Systematic Review/Meta-analysis

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Guillain–Barré syndrome following varicella–zoster virus infection: a case report and systematic review

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Background: Guillain–Barré syndrome (GBS) is an acute inflammatory disease of the peripheral nervous system, rarely following Varicella–zoster virus (VZV) infection. The authors aimed to review all cases in the English literature of GBS that occurred after primary VZV infection to investigate the clinical features, diagnostic workup, treatment, and outcome of patients with GBS following VZV.

Methods: PubMed, Scopus, and Embase are systematically searched from their inception to 9 May 2022 to collect all cases of GBS following varicella–zoster infection. Patients with GBS following VZV reactivation were excluded.

Results: Among the 29 patients, the age of presentation ranged from 1.5 to 70 years with a median of 37, with a yield for males (81.5%). Most of the patients presented with sensory-motor symptoms (65.4%) and suffered from tetraparesis (81.5%). Cranial nerve palsy was present in (84%) of patients, and the seventh cranial nerve was the most commonly affected nerve (75%). Lumbar puncture showed albuminocytological dissociation in (80%) of patients. The dominant nerve conduction study subtype was acute inflammatory demyelinating polyneuropathy (65.3%). in addition, the magnetic resonance imaging showed pathological findings in only (47.5%) of the patients. Intravenous immunoglobulin is now the drug of choice for all cases of GBS following VZV infection.

Conclusion: GBS is a rare neurological complication of primary infection with VZV. However, the authors should suspect this syndrome when a patient develops ascending weakness, regardless of the absence of areflexia and albuminocytological dissociation. Drug therapy with IIVIg ensures a gradual improvement for the patient over a period of weeks to several months.

Keywords: chickenpox, guillain–barre syndrome, varicella–zoster virus

Introduction

Guillain–Barré syndrome (GBS) is a rare acute inflammatory disease of peripheral nerves and nerve roots that usually occurs as a post-infective immune-mediated phenomenon. The disease manifests as ascending muscle weakness that starts from the lower extremities and moves to the upper extremities, hyporeflexia, and possible but less prominent sensory symptoms[1]. GBS usually follows a gastrointestinal or upper respiratory infection. It has been associated with Campylobacter jejuni, Mycoplasma pneumonia, varicella–zoster virus (VZV), cytomegalovirus, Epstein-Barr virus, HIV, or even COVID-19 infections[2].

Although VZV may trigger many neurological complications such as meningoencephalitis, cerebellitis, and myelopathy, the virus may also rarely trigger GBS.

Several studies have found that GBS following varicella infection is reported more commonly after secondary reactivation (herpes zoster) rather than primary varicella infection (chickenpox)[3]. There are many cases in the literature that reported Guillain-Barré that occurred following VZV reactivation, On the other hand, few cases showed the disease as a complication of a primary VZV infection. Welch and colleagues described the first case in the UK in 1960[4].

Here we report on a rare case of GBS after 2 weeks of Varicella–zoster infection in a 4-year-old male and review systemically all cases up to May 2022 without age restrictions.

HIGHLIGHTS

- Accepted studies are case reports or case series.
- That discussed Guillain–Barre syndrome after primary varicella–zoster virus infection.
- They showed that the presence of deep tendon reflexes does not rule out the diagnosis.
- The absence of albuminocytological dissociation does not rule out the diagnosis.
- Drug therapy ensures a gradual improvement over a period of weeks to several months.

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Case presentation

A 4-year-old male was admitted to the paediatric department of our hospital, complaining of ataxia and weakness in the lower extremities, which led to gait disturbance. The child was complaining of a typical rash of chickenpox 15 days earlier. Two days after admission, the weakness extended to the upper extremities. The parents ensure that the child has fully received his vaccinations. On physical examination, the child showed a low pulse rate, hypotension, and a neurological examination of deep tendon reflexes showed hyporeflexia. Then, a lumbar puncture was performed, showing normal cerebrospinal fluid (CSF) protein and cell values. The contrast MRI for the brain and spinal cord was also normal. We then performed an electroneurogram that showed peripheral neuropathy and axial radiculopathy, as well as nerve conduction studies (NCS), which showed acute motor axonal neuropathy (AMAN).

