

World Journal of *Clinical Cases*

World J Clin Cases 2019 September 26; 7(18): 2658-2915



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The *WJCC* is now indexed in PubMed, PubMed Central, Science Citation Index Expanded (also known as SciSearch®), and Journal Citation Reports/Science Edition. The 2019 Edition of Journal Citation Reports cites the 2018 impact factor for *WJCC* as 1.153 (5-year impact factor: N/A), ranking *WJCC* as 99 among 160 journals in Medicine, General and Internal (quartile in category Q3).

RESPONSIBLE EDITORS FOR THIS ISSUE

Responsible Electronic Editor: *Ji-Hong Liu*

Proofing Production Department Director: *Yun-Xiaoqian Wu*

NAME OF JOURNAL

World Journal of Clinical Cases

ISSN

ISSN 2307-8960 (online)

LAUNCH DATE

April 16, 2013

FREQUENCY

Semimonthly

EDITORS-IN-CHIEF

Dennis A Bloomfield, Bao-Gan Peng, Sandro Vento

EDITORIAL BOARD MEMBERS

<https://www.wjnet.com/2307-8960/editorialboard.htm>

EDITORIAL OFFICE

Jin-Lei Wang, Director

PUBLICATION DATE

September 26, 2019

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PUBLICATION MISCONDUCT

<https://www.wjnet.com/bpg/gerinfo/208>

ARTICLE PROCESSING CHARGE

<https://www.wjnet.com/bpg/gerinfo/242>

STEPS FOR SUBMITTING MANUSCRIPTS

<https://www.wjnet.com/bpg/GerInfo/239>

ONLINE SUBMISSION

<https://www.f6publishing.com>

Guillain-Barré syndrome in a patient with multiple myeloma after bortezomib therapy: A case report

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Author contributions: Xu YL wrote and revised the manuscript; Zhao WH and Tang ZY collected the clinical data; Li ZQ, Long Y, and Cheng P provided information consultation; Luo J reviewed the manuscript; all authors read and approved the final manuscript.

Supported by National Natural Science Foundation of China, No. 81660038.

Informed consent statement: Informed consent was obtained from the patient for this publication.

Conflict-of-interest statement: The authors declare that they have no competing interests.

CARE Checklist (2016) statement: The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016).

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Abstract

BACKGROUND

Bortezomib is a first-line drug approved for patients with multiple myeloma (MM) and has significantly increased their overall survival. However, bortezomib-induced peripheral neuropathy (PN) remains a significant side effect that has led to its discontinuation in some patients. Guillain-Barré syndrome (GBS) is recognized as an immune-mediated PN characterized by the involvement of multiple nerve roots and peripheral nerves and albuminocytologic dissociation in cerebrospinal fluid (CSF) tests. Intravenous immunoglobulin (IVIG) and plasmapheresis are effective.

CASE SUMMARY

A 45-year-old man diagnosed with stage III MM (λ type) was treated with bortezomib and dexamethasone. Fourteen days after the second course, he complained of intense burning sensation in the lower limbs and hands, loss of tactile sensation, and pain in the distal area of both thighs and in the distal part of both wrist joints. Neurological examination revealed absence of knee and ankle reflexes. CSF examination revealed albuminocytologic dissociation. Nerve conduction studies indicated sensory nerve action potential amplitudes, conduction velocity decrease, and F wave latency prolongation. He was diagnosed as MM complicated with GBS. Subsequently, he was treated with high-dose IVIG (400 mg/kg/d for five days). His symptoms fully resolved without relapse at the 6-month follow-up.

CONCLUSION

Our case highlights the differential diagnosis and management of complications after bortezomib treatment in MM.

