

The clinical characteristics and short-term prognosis in elderly patients with Guillain–Barré syndrome

Bing Zhang, MD, Xiujuan Wu, MD, Donghui Shen, MD, Ting Li, MD, Chunrong Li, MD, Mei Mao, MD, Hong-Liang Zhang, MD, PhD*, Kangding Liu, MD, PhD*

Abstract

To investigate the clinical characteristics and short-term prognosis of elderly patients with Guillain–Barré syndrome (GBS).

We retrospectively analyzed the clinical data of adult GBS. According to the age, the enrolled subjects were divided into 2 groups, that is, patients ≥ 60 years (elderly group) and those aged 18 to 59 years (nonelderly group). The clinical characteristics and short-term prognosis of the patients in the 2 groups were compared.

In total, 535 patients were enrolled. There were 67 patients fell into the elderly group with a mean age of 69 years old; while 468 patients fell into the nonelderly group with a mean age of 39 years old. We found that the elderly patients had significantly lower incidence of antecedent infections (49.3% vs 66.2%, $P < 0.01$). The time from onset to admission (5 vs 4 days, $P < 0.05$) and time from onset to nadir (7 vs 6 days, $P < 0.05$) were significantly longer in the elderly patients. It was noteworthy that more elderly patients were found with lymphocytopenia (55.4% vs 37.3%, $P < 0.01$), hyponatremia (25.0% vs 10.2%, $P < 0.01$), hypoalbuminemia (9.0% vs 2.6%, $P < 0.05$), and hyperglycemia (34.3% vs 15.2%, $P < 0.01$). Importantly, the elderly patients had longer duration of hospitalization (17 vs 14 days, $P < 0.05$), higher incidence of pneumonia (29.9% vs 18.8%, $P < 0.05$), and poorer short-term prognosis (58.2% vs 42.7%, $P < 0.05$). In patients with severe GBS, no significant differences were observed in disease severity, treatment modality, incidence of pneumonia, and duration of hospitalization between the 2 groups. However, more patients in the elderly group showed poor short-term prognosis (84.1% vs 63.8%, $P < 0.01$). Further, old age (≥ 60 years) (OR = 2.906, 95% CI: 1.174–7.194, $P < 0.05$) and lower Medical Research Council (MRC) score at nadir (OR = 0.948, 95% CI: 0.927–0.969, $P < 0.01$) were risk factors for poor short-term prognosis in severe GBS patients.

The clinical characteristics and short-term prognosis of elderly patients with GBS are distinct from nonelderly adults. Old age (≥ 60 years) and lower nadir MRC score serve as predictor for poor short-term prognosis in severe GBS patients.

Abbreviations: AMAN = acute motor axonal neuropathy, GBS = Guillain–Barré syndrome, HFGS = Hughes Functional Grading Scale, MRC = Medical Research Council.

Keywords: clinical features, elderly, Guillain–Barré syndrome, short-term prognosis

1. Introduction

Guillain–Barré syndrome (GBS) is an immune-mediated disease of the peripheral nervous system. About two-thirds of GBS are

Editor: Johannes Mayr.

BZ and XW contributed equally to this work.

Authorship: HLZ, KL, XW, and BZ participated in conception and design of the study, analysis and interpretation of data, drafting and revising the manuscript, as well critically revising it. TL participated in collecting clinical data and helping to revise the manuscript. DS participated in analyzing the clinical data and performing the statistical analysis. CL and MM participated in collecting and analyzing the clinical data. All authors have read and approved the manuscript.

Funding/support: The work was supported by grants from the National Natural Science Foundation of China (No. 81271294).

The authors have no conflicts of interest to disclose.

Neuroscience Center, Department of Neurology, the First Hospital of Jilin University, Jilin University, Changchun, China.

* Correspondence: Hong-Liang Zhang, Kangding Liu, Neuroscience Center, Department of Neurology, the First Hospital of Jilin University, Xinmin Street 71#, 130021 Changchun, China (e-mail: drzh@hotmial.com, kangdingliu@163.com).