He was diagnosed with post-varicella infection pure motor GBS despite normal CSF protein level, absence of albumino-cytological dissociation, and normal findings of MRI, due to neurological clinical features and NCS consistent with GBS. We had four differential diagnoses including post-varicella cerebellitis, aseptic meningitis, transverse myelitis, and GBSGBS. Based on clinical findings, we ruled out post-varicella cerebellitis which is the most common complication of Varicella infection because it is benign, self-limiting, and does not progress to the worst. Transverse myelitis and aseptic meningitis were also ruled out because other investigations were normal.

The typical treatment for post-varicella Guillain–Barre is intravenous immunoglobulin (IVIG), but because it was not available, we started with IV prednisolone 30 mg\kg for 3 days. He showed a positive response to this treatment and gradually improved. The child was discharged from the hospital after 15 days of admission, physical therapy was performed until he fully recovered. Two weeks after discharge, the patient was able to walk alone without any support.

Table 1

The characteristics of included studies

Last name	Year	Journal	Country	No. reported cases	No. cases eligible for inclusion	Follow-up	The NIH quality assessment tool
Welch *et al.*[17]	1962	Archives of Diseases in Childhood	UK	1	1	4 months	Fair
Leeming *et al.*[18]	1976	The Journal of laryngology and otology	UK	2	1	N/R	Poor
Twomey *et al.*[19]	1981	Postgraduate Medical Journal	UK	1	1	4 months	Fair
Arruda *et al.*[20]	1987	Arquivos de Neuro-Psiquiatria	Brazil	1	1	No follow-up	Poor
Sanders *et al.*[21]	1987	Journal of Neurology	N/R	2	1	3 months	High
Ormerod *et al.*[22]	1993	European Neurology	UK	2	1	1 year	High
Da Rosa-Santos *et al.*[23]	1996	International Journal of Dermatology	Brazil	1	1	4 months	Fair
Sabogal *et al.*[24]	1997	Pediatrics in Review	USA	3	1	N/R	Poor
Yoshikawa *et al.*[25]	2000	Archives of Diseases in Childhood	Japan	1	1	N/R	Fair
Hamad *et al.*[26]	2002	Neurosciences	Qatar	2	2	Case 1: 3 months Case 2: 2 monnths	Fair
Inan *et al.*[27]	2007	Journal of Pediatric Neurology	Turkey	1	1	2 months	High
Cresswell *et al.*[28]	2009	International Journal of Infectious Diseases	UK	1	1	2 months	High
Munoz-Sellart *et al.*[11]	2009	Enfermedades Infecciosas Y Microbiologia clinnica	Spain	2	1	6 months	High
Modi *et al.*[29]	2010	Taiwanese Journal of Obstetrics & Gynecology	India	1	1	1 months	High
Assi *et al.*[30]	2010	Transplant infectious diseases	USA	1	1	N/R	Fair
Paul *et al.*[31]	2010	Journal of Neurosciences in Rural Practice	India	3	1	N/R	High
Cokyaman et[32]	2014	Journal of Infection and Public Health	Turkey	1	1	3 months	High
Tatarelli *et al.*[33]	2015	International Journal of Neuroscience	Italy	3	3	N/R	High
Caramăngiu *et al.*[34]	2016	BMC Infectious Diseases	Romania	1	1	N/R	Fair
Bhatt *et al.*[35]	2019	Kurume Medical Journal	India	1	1	N/R	High
Xifaras *et al.*[36]	2019	Journal of the Neurological Sciences	Greece	1	1	N/R	Fair
Kofahi *et al.*[37]	2020	International Medical Case Reports Journal	Jordan	1	1	6 months	High
Balamurugesan *et al.*[38]	2021	Cureus	India	1	1	6 week	High
Arora *et al.*[39]	2021	Journal of Child Neurology	Greece	1	1	1 months	High
Spyromitrou-Xioufi *et al.*[40]	2021	Infectious Diseases in Clinical Practice	India	1	1	4 months	High
Nerabani *et al.*	2022	Annals of Medicine and Surgery	Syria	1	1	2 week	High

N/R, not reported, NIH, National Institutes of Health.