Key words: Case report; Guillain-Barré syndrome; Multiple myeloma; Bortezomib

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Manuscript source: Unsolicited manuscript

Received: April 11, 2019

Peer-review started: April 12, 2019

First decision: August 1, 2019

Revised: August 13, 2019

Accepted: August 26, 2019

Article in press: August 26, 2019

Published online: September 26, 2019

P-Reviewer: Mesquita J

S-Editor: Dou Y

L-Editor: Wang TQ

E-Editor: Liu JH



Core tip: Multiple myeloma (MM) is a malignant clonal disease, which shortens the survival time of the patients. Bortezomib, a first-line drug for the treatment of MM, has prolonged patient survival. Therefore, the adverse reactions have become a hot topic. We can have a better understanding of its complications and treatment through this case.

Citation: Xu YL, Zhao WH, Tang ZY, Li ZQ, Long Y, Cheng P, Luo J. Guillain-Barré syndrome in a patient with multiple myeloma after bortezomib therapy: A case report. *World J Clin Cases* 2019; 7(18): 2905-2909

URL: <https://www.wjgnet.com/2307-8960/full/v7/i18/2905.htm>

DOI: <https://dx.doi.org/10.12998/wjcc.v7.i18.2905>

INTRODUCTION

Multiple myeloma (MM) is a malignant neoplasm with abnormal proliferation of clonal plasma cells, accounting for 10% of hematologic malignancies^[1]. Chemotherapy and autologous transplantation are the major therapeutic tools for MM. Bortezomib, a first-line drug approved for patients with MM, has reduced the risk of progression or death and prolonged patient survival. A common side effect of bortezomib is peripheral neuropathy (PN). Nearly half of the patients treated with bortezomib had PN of varying degrees, and 30% had moderate to severe PN. In some patients, the symptoms did not improve after treatment. Therefore, about 10% of patients have to discontinue bortezomib treatment because they cannot tolerate the pain caused by PN^[2]. About three months after withdrawal, 85% of patients did experience full relief. During follow-up after drug withdrawal, up to 30% of patients failed to have complete remission^[3].

Guillain-Barré syndrome (GBS) is recognized as an immune-mediated PN characterized by involvement of multiple nerve roots and peripheral nerves, usually associated with infection. The clinical features are characterized by an acute onset, rapid progression, symmetric muscle weakness, and unstable gait. The weakness is predominantly distal, and many patients feel neuropathic pain. However, there are a number of variants in which sensory abnormalities are the main feature. Cytoalbuminological dissociation in the cerebrospinal fluid (CSF) is seen, and intravenous immunoglobulin (IVIG) therapy or plasmapheresis is effective^[4,5].

In addition to bortezomib-induced PN, other factors may cause numbness in patients with MM. Herein, we present the case of a patient with MM who developed GBS after two courses of bortezomib therapy.

CASE PRESENTATION

Chief complaints

A 45-year-old man was hospitalized for intense burning sensation in the lower limbs and hands.

History of present illness

A 45-year-old man was hospitalized at the First Affiliated Hospital of Guangxi Medical University for pain in the sternum and ribs. According to the International Staging System, the patient was diagnosed with stage II MM (λ type) and stage III disease according to the Durie-Salmon Staging system. A chemotherapy protocol was followed, consisting of bortezomib (1.3 mg/m² on days 1, 4, 8, and 11, and every 21 d thereafter), cyclophosphamide, and dexamethasone. Numbness of limbs began on the fourteenth day after the second cycle of chemotherapy. The patient began to complain of intense painful and burning sensation in the lower limbs and hands, loss of tactile sensation, and pain bilaterally in the distal parts of the thighs and bilaterally in the distal parts of the wrist joint. Gabapentin and pregabalin did not improve his symptoms.

History of past illness

Nothing to declare.

Personal and family history

He have been smoking 20 cigarettes a day for more than 20 years.

Physical examination upon admission

The pain and tactile sensation of distal wrist joints of both upper limbs and ankle joints of both lower limbs had decreased. Knee-jerk and ankle reflexes were absent.