Copyright © 2017 the Author(s). Published by Wolters Kluwer Health, Inc. This is an open access article distributed under the Creative Commons Attribution License 4.0 (CCBY), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Medicine (2017) 96:1(e5848)

Received: 25 April 2016 / Received in final form: 28 October 2016 / Accepted: 15 December 2016

<http://dx.doi.org/10.1097/MD.0000000000005848>

triggered by antecedent infectious agents, leading to the concept that GBS is a postinfectious immune-mediated disorder; however, accumulating GBS following noninfectious factors has also been reported in recent years.^[1] Additionally, it was reported that transient immunosuppression may be an important link in the pathogenesis of GBS.^[2] GBS could occur at any age.^[3] The epidemiological data from western countries found that the incidence of GBS increased with age;^[4] however, the incidence of GBS based on the epidemiological data in the elderly patients in China showed significant differences, which found that the incidence of GBS among elderly patients was remarkably lower in Harbin while the GBS incidence increased with age in Jiangsu province.^[5,6] In addition, studies have shown that elderly patients with GBS have more severe disease, less involvement of cranial nerve, more axonal damage, and slower recovery; however, the results of different studies show variation.^[7–9] Also, some studies have demonstrated that old age was an important factor in predicting poor prognosis of GBS besides the severity of the disease.^[10–12] The aim of this study was to explore the clinical features and short-term prognosis of elderly patients with GBS through a retrospective study.

2. Subjects and methods

2.1. Subjects

This retrospective study was approved by the ethics committee of The First Hospital of Jilin University, Changchun, China.

Subjects were selected from patients who met the diagnostic criteria of GBS^[13] and received sequential treatment during hospitalization in the Department of Neurology of the First Hospital of Jilin University, during January 2003 to December 2014. Subjects were excluded from the study if they: were aged < 18 years; refused the treatment or diagnosed with chronic inflammatory demyelinating polyradiculoneuropathy or Miller Fisher syndrome. The subjects were categorized into 2 groups based on their age: elderly group (≥ 60 years) and nonelderly group (<60 years). Clinical data from all subjects were analyzed retrospectively including age, gender, season of disease occurrence, antecedent infections (mainly include upper respiratory tract infection, diarrhea, and fever of unknown origin), initial symptoms, time from onset to admission/nadir, tendon reflex, sensory disturbances, cranial nerve damage, Medical Research Council (MRC) score, and Hughes Functional Grading Scale (HFGS) score at nadir, whether requiring mechanical ventilation, complications, treatment modality, duration of hospitalization, MRC and HFGS score at discharge, laboratory test results, and electrophysiological findings. The laboratory test results include complete blood count (lymphocytopenia: <20% of lymphocytes), serum sodium (hyponatremia: <135 mmol/L), serum potassium (hypokalemia: <3.5 mmol/L), serum albumin (hypoalbuminemia: <30 g/L), blood glucose level (hyperglycemia: fasting blood glucose ≥ 7.0 mmol/L or glycated hemoglobin $\geq 7.0\%$), liver function level and cerebrospinal fluid protein (g/L), and cell number (unit/mm³). And the tests of complete blood count, serum sodium, and serum potassium were examined in the 1st day of after admission, while the tests of serum albumin, fasting blood glucose, glycated hemoglobin, and liver function examined in the 2nd day of after admission. Generally we did lumbar puncture and electrophysiological examination around 2 weeks after onset. The results of electrophysiological examination were categorized into acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy (AMAN), and unclassifiable group using the electrophysiological criteria proposed by Hadden et al.^[14]

2.2. Clinical score and evaluation of short-term prognosis

Clinical scores of all the patients in these groups were evaluated at 2 time points (at nadir and at discharge) using HFGS and MRC score. The HFGS score was defined as follows:^[15] 0, healthy state; 1, minor symptoms and capable of running; 2, able to walk ≥ 5 m without assistance but able to run; 3, able to walk 5 m across an open space with help; 4, bedridden or chair-bound; 5, require assisted ventilation for at least a part of the day; and 6, dead. Muscle weakness was evaluated by the MRC sum score of 6 bilateral muscles in arms and legs, ranging from 0 (tetraparalytic) to 60 (normal strength).^[16] If the HFGS score was ≥ 4 points at nadir, it is regarded as severe GBS.^[17] Generally, the patient was allowed to discharged from the hospital when his condition was improved or stable in our department. Thus, if the HFGS score was ≥ 3 points when a patient was discharged from the hospital, the patient was considered to have a poor short-term prognosis.