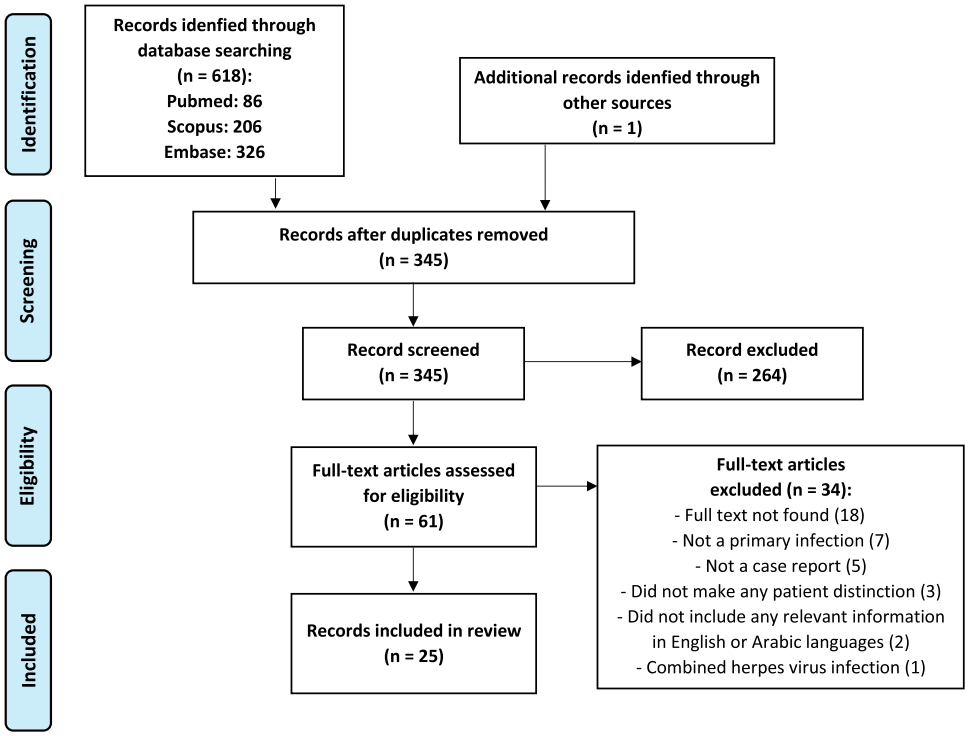

Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram.

Materials and methods

This systematic review was performed according to the protocol previously published on PROSPERO, We followed the recommendations of Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA)[5], Supplemental Digital Content 3, http://links.lww.com/MS9/A273 when reporting the results of our study. The work has been reported in line with Assessing the methodological quality of systematic reviews (AMSTAR), Supplemental Digital Content 4, http://links.lww.com/MS9/A274[6].

Table 2

Summary of patients' characteristics and clinical features

Summary of patients' characteristics and clinical features	
Age, years (median, IQR)	37 (10–47.5)
Sex	
Male	(22/28), 81.5%
Female	(6/28), 19.5%
VZV skin lesions to weakness, days (median, IQR)	10 (7–19)
Clinical type	
Sensory-motor	(17/26), 65.4%
Pure motor	(9/26), 34.6%
Neurological features	
Severity of weakness	
Tetraparesis	(22/27), 81.5%
Paraparesis	(4/27), 14.8%
No weakness	(1/27), 3.7%
Deep tendon reflex	
Absent	(21/24), 87.5%
Hyporeflexia	(3/24), 12.5%
Cranial nerve paresis	(16/19), 84%
Facial	(12/16), 75%
Bulbar	(4/16), 25%
Abducens	(2/16), 12.5%
Trigeminal	(1/16), 6%

IQR, interquartile range; VZV, varicella–zoster virus.

Search strategy and study selection

PubMed, Scopus, and Embase are systematically searched from their inception to 9 May 2022 to collect case reports and case series of GBS following VZV infection. The keywords of the search strategy were: (Chickenpox OR Varicella–zoster) AND (Guillain–Barre OR "Acute inflammatory demyelinating polyradiculoneuropathy"). The search terms were modified to fit each database, and the search strategy for each database is appended in Appendix I, Supplemental Digital Content 1, http://links.lww.com/MS9/A271. No restrictions were applied regarding the date of publication. To find more studies eligible for inclusion, the reference sections of the included full-text articles were manually evaluated, in addition to the similar articles for each included study. Both subsequent Screening processes (title-abstract screening and full-text screening) were conducted independently and simultaneously by two reviewers to get relevant articles according to the inclusion and exclusion criteria. Disagreements were resolved by a third reviewer intervention or discussion.

