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Guillain-Barré syndrome in patients with multiple myeloma: three cases report and literature review

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Abstract

Background Multiple myeloma (MM) with Guillain-Barré syndrome (GBS) is relatively rare, and the specific mechanism is still unclear. The previous infection, surgery, and medication use may have contributed to the occurrence of GBS. The use of bortezomib in patients with MM can easily lead to peripheral neuropathy, which is similar to the symptoms of GBS, making it challenging to diagnose GBS.

Cases presentation Three patients with IgA type MM experienced lower limb weakness during treatment. Combined with lumbar puncture, nerve conduction studies, and other tests, the diagnosis was confirmed as GBS. All three patients had a history of spinal surgery before the onset of GBS, and had been treated with bortezomib which induced peripheral neuropathy. Two of the three patients had a clear history of upper respiratory tract infection before the onset of GBS. After treatment with intravenous immunoglobulin, one patient died and two patients showed improvement in GBS symptoms.

Conclusion Patients with MM often have concurrent infections and spinal surgery, which may contribute to the occurrence of GBS. The symptoms of bortezomib-induce peripheral neuropathy overlap with those of GBS, which can easily lead to misdiagnosis or missed diagnosis of GBS. Timely lumbar puncture and nerve conduction studies may help to diagnose GBS and improve the prognosis.

Keywords Multiple myeloma, Guillain-Barré syndrome, Bortezomib, Case report, Neuropathy

Background

Multiple myeloma(MM) is a malignant clonal plasma cell disease characterized by infiltration of bone marrow plasma cells and an increase in peripheral blood monoclonal immunoglobulin, leading to complications such as bone destruction, hypercalcemia, renal insufficiency, and anemia, which seriously affect the quality of life and lifespan of patients [1]. With the emergence of new anti-myeloma drugs, including proteasome inhibitors, immune modulators, and CD38 monoclonal antibodies, the prognosis of MM patients has greatly improved, and many patients have the hope of surviving for more than 10 years [2]. However, patients with MM usually have

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decreased body resistance, and with continuous maintenance treatment, these patients still belong to a vulnerable population and are prone to various complications.

Guillain-Barré syndrome (GBS) is a type of immune-mediated acute inflammatory peripheral neuropathy, characterized by involvement of multiple nerve roots and peripheral nerve damage, with clinical symptoms typically peaking within two weeks [3]. Cerebrospinal fluid (CSF) analysis reveals a high protein content with a normal cell count, and nerve conduction studies(NCS)show peripheral nerve damage, while both tests can be normal in the early stages. High-dose intravenous immunoglobulins (IVIG) and plasma exchange are the main treatment methods [4, 5]. Although the overall prognosis of GBS is good, 3 -7% of patients still die, mainly from cardiovascular and respiratory complications such as cardiac arrest, autonomic dysfunction, respiratory failure, infection, pulmonary embolism, etc [6]. Moreover, a considerable number of patients will have sequelae, including neuropathic pain, weakness, and fatigue, which have a significant impact on daily activities and quality of life [7].

There are few reported cases of MM with GBS [8–11]. Patients with MM often have decreased body resistance and are prone to infection. Pathological fractures usually require surgical intervention. Also the therapeutic drugs used for MM, such as bortezomib, often cause peripheral neuropathy (PN). It is not yet clear whether these factors are correlated with the occurrence of GBS. This article retrospectively analyzes the diagnosis and treatment process of three patients with MM complicated with GBS, and considers the possible reasons for MM complicated with GBS based on literature review. Written informed consents were obtained from the patients or their first-degree relatives.

Cases presentation

Case 1

A 68 year old male patient experienced lower back pain after trauma in April 2020, which gradually worsened and restricted lower back movement. Lumbar magnetic resonance imaging (MRI) revealed a compression fracture of the second vertebra of the lumbar spine. The laboratory tests were as follows: IgA λ type by M protein electrophoresis, hemoglobin(HB) 87 g/L, albumin 26 g/L, β2-microglobulin(β2-MG) 7.5 mg/L, plasma cells accounting for 44% by bone marrow cytology examination. Percutaneous vertebroplasty (PVP) of second lumbar vertebrae was performed in July 2021, and postoperative pathological reported plasma cell tumor. Based on M-protein electrophoresis, bone marrow cytology examination and biopsy pathology, the patient was diagnosed with stage III MM (IgA λ type) according to the International Staging System(ISS). The patient was treated with VRD (bortezomib, lenalidomide and dexamethasone) regimen chemotherapy 4 times, and the subsequent efficacy evaluation was complete remission. Then the patient received autologous hematopoietic stem cell transplantation treatment in November 2021, followed by maintenance therapy with lenalidomide. After treatment, the patient's lower back pain symptoms were significantly relieved and he was able to live normally.