2.3. Statistical analysis

Data were analyzed using the SPSS 17.0 software (IBM, West Grove, PA), and normality and homogeneity of variance tests were performed. The normally distributed measurement data were represented by mean \pm standard deviation, and the means were compared using independent samples *t* test. Although the

nonnormally distributed measurement data were represented by medians and interquartile ranges [M (Q1–Q3)] and were compared using the independent sample rank sum test. Chi-square test was used for evaluating the difference in the count data of patients in different groups. Logistic regression models were performed to determine risk factors of poor short-term prognosis. Variables that were statistically significant in univariate analysis were further analyzed in a multivariate regression analysis. For all statistical tests, $P < 0.05$ was considered to be significant.

3. Results

3.1. Distinct clinical features of GBS between elderly and nonelderly group

In total, 535 patients with GBS were enrolled in the study. There were 67 patients in the elderly group with a mean age of 69 years old, and 468 patients fell into the nonelderly group with a mean age of 39 years old. Higher proportion of male was found in both groups. The ratio of male to female in the 2 groups showed no significant difference (1.39:1 vs 1.64:1, $P > 0.05$). Comparisons of the clinical characteristics between the 2 groups were illustrated in Table 1. The incidence of antecedent infection in the elderly group was significantly lower than the nonelderly group (49.3% vs 66.2%, $P < 0.01$). Moreover, time from onset to admission (5 vs 4 days, $P < 0.05$) and time from onset to nadir in the elderly group (7 vs 6 days, $P < 0.05$) were both longer than the nonelderly group, indicating that the progression of elderly GBS patients was slower. Figure 1 revealed the comparisons of initial symptoms between the 2 groups and there was no statistically significant difference between the 2 groups ($P > 0.05$, excluding 2 patients with limb pain). In addition, the season of morbidity, tendon reflex, sensory dysfunction, cranial nerve damage, MRC

Table 1
Comparison of clinical characteristics of GBS between elderly and nonelderly group.

	Group 1 (n=67)	Group 2 (n=468)	P
Age, years	69 (62–74)	39 (29–48)	
Male to female ratio	1.39:1	1.64:1	0.532
Incidence of GBS in different seasons			
Spring	16 (23.9%)	118 (25.2%)	0.476
Summer	19 (28.4%)	171 (36.5%)	
Autumn	19 (28.4%)	105 (22.4%)	
Winter	13 (19.4%)	74 (15.8%)	
Antecedent infection	33 (49.3%)	310 (66.2%)	0.000
URI	22 (32.8%)	174 (37.2%)	0.490
Diarrhea	14 (20.9%)	153 (32.7%)	0.051
Time from onset to admission, days	5 (3–10)	4 (3–7)	0.010
Hyporeflexia or areflexia	63 (94.0%)	443 (94.7%)	0.834
Sensory disturbance	36 (53.7%)	229 (48.9%)	0.462
Cranial nerve deficits	26 (38.8%)	198 (42.3%)	0.587
Facial nerve involvement	19 (28.4%)	145 (31.0%)	0.663
Bulbar palsy	8 (10.7%)	75 (16.0%)	0.388
Time from onset to nadir, days	7 (5–10)	6 (4–9)	0.047
MRC score at nadir	38 (24–48)	42 (30–48)	0.281
HFGS score at nadir	4 (3–4)	4 (3–4)	0.105
Severe type	44 (65.7%)	260 (55.6%)	0.118
Mechanical ventilation	13 (29.4%)	65 (13.9%)	0.232

Group 1, elderly group, that is, patients aged ≥ 60 years; group 2, nonelderly group, that is, patients aged 18 to 59 years. GBS=Guillain-Barré syndrome, HFGS=Hughes Functional Grading Scale, MRC=Medical Research Council sum score, URI=upper respiratory tract infection.