Eligibility criteria

We included in this systematic review full-text case reports and case series of patients with GBS that occurred following primary infection with VZV with no age restrictions. Patients with GBS that occurred following VZV reactivation or combined virus infection were excluded. Prospective studies and reviews were excluded. We excluded any study that did not make any patient

Table 3

Summary of diagnostic workup outcomes

Summary of diagnostic workup outcomes	
CSF findings	
CSF protein, mg/dl (median, IQR)	141 (68.57–245.65)
CSF cells, n/ml (median, IQR)	3 (0–7.5)
CSF, ACD	(20/26), 77%
Serologic tests	
VZV IgM	
Positive	(13/14), 93%
Negative	(1/14), 7%
VZV IgG	
Positive	(4/8), 50%
Negative	(4/8), 50%
NCS subtype	
AIDP	(15/23), 65.3%
AMAN	(4/23), 17.4%
AMSAN	(2/23), 8.7%
MFS	(1/23), 4.3%
Normal	(1/23), 4.3%
PCR/ VZV-DNA	
Positive	(2/7), 29%
Negative	(5/7), 71%
MRI	
Pathological changes	(7/15), 47.7%
Unremarkable	(8/15), 53.3%
Hospital Stay, days (median, IQR)	14 (12–31.5)

ACD, albuminocytological dissociation; AIDP, acute inflammatory demyelinating polyneuropathy; AMAN, acute motor axonal neuropathy; AMSAN, acute motor and sensory axonal neuropathy; CSF, cerebrospinal fluid; IgG, immunoglobulin G; IgM, immunoglobulin M; IQR, interquartile range; MFS, Miller–Fisher syndrome; NCS, nerve conduction study; PCR, polymerase chain reaction; VZV, varicella–zoster virus.

distinction or did not include any relevant information in the English or Arabic languages.

Data extraction, quality assessment, and statistical analysis

The data were extracted from the eligible studies including the first author's last name, year, journal, country, clinical neurological features, clinical and NCS subtypes, immunological and serological diagnostic workup outcomes, treatment, and outcome. A reviewer performed the data extraction process and checked by another for accuracy and consistency. Simultaneously, we assessed the risk of bias in included studies based on the NIH quality assessment tool for case series/case reports[7]. For each eligible study, the quality assessment was performed by one investigator and reviewed by another. Conflicts in data extraction and quality assessment processes were resolved through discussion among authors, or the involvement of a third author (Y.N.) if necessary. Data were analyzed using BMI SPSS Statistics 24. A descriptive and qualitative synthesis of data was performed, in a way that the pooled frequencies and percentages are used for reporting the categorical variables, and medians with interquartile ranges for the continuous variables.

Results

Literature search and quality assessment

Out of 618 articles identified by searching the current literature, only 25 articles were eligible for inclusion. These articles reported 28 cases. Of these studies, three were of poor quality, eight were of fair quality, and 14 were of high quality, and the details of the quality assessment process are included in (Appendix 2, Supplemental Digital Content 2, http://links.lww.com/MS9/A272). The characteristics of included studies are listed in (Table 1), and the reasons for exclusion after full-text screening can be found in the PRISMA flow diagram (Fig. 1).

Patient presentation and clinical features

Among the 29 patients including our patient, the age of presentation ranged from 1.5 to 70 years [median, interquartile range (IQR)]: 37 (10–47.5) with a yield for males ($n = 22/28$, 81.5%). The time interval between the appearance of the rash and the onset of muscle weakness ranged from 2 to 28 days (median, IQR): 10[7–19]. Most of the patients presented with sensory-motor symptoms ($n = 17/26$, 65.4%), and suffered from tetraparesis ($n = 22/27$, 81.5%). Deep tendon reflexes were absent in 21 patients (87.5%) and weak in three patients (12.5%). Cranial nerve palsy was present in 16/19 patients (84%), and the seventh cranial nerve was the most commonly affected nerve ($n = 12/16$, 75%), while only one injury to the fifth nerve was reported (6%). (Table 2).