In January 2023, the patient experienced symptoms of upper respiratory tract infection, and in February 2023, the patient had limb weakness and numbness, making him impossible to walk. One week after the onset of illness, the patient was admitted to the hospital. After admission, the neurological examination revealed the presence of peripheral facial paralysis and quadriplegia in the patient. The muscle tone in both limbs was normal. The proximal Medical Research Council (MRC) muscular strength of both upper limbs was grade 4/5, and the distal MRC muscle strength was grade 3/5. The proximal MRC muscle strength of both lower limbs was grade 2/5, and the distal MRC muscle strength was grade 2^{-}/5. The pinprick sensation in both upper limbs was symmetrical without decrease, while the sensation below the ankles in both lower limbs was decreased. The symptoms were possibly a result of GBS. So NCS and lumbar puncture examination were performed. NCS indicated slow motor conduction velocity in bilateral peroneal and right tibial nerves, and prolonged latencies in bilateral peroneal nerves, suggesting demyelination. F wave latency of left median nerve was prolonged (Table 1). On day 10 after the onset, the lumbar puncture examination was operated and showed albuminocytological dissociation, and no MM cells was found in CSF smear (Table 2). The cranial MRI revealed lacunar cerebral infarction and senile brain changes, which could not account for the patient's symptoms and signs. M protein electrophoresis test was negative, and the serum free light chain test was within the normal range, indicating that the status of MM was in complete remission.

Based on CSF examination, NCS, and physical examination of the nervous system, the patient was diagnosed with GBS. On day 11, the patient received IVIG (0.4 g/kg/d for 5 days) therapy, but the symptoms of limb weakness did not improve. On day 14, bladder dysfunction occurred, which required catheterization. One day 17, the patient developed fever, weakened cough reflex, and decreased blood oxygen saturation. Bedside chest radiography indicated white lung. The patient was transferred to the intensive care unit and received mechanical ventilation treatment. The patient died of lung infection and respiratory failure 24 days after onset.

Case 2

A 70-year-old female patient experienced numbness and weakness in both lower limbs, unable to walk,

Table 1 Selected NCS findings of three cases

Nerve/Sites	Case 1			Case 2			Case 3		
	Latency (ms)	AMP. (mv)	Velocity (m/s)	Latency (ms)	Amp. (mv)	Velocity (m/s)	Latency (ms)	Amp. (mv)	Velocity (m/s)
Motor NCS									
Left Ulnar									
Wrist	2.85	7.1		1.64	7.6		3.29	3.0	
Elbow		8.1	51		4.4	49.3		2.6	37.5
Left Median									
Wrist	3.3	7.3		6.13	1.63		3.29	4.1	
ELbow		6.3	49		0.72	48		3.1	39.5
Right Tibial									
Ankle	3.7	3.4		4.81	5.3		4.04	3.8	
Popliteal Fossa		4.6	38		4.5	48.5		3.0	33.1
Left Peroneal									
Ankle	15	4.2		4.99	0.43		4.36	0.48	
Below Knee		3.4	29.6		0.53	29.5		0.22	35.6
Right Peroneal									
Ankle	6.9	5.2		3.85	0.4		4.36	0.36	
Below Knee		4.3	25.1		0.33	28.6		0.23	37.6
Sensory NCS									
Nerve/Sites (μV) (μV)									
Right sural Ankle-Calf	2.9	3.5	53,2	1.72	2.1	58.1	No response		
Left sural Ankle-Calf	2.1	4,1	48.5	2.15	4.2	51.2	No response		
	Case 1			Case 2			Case 3		
F Latency (ms)									
Left Median	48.5			39.5			33.8		
Left Ulnar				41.3			38.6		