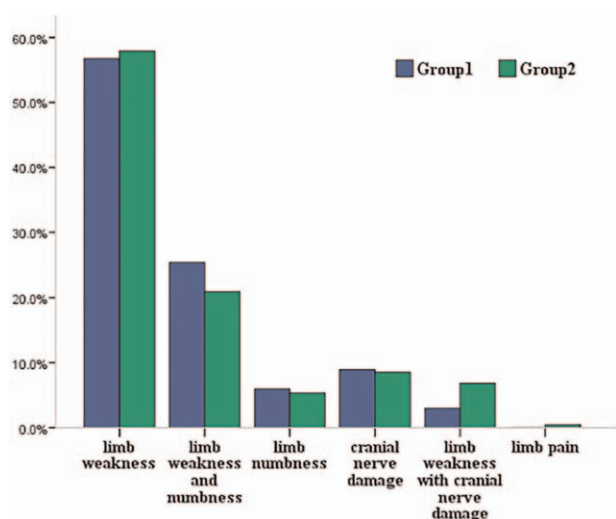


Figure 1. Comparisons of the initial symptoms between the 2 groups. Group 1 was patients aged ≥ 60 years while group 2 was patients aged 18 to 59 years. The initial symptoms of the patients in the 2 groups were as follows (group 1 vs group 2): limb weakness (56.7% vs 57.9%), limb weakness and numbness (25.5% vs 20.9%), limb numbness (6.0% vs 5.3%), cranial nerve damage (9.0% vs 8.5%), limb weakness with cranial nerve damage (3.0% vs 6.8%), and limb pain (0% vs 0.4%).

score at nadir, HFGS score at nadir, the proportion of patients with severe GBS, and the proportion of patients requiring mechanical ventilation did not show statistically significant difference ($P > 0.05$).

3.2. Abnormal laboratory tests were more common in elderly patients with GBS

We further compared the laboratory tests between the elderly and nonelderly groups. We found the proportion of lymphocytes in the elderly group was significantly lower than the nonelderly group ($20.0\% \pm 10.0\%$ vs $23.0\% \pm 10.4\%$, $P < 0.05$), and patients with lymphocytopenia were more common in the elderly group (Fig. 2A). Similarly, the proportion of patients with hyponatremia, hypoalbuminemia, and hyperglycemia was all higher in the elderly group than the nonelderly group (Fig. 2B–D). The incidence of hypokalemia and abnormal liver function in the 2 groups showed no statistically significant differences (Fig. 2E and F). Cerebrospinal fluid protein ($0.76 [0.57-1.23]$ vs $0.81 [0.53-1.26]$, $P > 0.05$) (reference value: $0.15-0.45$ g/L) and the cell count ($3 [2-6]$ vs $4 [2-7]$, $P > 0.05$) also showed no statistically significant differences (40 patients in the elderly group and 292 patients in the nonelderly group received lumbar puncture). As to the results of the electrophysiological examination, we found no statistically significant differences between the 2 groups in Table 2 (41 patients in the elderly group and 213