Diagnostic workup and treatment

Lumbar puncture showed elevated protein level in 23/26 patients (88%) (median, IQR): 141 mg/dl (68.57–245.65) with normal CSF cells number in 22/25 patients (88%) (median, IQR): 3 n/ml (0–7.5). As a result, albuminocytological dissociation was present in 20/26 patients (77%). The results of the serological tests included a positive VZV IgM in most of the cases that reported this result ($n = 13/14$, 93%), while VZV IgG was positive in only half of the cases. The NCS results showed the dominance of the acute inflammatory demyelinating polyneuropathy subtype over the other subtypes ($n = 15/23$, 65.3%). in addition, polymerase chain reaction (PCR)/ VZV-DNA was negative in most patients ($n = 5/7$, 71%). The length of hospital stay ranged from 8 to 67 days (median, IQR): 14 (12–31.5). (Table 3) The magnetic resonance imaging showed pathological findings in only 47.7% of the patients, for all the cases after 2007, IVIg was the drug of choice, in addition to acyclovir in some cases. The details of CSF, NCS, MRI findings, treatment, and the outcome for each patient are in (Table 4).

Discussion

GBS is a rare acute inflammatory disease that affects the peripheral nervous system and is caused by autoimmune-mediated nerve damage triggered by a prior infectious event[4]. The diagnosis of GBS is based on clinical features and a neurological examination and is supported by the nerve conduction study and CSF analysis. The most important feature of GBS is albuminocytological dissociation which means elevated protein but absent pleocytosis on cerebrospinal fluid. However, in a small percentage of cases, this dissociation is absent, including our case[8]. Moreover, GBS is also characterized by areflexia or hyporeflexia of deep tendon reflexes while normal or hyper-reflexia was rarely observed[8,9]. Based on the nerve conduction study, GBS is classified into four subtypes. AMAN is the most common in Asia and Central and South America 30–65%, while in the USA and Spain

Table 4

The details of CSF, NCS, MRI findings, treatment, and the outcome for each patient

	CSF findings						
References	Protein (mg/dl)	Cells, n/c. mm	ACD	NCS subtype	MRI findings	Treatment	Outcome
Welch *et al.*[17]	240	2	✓	N/R	N/R	Soluble aspirin	After the patient was discharged, he was well. But stated to get slightly tired after walking two or three miles.
Leeming *et al.*[18]	N/R	N/R	N/R	AIDP	N/R	N/R	N/R
Twomey *et al.*[19]	10	N/R	N/R	AIDP	N/R	N/R	Power had returned to normal by 4 months, although the Achilles tendon reflexes were still absent.
Arruda *et al.*[20]	92	3	✓	N/R	N/R	Propranolol, and Carbamazepine 600 mg/day for dysesthesias.	The patient was discharged when he was tetraparetic and bedridden and did not return for follow-up.
Sanders *et al.*[21]	183	12	✓	N/R	N/R	Without any drug treatment	After 1 month, his motor disturbances had recovered completely. The tendon reflexes remained absent for another 3 months.
Ormerod *et al.*[22]	500	< 12	✓	AIDP	N/R	Without any drug treatment	After 1 year, he was asymptomatic.
Da Rosa-Santos *et al.*[23]	1500	0	✓	AIDP	N/R	Orotracheal intubation and controlled mechanical ventilation	After 4 months, the paralysis was limited to the distal third muscles of both lower extremities.
Sabogal *et al.*[24]	215	3	✓	AIDP	N/R	N/R	N/R
Yoshikawa *et al.*[25]	78	0	✓	AMAN	N/R	IV gamma globulin 1 g/kg/day	After treatment, the patient's respiration quickly improved.
Hamad *et al.*[26]	62	0	✓	N/R	N/R	N/R	After 3 months, the patient recovered completely with mild facial palsy.
	210	0	✓	AMAN	N/R	N/R	One month after discharge, the patient recovered completely.
Inan *et al.*[27]	> 55	0	✓	AIDP	Diffuse thickening of the cauda equina	Acyclovir + IVIg	Gradually recovery (after 1 month walking with help, after 2 months running).
Cresswell *et al.*[28]	100	0	✓	AIDP	Unremarkable	IV acyclovir 10 mg/kg + valacyclovir 1 g three times/day orally for 7 days + IVIg (Vigam) 0.4 mg/kg/d for 5 days	The power in his arms and legs quickly improved, but speech and facial nerve palsies have been slower to improve.
Munoz-Sellart *et al.*[11]	141	0	✓	AIDP	Unremarkable	IV acyclovir 10 mg/kg every 8 h for 14 days + dexamethasone 16 mg/day for 10 days + IVIg 0.4 mg/kg/d for 5 days.	Slight peripheral facial palsy on the right side after 6 months.
Modi *et al.*[29]	72	5	✓	AIDP	N/R	IVIg at a dose of 2 g/kg/d for 5 days.	After 1 month, the power of both upper and lower limbs improved and she had no neurological symptoms.
Assi *et al.*[30]	452	1055	×	AIDP	Unremarkable	Acyclovir 10 mg/kg every 12 h + Piperacillin + Tazobactam	The treatment was stopped at the patient's request and his family, and they opted for comfort care.
Paul *et al.*[31]	301	5	✓	AMAN	Unremarkable	IV methylprednisolone 1 g + Physiotherapy, posture, and skin care	When the patient was discharged, she had a significant weakness. And she was improving very slowly during follow-up.
Cokyaman *et al.*[32]	N/R	N/R	N/R	AIDP	Unremarkable	IVIg 0.4 mg/kg/d for 5 days + Ceftriaxone + Acyclovir	After 3 months, the patient muscle strength improved, and deep tendon reflexes were hypoactive.
Tatarelli *et al.*[33]	251.3	0.8	✓	AIDP	N/R	Acyclovir + IVIg 400 mg/kg/d for 5 days + Plasma exchange	Recovery almost occurred for neurological function except for continuing painful acral paresthesias.
	65.5	4	✓	N/R	Posterior reversible encephalopathy	IVIg + Oral acyclovir 800 mg 5 times/d	After rehabilitation, she was discharged without neurological problems.
	103.7	0.8	✓	N/R	Unremarkable	IVIg	The patient got recovery without neurological symptoms.
Caramăngiu *et al.*[34]	54	0	×	AIDP	Unremarkable	Immunoglobulin and plasmapheresis, antibiotics, gastric antisecretory, anticoagulants, analgesics, opioids,	N/R