Table 2 The results of analysis of CSF samples obtained by lumbar puncture

Characteristics	Reference range	Case 1	Case 2	Case 3
WBC count	0–5/μl	4	2	1
Glucose	2.2-3.9mmol/L	3.6	3.48	3.3
Total protein	150-450 mg/L	1301	1082	1403
Chlorides	120–132 mmol/L	121	118	116
LDH	20-25U/L	25	40	1
MM cells by CSF smear		negative	negative	negative

WBC, white blood cell; *LDH*, Lactic dehydrogenase; *MM*, multiple myeloma; *CSF*, cerebrospinal fluid

accompanied by pain in the lower back and ribs in December 2023. Lumbar MRI showed that the space occupying lesion at 5th thoracic vertebra was locally protruding into the spinal canal and compressing the thoracic spinal cord. In January 2024, she underwent posterior thoracic tumor resection, bone graft fusion, and internal fixation. Postoperative pathology revealed plasma cell tumor. Laboratory examination showed the following: IgA κ type by M protein electrophoresis, β2-MG 9.31 mg/L, 42% plasma cells showed by bone marrow cytology. The patient was diagnosed with multiple myeloma (IgA κ type, ISS stage III). After 3 times of chemotherapy with PLD (bortezomib, liposomal doxorubicin, and dexamethasone) regimen, the patient's bone pain symptoms were significantly relieved, and the lower limb weakness symptoms improved. The use of a walking aid allowed the patient to walk. After re-examination, the M protein electrophoresis was negative and the serum free light chain was within the normal range, indicating that the patient had achieved complete remission.

In early March 2024, the patient experienced gingival pain and oral ulcers. On March 24, 2024, the patient began to experience weakness in the lower limbs accompanied by pain, and was unable to stand independently. On March 31, 2024, the patient was admitted to the hospital. After admission, a neurological physical examination was conducted. The muscle tone of the limbs was normal. The muscle strength of both upper limbs was normal. The muscle strength of both lower limbs decreased symmetrically, with grade 4/5 (MRC)proximally and grade 3/5(MRC) distally. Tendon reflexes in both lower limbs could not be elicited. On April 1, 2024

(the 8th day after onset), a lumbar puncture examination was performed, and the CSF results were shown on Table 2. The NCS examination indicated decreased motor nerve conduction velocity in distal ends of both lower limbs and the left upper limb, with prolonged latencies in light median nerve, suggesting demyelination. The significant decrease in the amplitude of motor conduction in bilateral peroneal nerves indicated the presence of axonal lesions. The latency of F-waves in the left upper limb was prolonged. (Table 1). No space-occupying lesions were found on the MRI of the thoracic and lumbar spine.

The patient was diagnosed with GBS, and was treated with IVIG (0.4 g/kg/d for 5 days) on day 9. On day 14, the patient's pain in both lower limbs improved, but there was no significant improvement in lower limb muscle strength, with MRC muscle strength of 3/5 proximally and 2/5 distally. The patient was discharged to a rehabilitation hospital for further treatment. We followed up the patient by phone one week after discharge and were told that she could walk with a walker.

Case 3

A 70-year-old female patient developed bilateral low back pain without apparent cause in February 2024, which progressively worsened, making her difficult to turn over and gradually causing numbness in both feet. Lumbar MRI showed first lumbar vertebral (L1) compression fractures. Laboratory examination showed HB 78 g/L, β2-MG 3.51 mg/L, IgA λ type by M protein electrophoresis, and 40% plasma cells found in bone marrow cytology. The patient was diagnosed with multiple myeloma (IgA λ type, ISS stage II). In May 2024, she underwent L1 vertebral PVP surgery and began chemotherapy with VRD regimen. Then the bone pain symptoms were significantly alleviated. After 3 courses of VRD regimen, the M protein was negative.

In August 2024, the patient experienced numbness in all four limbs, particularly in both lower limbs, which was considered to be bortezomib-induced peripheral neuropathy(BiPN).The treatment plan was adjusted to RD (lenalidomide and dexamethasone) regimen, and gabapentin was used to improve the PN symptoms. The patient's numbness in both lower limbs gradually worsened, and she experienced fatigue and was unable to walk. On September 11, 2024, the patient was admitted and the neurological examination was conducted. The muscle strength of both upper limbs was normal. There was a symmetrical decrease in muscle strength of both lower limbs, with grade 4/5 (MRC) proximally and grade 3/5 (MRC) distally. The acupuncture sensation below the middle of both lower limbs was decreased. Tendon reflexes in both lower limbs were absent.

