neurol Sci 2013;330:119-20.
14. Reda H, Watson JC, Jones LK Jr. Zoster-associated mononeuropathies (ZAMs): a retrospective series. Muscle Nerve 2012;45:734-9.
15. Umehara T, Sengoku R, Mitsumura H, Mochio S. Neurological picture. Findings of segmental zoster paresis on MRI. J Neurol Neurosurg Psychiatry 2011;82:694.
16. Jones LK Jr., Reda H, Watson JC. Clinical, electrophysiologic, and imaging features of zoster-associated limb paresis. Muscle Nerve 2014;50:177-85.