Laboratory examinations

The blood testing results were: Hemoglobin, 139.8 g/L (normal range, 130-175.00); creatinine, 54 $\mu\text{mol/L}$ (59-104); creatinine clearance, 81 mL/min (85-125); calcium, 2.13 mmol/L (2.080-2.600); albumin, 40.7 g/L, and globulin, 24.3 g/L. The 24-h urine analysis showed a total protein of 84.6 mg/24 h (0.0-120.0). Serum lactate dehydrogenase was 247 U/L (109-245) and beta-2 microglobulin was 3.03 $\mu\text{g/mL}$ (0.0-120.0). Serum immunoelectrophoresis showed no M protein band. Serum free light chain assay showed free light chain κ of 11.11 mg/L and free light chain λ of 21.69 mg/L. Folic acid and vitamin B12 levels were normal. Bone marrow had 3% plasma cells.

CSF pressure was 124 mmH₂O, CSF protein was 931.1 mg/L (150-450), and CSF cell count was $0 \times 10^6/\text{L}$. CSF syphilis antibody and *Cryptococcus neoformans* were negative. Paraneoplastic antibodies in CSF and serum were negative, which included anti-Hu, Yo, Ri, CV2, Ma2/Ta, amphiphysin, ANNA3, Tr, PCA2, and GAD.

Nerve conduction studies: The sensory conduction velocity and amplitude of the median nerve and ulnar nerve were normal bilaterally, and the motor conduction velocity and amplitude were also normal. The latency and occurrence rate of F wave in the median nerve and bilateral ulnar nerve were normal bilaterally. The latency of F wave in the tibial nerve was prolonged bilaterally and the rate of occurrence was normal. The tibial nerve H reflex could not be induced bilaterally. The sensory potentials of both superficial peroneal nerves and both sural nerves could not be induced. The conduction velocity and amplitude of the common peroneal nerve and the tibial nerve were normal. No spontaneous potential was found in the right anterior tibial electromyography. The time limit was prolonged, the amplitude was increased, and the multiple waves were normal. No spontaneous potential was found in the left anterior tibial muscle, bilateral gastrocnemius muscle, or right medial femoral head muscle. The time limit, amplitude, and multiple waves were normal, and the maximum force was mixed. There was no spontaneous potential in the EMG of the right first interphalangeal muscle and the right biceps brachii. The time limit, amplitude, and multiple waves were normal, and the maximum force was mixed.

Mild to moderate multiple sensory neuron damage could be considered.

Imaging examinations

The imaging findings are presented in [Figure 1](#).

FINAL DIAGNOSIS

MM complicated with GBS.

TREATMENT

The patient was treated with high-dose IVIG (400 mg/kg/d for five days).

OUTCOME AND FOLLOW-UP

The symptoms improved partially, and CSF proteins returned to normal after four weeks. At the 6-mo follow-up, he had complete symptom remission.

DISCUSSION

GBS is one of the most common causes of acute flaccid paralysis, characterized by symmetrical limb weakness and sensory abnormalities. These usually begin at the distal end and appear in a symmetrical pattern, causing decreased or absent reflexes, and usually reach the peak within four weeks. The most common subtypes are acute inflammatory demyelinating polyneuropathy and acute motor axonal neuropathy, among other variants. The pathogenesis of GBS is mainly related to immune-mediated demyelination or axonal degeneration^[5,6]. The pathogenesis is still not clear.