BiPN could not account for the weakness symptoms in both lower limbs of the patient. The patient might suffer from other polyneuropathies, such as GBS. Lumbar puncture was performed and the results of CSF were shown in Table 2. Meanwhile, metagenomic next-generation sequencing (mNGS) technology was used to detect pathogenic microbial DNA in CSF, and no pathogenic sequences were detected. NCS indicated a decrease in motor nerve conduction velocity in the left upper limb and both lower limbs, with prolonged latencies in bilateral peroneal nerves, suggesting demyelination. Motor conduction amplitude in distal lower limbs significantly decreased, indicating the presence of axonal lesions. The latency of F-wave in the left upper limb was prolonged (Table 1). Subsequently, IVIG (0.4 g/kg/d for 5 days) was administered for treatment. After a follow-up of one month, the patient's symptoms of weakness in both lower limbs improved, and she could walk with the help of a walker. However, she still had symptoms of numbness in her lower limbs, which might be due to BiPN.

Discussion and conclusions

The causes and mechanisms of GBS are still unclear, and may be related to previous infections, surgery, vaccination, and immune checkpoint inhibitors therapy. Whether the combination of MM and GBS is an occasional event or there is some connection between them, there is currently no large-scale clinical data to support it, only scattered case reports. Currently, based on the data of 3 patients and literature, we are thinking about the possible causes of MM combined with GBS. The clinical characteristics of the three patients are shown in Table 3.

Infection is the most common predisposing factor for the occurrence of GBS. Two-thirds of adult patients with GBS have premonitory symptoms of respiratory or gastrointestinal infection within 4 weeks before the onset of weakness symptoms, with upper respiratory tract infection being the most common precursor event worldwide [12]. The reported pathogens of GBS precursor infection include Campylobacter jejuni, Mycoplasma pneumoniae, Haemophilus influenzae, Japanese encephalitis virus, Epstein-Barr virus, herpes simplex virus, cytomegalovirus, and Zika virus [13–15]. COVID-19 infection was also associated with increased risk of GBS [16]. Molecular mimicry between microorganisms and neural antigens may be the most well established mechanism for the occurrence and development of GBS [3]. Two of the three patients had a history of significant pre-infection. It is noteworthy that the three patients we reported were all IgA type MM. IgA is the main component of the body's mucosal immune defense, and the loss of IgA function may lead to more susceptibility to respiratory and gastrointestinal infections in MM, which may promote the occurrence of GBS [17]. However, there are also case reports of light chain multiple myeloma with GBS [10, 11].

Table 3 The clinical characteristics of the three patients of MM with GBS

	Sex/age	Type of M protein	treatment regimens of MM	Interval from diagnosis of MM to GBS	Antecedent infections	Antecedent spinal surgery	Treatment of GBS	Outcome of GBS
Case 1	M/68	IgA λ	VRD, ASCT and lenalidomide	22 m	Yes	Yes	IVIG	Die
Case 2	F/70	IgA κ	PLD	4 m	Yes	Yes	IVIG	Independent transfer from bed to chair
Case 3	F/70	IgA λ	VRD, RD	6 m	No	Yes	IVIG	Some residual numbness of legs

MM, multiple myeloma; *GBS*, Guillain-Barré syndrome; *VRD*, bortezomib, lenalidomide and dexamethasone; *ASCT*, autologous stem cell transplantation; *PLD*, bortezomib, liposomal doxorubicin, and dexamethasone; *RD*, lenalidomide and dexamethasone; *IVIG*, intravenous immunoglobulins