Table 4

(*Continued*)

References	CSF findings: Protein (mg/dl)	CSF findings: Cells, n/c. mm	ACD	NCS subtype	MRI findings	Treatment	Outcome
						antipyretics, corticosteroids, vitamins, blood products, solutions, and calorie electrolytic rebalancing.	
Bhatt *et al.*[35]	168	170	×	AMSAN	Abnormalities areas in cortical and subcortical regions	IVIg + IV acyclovir + IV steroid 1 g for 5 days	The patient had started to recover power in his limbs, but he also developed Infective endocarditis following ChickenPox.
Xifaras *et al.*[36]	96	5	✓	AIDP	Brain MRI scan showed chronic microvascular ischaemic disease. The cervical/thoracic MRI scan was normal	N/R	N/R
Kofahi *et al.*[37]	61.5	3	✓	AMSAN	Enhancement in multiple thoracic and lumbar nerve roots	IVIg 400 mg/kg/d for 5 days + oral acyclovir.	After 6 months, she reported significant improvement in pinprick and temperature sensation and walked independently.
Balamurugesan *et al.*[38]	220	620	×	AIDP	Surface enhancement over the cauda equina roots and the conus medullaris	IVIg 2 g/kg/d for 5 days	After 6 weeks, the patient had power in all four limbs 5/5, and his cranial nerve palsies had improved.
Arora *et al.*[39]	270	40	✓	Normal	N/R	IVIg 400 mg/kg/d for 5 days	The patient rapidly improved with milder ptosis, better gait, and improved pupillary reflexes, and improved visual acuity bilaterally. The imaging findings were also improved during the 4 months follow-up.
Spyromitrou-Xioufi *et al.*[40]	14	10	×	MFS	Symmetric enhancement of the fifth cranial nerves bilaterally and clear enhancement of the right sixth nerve and there was no enhancement of the optic nerve	IVIg 400 mg/kg/d for 5 days + injection acyclovir 10 mg/kg per dose thrice a day for 7 days.	Four weeks after discharge, the patient was without support, and showed complete recovery of right-sided facial palsy and bulbar palsy.