In fact, PN can occur before and after bortezomib treatment. Our patient was

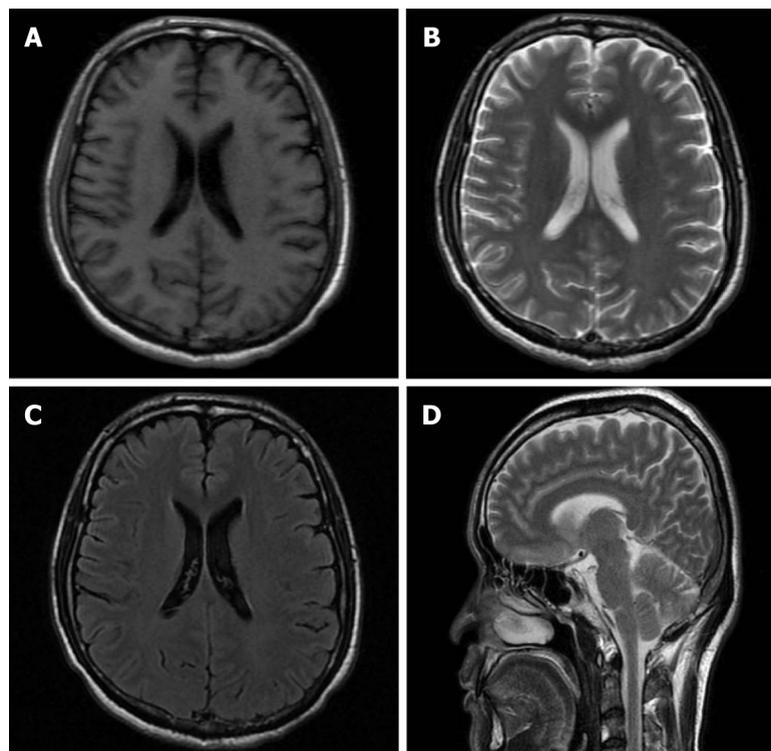


Figure 1 Magnetic resonance imaging revealing no intracranial mass. A: T1 weighted image; B: T2 weighted image; C: T2 fluid-attenuated inversion recovery; D: Sagittal T2 weighted image.

diagnosed with MM. Since PN ensued only after two cycles of bortezomib treatment, the following aspects were considered. First, polyneuropathy, organomegaly, endocrinopathy, monoclonal protein, and skin changes (POEMS) syndrome caused by MM is a possibility. In our case, after two cycles of chemotherapy, the burden of myeloma and bone marrow plasma cells were reduced, there were no skin lesions or hepatosplenomegaly, endocrine tests were negative, and M protein was not detected; therefore, POEMS syndrome was excluded. The second possibility is paraneoplastic syndrome caused by MM, which refers to the production of paraneoplastic antibodies in tumor patients, mediating the destruction of peripheral nerve lesions. Plasma cell tumors are closely associated with PN caused by paraneoplastic syndrome, especially in MM. Research shows that about 20% of patients with MM have neuropathy before treatment, which may be related to a primary disease^[2]. Paraneoplastic antibodies were negative and myeloma burden was relieved, so PN caused by paraneoplastic syndrome was not considered. Finally, it may be PN caused by bortezomib, or other neurological diseases. The patient developed numbness and pain at the end of the second course of treatment. No significant improvement was found after analgesic treatment and the patient continued to complain about intolerable pain and poor quality of life. A combination of elevated protein levels and normal cell counts in the CSF was indicative of GBS. Fortunately, the patient experienced relief after IVIG treatment.

In 1987, the first case of MM with kappa light chain combined with GBS was reported^[7]. In 2008, Iqbal *et al*^[8] also reported five patients with MM complicated by GBS, three of whom developed GBS after transplantation, one received dexamethasone combined with lenalidomide treatment, and one developed GBS without treatment. Subsequently, MM combined with GBS was reported after bone-marrow transplantation^[9,10].

Although bortezomib associated GBS is rare, MM with GBS after bortezomib therapy has been reported. Philippe Moreau *et al*^[11] in 2017 compared the safety and efficacy of venetoclax plus bortezomib and dexamethasone in relapsed/refractory MM. Before enrollment, a patient with MM complicated by GBS was considered, which was possibly related to bortezomib.

CONCLUSION

With the emergence of bortezomib, the prognosis of patients with MM has been

improved. In addition, more and more attention has been paid to adverse drug reactions and treatment. This case report suggests that the treatment of MM with bortezomib can simultaneously aggravate GBS.

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