Several studies have shown a link between surgery and GBS [18–20]. A retrospective analysis in Switzerland in 2012 showed that the incidence rate of GBS after surgery increased significantly, with a relative risk of 13.1 times higher than the baseline incidence rate [18]. A national epidemiological study in France in 2018 showed that 5% of patients developed GBS after surgery, with orthopedic surgery and gastrointestinal surgery significantly associated with GBS [20]. GBS usually occurs within 6 weeks after surgery. The pathogenesis of surgery-related GBS is still unclear, and may be related to immune dysfunction or secondary infection caused by surgery. Patients with multiple myeloma often have vertebral compression fractures, which require vertebral surgery. Wei-Cheng Tu et al. [21] summarized 16 case reports of GBS after spinal surgery, with short-term GBS symptoms occurring immediately after surgery and long-term GBS symptoms occurring 17 days after surgery. Ten of the 16 patients developed respiratory failure. Patients with spinal cord injury often experience symptoms such as weakness and numbness in the lower limbs, which overlap with the symptoms of GBS, making it easy to misdiagnose or miss the diagnosis of GBS. The three patients we reported all had a history of spinal surgery, but the interval between surgery and GBS was relatively long, which may not be related to each other.

Bortezomib is one of the most important drugs for the treatment of MM, and PN is its main side effect [22]. VRD regimen is the most commonly used induction therapy for multiple myeloma. With this regimen, the incidence of PN is as high as 80%, with a grade ≥ 3 PN incidence of 33% [23]. Subcutaneous administration instead of intravenous administration does not affect the efficacy, but can significantly reduce the incidence of PN [24]. Typical manifestations of BiPN include neuropathic pain, numbness, and sensory abnormalities in the distal limbs, with a small number of patients experiencing motor dysfunction, manifested as muscle weakness, muscle atrophy, and unilateral or bilateral foot drop [25]. There are overlaps and differences in the clinical manifestations of BiPN and GBS. The typical clinical manifestation of GBS is rapidly progressive bilateral muscle weakness, with some patients experiencing cranial nerve involvement, nerve root pain, and muscle pain [26]. Patients with severe GBS may experience respiratory muscle paralysis leading to respiratory failure, and autonomic nerve damage can cause fatal arrhythmia and hypotension [6]. There are case reports of MM patients who developed GBS after using bortezomib, which is considered to be caused by bortezomib [10, 11]. The three patients we reported had all been treated with bortezomib before GBS, and had experienced varying degrees of PN, followed by lower limb weakness symptoms. After undergoing lumbar puncture and NCS, they were diagnosed with GBS.

Table 4 Comparisons between BiPN and GBS

	BiPN	GBS
Antecedent events	Bortezomib therapy	Infections, vaccinations, immune checkpoint inhibitor therapy and surgery
Time of onset	Typically within the first courses of bortezomib [34].	Within 4 weeks of antecedent infections,6 weeks of surgery
Clinical features	Mild to moderate distal sensory loss; Mild to very severe pain, mainly at fingertips and toes; Mild motor weakness in distal muscles of the lower limbs; Rare autonomic dysfunction	Bilateral and flaccid weakness of limbs; Decreased or absent deep tendon reflexes; Mild sensory symptoms or signs; Cranial nerve involvement; Autonomic dysfunction
CSF analysis	Normal	CSF white cell count < 50 /μl (usually < 10); CSF protein raised (after week 1)
NCS	Mild distal slowing of sensory and motor conduction velocities and increase in distal motor latencies.	Slow nerve conduction velocities, prolonged distal latencies, and temporal dispersion in AIDP; Decreased CMAP amplitude in AMAN
Locations of lesion	Schwann cells and dorsal root ganglion neurons [35]	Axonal injury in AMAN Inflammatory infiltrates and demyelination in AIDP
Management	Prevention first(subcutaneous injection, dose reduction, prolonged administration of bortezomib); Medical therapy(Opioids, Tricyclic antidepressants, Anticonvulsants, SNRIs, NSAIDs, Vitamins)	IVIG; plasma exchange
Outcome	Improve or completely resolve in most patients after a median interval of 3 months after discontinuation of bortezomib treatment	Following immunotherapy, most patients have a good recovery, with 81% able to walk independently at 12 months
Mortality	Cases report [36]	3-7% [37]

BIPN, bortezomib-induced peripheral neuropathy; *GBS*, Guillain-Barré syndrome; *CSF* cerebrospinal fluid; *NCS*, nerve conduction study; *IVIG*, intravenous immunoglobulins; *CMAP* compound muscle action potential; *AIDP*, acute inflammatory demyelinating neuropathy; *AMAN*, acute axonal motor neuropathy; *SNRIs*, serotonin norepinephrine reuptake inhibitors; *NSAIDs*, nonsteroidal anti-inflammatory drugs

The relationship between bortezomib and GBS remains unclear.