ACD, albuminocytological dissociation; AIDP, acute inflammatory demyelinating polyneuropathy; AMAN, acute motor axonal neuropathy; AMSAN, acute motor and sensory axonal neuropathy; CSF, cerebrospinal fluid; IVIg, intravenous immunoglobulin; MFS, Miller–Fisher syndrome; N/R, not reported; NCS, nerve conduction study.

(AMAN) subtype ranges between 6 and 7%[10]. The nerve conduction study of our patient is consistent with this subtype.

For the diagnostic aspect and the steps involved in the diagnosis, the diagnostic criteria for GBS issued by NINCDS in 1978 are always relied on, which are based on the dominant symptoms of the patient such as progressive weakness in one or all of the limbs associated with the absence or weak reflexes[1]. In the current study, we reviewed the medical literature and compiled previously reported cases of GBS that were specifically induced by a primary VZV infection. As a comparison of the points of agreement between our review and other studies related to the same subject, we have found that the predominant clinical type in most of the reviewed cases is the sensory-motor type, and this agrees with the findings of other studies. As for the association of symptoms with some additional neurological manifestations, we have found a remarkable association in most of the patients with peripheral facial palsy by 75%, while previous studies indicated this association by almost 40%[11].

In addition to the above, we took into account the presence or absence of an MRI of patients, which aims to rule out other possible causes of symptoms other than GBS. Only in half of the cases, MRI shows pathological change, which is the enhancement of the thickened nerve roots in the conus medullaris and cauda equina. In the other half, this enhancement was unremarkable. However, in some cases, MRI can be an additional and supportive diagnostic tool in diagnosing GBS and in monitoring response to treatment, especially when the diagnosis is difficult based on clinical, serological, and CSF findings[12,13], As well as the presence of a positive PCR test, which appeared in only about a third of the cases included.

The role of Acyclovir and IVIG in the treatment of such conditions is well known[14,15]. As well as the occurrence of a clinical response when using corticosteroids only in the acute phase of the disease, as was the management of our previous case and some other similar cases. Although corticosteroid use has no effect on the long-term outcome[16].

The literature lacks high-quality cohort studies on rare neurological complications of VZV, particularly Guillain–Barre. This explains the lack of systematic reviews with a large sample size. Hence the importance of our review in summarizing published cases of this rare complication. Although this review was built and conducted according to systematic criteria, the evidence it provides remains weak, because we reviewed case reports and case series. What limits our study is also the differences in the methods used in diagnosis and treatment between the past and the present, the old cases neither use modern diagnostic methods such as MRI and PCR, nor did they use the specific medical treatment of the disease. In addition, follow-up duration was not specified in some cases, and there are a few cases of low quality in our review. Nevertheless, this systematic review provides the most recent evidence for clinical manifestations, diagnostic workup, and treatment of Guillain-Barré patients following primary VZV infection.

Conclusion

Although GBS is a rare neurological complication of primary infection with VZV, we should suspect this syndrome when a patient develops progressive muscle weakness within 1–2 weeks of the onset of the rash, regardless of the presence or absence of deep tendon reflexes and albuminocytological dissociation. The diagnosis is then confirmed by subsequent diagnostic procedures to give specific treatment. According to the results of our systematic review, IVIg is a safe and effective treatment for this complication, in addition to treatment with acyclovir, which may be necessary if the patient has not achieved complete recovery from chickenpox. Drug therapy ensures a gradual improvement for the patient over a period of weeks to several months. In the future, comparative studies with a large sample size are needed. In order to get better evidence.

Ethical approval

The requirement for institutional review board approval was waived because this case report did not contain the content required for ethical approval.

Consent

Written informed consent was obtained from the patient's parents/legal guardian for publication and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Author contribution

Y.N.: conceptualization, methodology, corresponding author, project administration, screening, data extraction, quality assessment, and writing process. O.H., A.A.A., N.H., A.H.M., H.M.: screening, data extraction, quality assessment, and writing process. S.H.: managed the patient and followed up, validity, reviewing and editing. N.A.: managed the patient and followed up, validity and scientific supervision.

Conflicts of interest disclosure

NA.

Research registration unique identifying number (UIN)

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