10-20% of multiple myeloma patients coexist with light-chain (AL) amyloidosis, and one-quarter of patients with AL amyloidosis may experience neuropathy [27]. For MM patients who exhibit neurological symptoms, it is necessary to differentiate whether amyloidosis is present. In addition to peripheral neuropathy, the autonomic nervous system is often affected, manifested as secretory diarrhea, orthostatic hypotension, erectile dysfunction, etc. NCS and pathological examination often indicate axonal lesions [28]. Unlike GBS, patients with AL amyloidosis often accompanied by heart and kidney disease. Subcutaneous fat biopsy, bone marrow biopsy, or biopsy of diseased organs can determine the presence of AL amyloidosis. For patients with multiple myeloma complicated with amyloidosis, the symptoms of amyloidosis usually alleviate after controlling the myeloma. The three patients we reported did not show amyloid deposition in bone marrow pathology at the onset of the disease, and when neurological symptoms appeared, all patients were in remission status of multiple myeloma, so neurological symptoms caused by amyloidosis were not considered.

POEMS syndrome(polyneuropathy, organomegaly, endocrinopathy, M-protein, and skin changes)is rare type of plasma cell diseases that can also cause peripheral neuropathy, leading to symptoms such as limb numbness, pain, and weakness. Occasionally, there are case reports of multiple myeloma accompanied by POEMS syndrome [29, 30]. Unlike the acute onset and monophasic course of GBS, the neurological symptoms of POEMS syndrome often persist for a long time and gradually worsen, resembling chronic inflammatory demyelinating polyneuropathy (CIDP) [31]. NCS show that demyelination is predominant in the nerve trunk rather than the distal nerve terminals and axonal loss is predominant in the lower limb nerves [32]. Skin lesions (such as hemangioma, hypertrichosis, skin pigmentation, white nails, etc.) and thrombocytosis suggest the possible presence of POEMS syndrome. Further vascular endothelial growth factor (VEGF) testing, bone marrow biopsy, M protein detection, and imaging studies are helpful in confirming the diagnosis of POEMS syndrome. Routine VEGF testing for all patients with CIDP may reduce misdiagnosis, mortality, and treatment costs associated with POEMS syndrome [33].

The cause of multiple myeloma with GBS is unclear, and previous infections, spinal surgery, and medication use may be involved in the occurrence of GBS. Vertebral lesions and drugs such as bortezomib may cause limb numbness and fatigue, which can be easily confused with the clinical manifestations of GBS and require vigilance from clinicians. The identification of BiPN and GBS is shown in Table 4. Amyloidosis and POEMS syndrome can also cause neuropathy, necessitating careful differentiation. For MM patients, when they suddenly experience unexplained limb weakness, we need to be aware of GBS and promptly perform lumbar puncture and NCS to help differentiate it. Early diagnosis and treatment of GBS may reduce the occurrence of emergency situations and improve prognosis [38].

Abbreviations

MM	Multiple myeloma
GBS	Guillain-Barré syndrome
CSF	Cerebrospinal fluid
NCS	Nerve conduction studies
MRC	Medical research council
IVIG	Intravenous immunoglobulins
PN	Peripheral neuropathy
MRI	Magnetic resonance imaging
HB	Hemoglobin
β2-MG	β2-microglobulin
PVP	Percutaneous vertebroplasty
ISS	International staging system(ISS)
VRD	Bortezomib, lenalidomide and dexamethasone
PLD	Bortezomib, liposomal doxorubicin, and dexamethasone
BiPN	Bortezomib-induced peripheral neuropathy
RD	Lenalidomide and dexamethasone
VEGF	Vascular endothelial growth factor
CIDP	Chronic inflammatory demyelinating polyneuropathy
POEMS	Polyneuropathy, organomegaly, endocrinopathy, M-protein, and skin changes

Author contributions

Zhichao Li and Fang Huang contributed to the conception and design of the manuscript. Zhichao Li collected the data and drafted the manuscript. Siguo Hao reviewed and modified the manuscript. All authors contributed to manuscript revision and read and approved the final submitted version.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Approval for this study was provided by Ethics Committee of Xinhua Hospital Affiliated to Shanghai Jiaotong University School of Medicine.

Consent for publication

Written informed consents for publication of the clinical details were obtained from the patients or their first-degree relatives.

Competing interests

The authors declare no competing interests.

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References

1. Malard F, Neri P, Bahlis NJ, et al. Multiple myeloma [J]. Nat Rev Dis Primers. 2024;10(1):45.
2. Rodriguez-Otero P, Paiva B, San-Miguel JF. Roadmap to cure multiple myeloma [J]. Cancer Treat Rev. 2021;100:102284.
3. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barre syndrome [J]. Lancet. 2021;397(10280):1214–28.

4. Hughes RA, Swan AV, Van Doorn PA. Intravenous immunoglobulin for Guillain-Barre syndrome [J]. Cochrane Database Syst Rev. 2014;2014(9):CD002063.
5. Chevret S, Hughes RA, Annane D. Plasma exchange for Guillain-Barre syndrome [J]. Cochrane Database Syst Rev. 2017;2(2):CD001798.
6. Van Den Berg B, Walgaard C, Drenthen J, et al. Guillain-Barre syndrome: pathogenesis, diagnosis, treatment and prognosis [J]. Nat Rev Neurol. 2014;10(8):469–82.
7. Darweesh SK, Polinder S, Mulder MJ, et al. Health-related quality of life in Guillain-Barre syndrome patients: a systematic review [J]. J Peripher Nerv Syst. 2014;19(1):24–35.
8. Lu C, Hu H, Ye Y et al. Guillain-Barr Syndrome caused by Cytomegalovirus after multiple myeloma treatment [J]. Clin Lab, 2024;70(6).
9. Krecak I, Gveric-Krecak V. Refractory Guillain-Barre Syndrome in a patient with asymptomatic multiple myeloma successfully treated with low-dose rituximab [J]. Acta Clin Croat. 2023;62(2):382–6.
10. Dai X, Sun X, Ni H, et al. Guillain Barre Syndrome in a multiple myeloma patient after the first course of bortezomib therapy: a case report [J]. Oncol Lett. 2015;10(5):3064–6.
11. Xu YL, Zhao WH, Tang ZY, et al. Guillain-Barre syndrome in a patient with multiple myeloma after bortezomib therapy: a case report [J]. World J Clin Cases. 2019;7(18):2905–9.
12. Jacobs BC, Rothbarth PH, Van Der Meche FG, et al. The spectrum of antecedent infections in Guillain-Barre syndrome: a case-control study [J]. Neurology. 1998;51(4):1110–5.
13. Cao-Lormeau VM, Blake A, Mons S, et al. Guillain-Barre Syndrome outbreak associated with Zika virus infection in French polynesia: a case-control study [J]. Lancet. 2016;387(10027):1531–9.
14. Dalton HR, Kamar N, Van Eijk JJ, et al. Hepatitis E virus and neurological injury [J]. Nat Rev Neurol. 2016;12(2):77–85.
15. Meyer Sauteur PM, Huizinga R, Tio-Gillen AP, et al. Mycoplasma pneumoniae triggering the Guillain-Barre syndrome: a case-control study [J]. Ann Neurol. 2016;80(4):566–80.
16. Bishara H, Arbel A, Barnett-Griness O, et al. Association between Guillain-Barre Syndrome and COVID-19 infection and vaccination: a Population-based nested case-control study [J]. Neurology. 2023;101(20):e2035–42.
17. Mantis NJ. Rediscovering IgA [J]. Mucosal Immunol. 2011;4(6):588–9.
18. Gensicke H, Datta AN, Dill P, et al. Increased incidence of Guillain-Barre syndrome after surgery [J]. Eur J Neurol. 2012;19(9):1239–44.
19. Gong Q, Liu S, Liu Y, et al. Guillain-Barre syndrome triggered by surgery in a Chinese population: a multicenter retrospective study [J]. BMC Neurol. 2021;21(1):40.
20. Rudant J, Dupont A, Mikaeloff Y, et al. Surgery and risk of Guillain-Barre syndrome: a French nationwide epidemiologic study [J]. Neurology. 2018;91(13):e1220–7.
21. Tu WC, Chang ST, Huang CH et al. Guillain-Barre syndrome with respiratory failure following spine surgery for Incomplete Cervical Cord Injury: a Case Report and Literature Review [J]. Med (Kaunas), 2022;58(8).
22. Scott K, Hayden PJ, Will A, et al. Bortezomib for the treatment of multiple myeloma [J]. Cochrane Database Syst Rev. 2016;4(4):CD010816.
23. Durie BGM, Hoering A, Abidi MH, et al. Bortezomib with lenalidomide and dexamethasone versus lenalidomide and dexamethasone alone in patients with newly diagnosed myeloma without intent for immediate autologous stem-cell transplant (SWOG S0777): a randomised, open-label, phase 3 trial [J]. Lancet. 2017;389(10068):519–27.
24. Moreau P, Pylypenko H, Grosicki S, et al. Subcutaneous versus intravenous administration of bortezomib in patients with relapsed multiple myeloma: a randomised, phase 3, non-inferiority study [J]. Lancet Oncol. 2011;12(5):431–40.
25. Vaxman I, Mauerman ML, Gatt ML, et al. Foot drop in patients treated with bortezomib - a case series and review of the literature [J]. Leuk Lymphoma. 2022;63(3):722–8.
26. Van Doorn PA. Diagnosis, treatment and prognosis of Guillain-Barre syndrome (GBS) [J]. Presse Med. 2013;42(6 Pt 2):e193–201.
27. Muchtar E, Gertz MA, Kyle RA, et al. A modern primer on light chain amyloidosis in 592 patients with Mass Spectrometry-verified typing [J]. Mayo Clin Proc. 2019;94(3):472–83.
28. Chompoopong P, Mauermann ML, Siddiqi H, et al. Amyloid neuropathy: from pathophysiology to treatment in Light-Chain Amyloidosis and Hereditary Transthyretin amyloidosis [J]. Ann Neurol. 2024;96(3):423–40.
29. Brioli A, Wyrwa A, Ruddel U, et al. Mutations in the plasma cell clone identify mechanism of polyneuropathy in a case of POEMS syndrome associated with Castleman disease and multiple myeloma [J]. Ann Hematol. 2023;102(1):239–42.
30. Sano M, Iseki T, Sasaki M, et al. Improvement in the symptoms and VEGF levels after resection of an Extrame Dullary spinal tumor and additional chemotherapy in a patient with multiple myeloma complicated with POEMS syndrome [J]. Intern Med. 2021;60(22):3625–30.
31. Dispenzieri A. POEMS syndrome: update on diagnosis, risk-stratification, and management [J]. Am J Hematol. 2023;98(12):1934–50.
32. Nasu S, Misawa S, Sekiguchi Y, et al. Different neurological and physiological profiles in POEMS syndrome and chronic inflammatory demyelinating polyneuropathy [J]. J Neurol Neurosurg Psychiatry. 2012;83(5):476–9.
33. Marsh ES, Keddie S, Terris-Prestholt F, et al. Early VEGF testing in inflammatory neuropathy avoids POEMS syndrome misdiagnosis and associated costs [J]. J Neurol Neurosurg Psychiatry. 2021;92(2):172–6.
34. Staff NP, Grisold A, Grisold W, et al. Chemotherapy-induced peripheral neuropathy: a current review [J]. Ann Neurol. 2017;81(6):772–81.
35. Meregalli C, Chiorazzi A, Carozzi VA, et al. Evaluation of tubulin polymerization and chronic inhibition of proteasome as citotoxicity mechanisms in bortezomib-induced peripheral neuropathy [J]. Cell Cycle. 2014;13(4):612–21.
36. Gupta S, Pagliuca A, Devereux S, et al. Life-threatening motor neurotoxicity in association with bortezomib [J]. Haematologica. 2006;91(7):1001.
37. Islam Z, Papri N, Ara G, et al. Risk factors for respiratory failure in Guillain-Barre syndrome in Bangladesh: a prospective study [J]. Ann Clin Transl Neurol. 2019;6(2):324–32.
38. Ho TW, Mishu B, Li CY et al. Guillain-Barre syndrome in northern China. Relationship to Campylobacter jejuni infection and anti-glycolipid antibodies [J]. Brain, 1995;118(Pt 3):597–605.

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