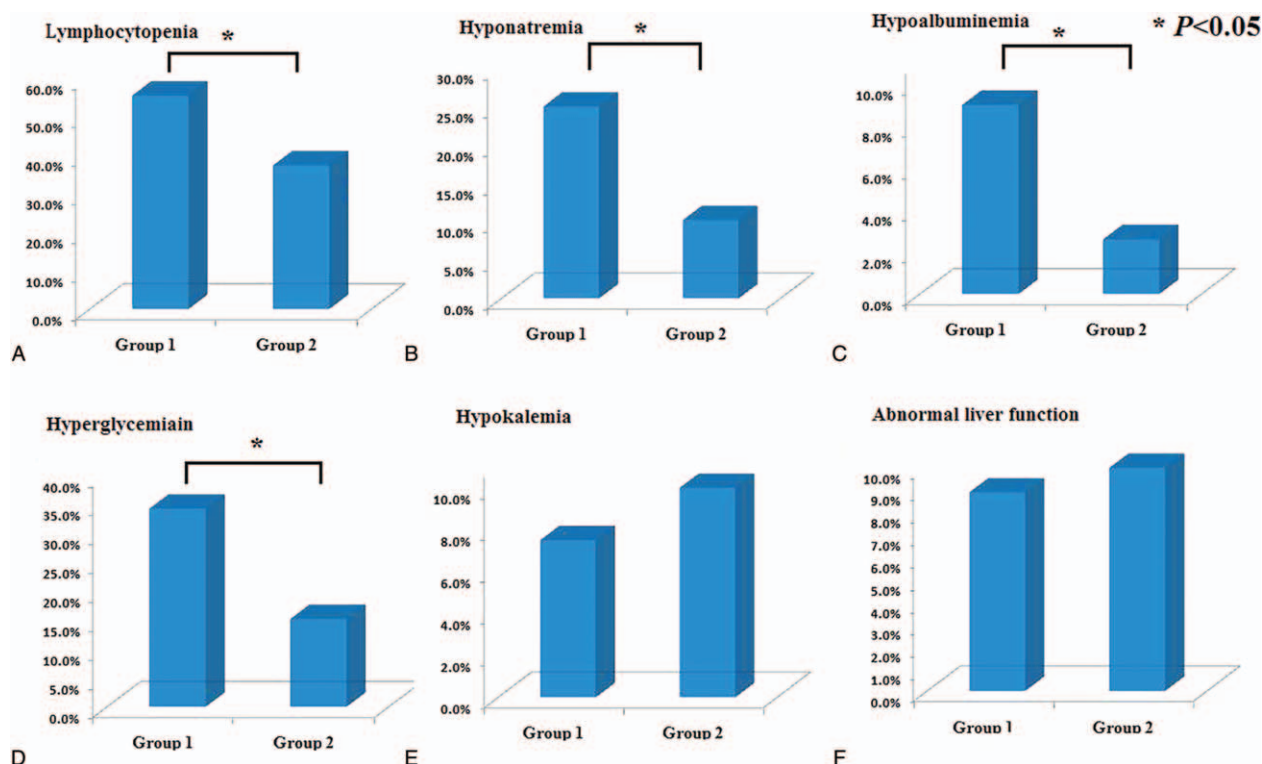


Figure 2. Comparisons of results of laboratory test. Group 1 was patients aged ≥ 60 years while group 2 was patients aged 18 to 59 years. The proportion of patients with lymphocytopenia was higher in group 1 than the group 2 (36/65 [55.4%] vs 167/448 [37.3%], $P < 0.01$) (A). The proportion of patients with hyponatremia was higher in group 1 than the group 2 (16/64 [25.0%] vs 44/432 [10.2%], $P < 0.01$) (B). The proportion of patients with hypoalbuminemia was higher in group 1 than the group 2 (6/67 [9.0%] vs 12/468 [2.6%], $P < 0.05$) (C). The proportion of patients with hyperglycemia was higher in group 1 than the group 2 (23/67 [34.3%] vs 71/468 [15.2%], $P < 0.01$) (D). The proportion of patients with hypokalemia showed no statistically significant difference in the 2 group (5/64 [7.8%] vs 47/440 [10.7%], $P > 0.05$) (E). The proportion of patients with abnormal liver function showed no statistically significant difference in the 2 group (5/64 [7.8%] vs 47/440 [10.7%], $P > 0.05$) (F).

Table 2**Comparison of electrophysiological examination of GBS between elderly and nonelderly group.**

	Group 1 (n=41)	Group 2 (n=213)	P
AIDP	29 (70.7%)	133 (62.4%)	0.312
AMAN	11 (26.8%)	54 (25.4%)	0.843
unclassifiable	1 (2.4%)	26 (12.2%)	0.114

Group 1, elderly group, that is, patients aged ≥ 60 years; group 2, nonelderly group, that is, patients aged 18 to 59 years. AIDP = acute inflammatory demyelinating polyneuropathy, AMAN = acute motor axonal neuropathy, GBS = Guillain-Barré syndrome.

Table 3**Comparison of short-term prognosis of GBS between elderly and nonelderly group.**

	Group 1 (n=67)	Group 2 (n=468)	P
Treatment modality			
IVIg	32 (47.8%)	210 (44.9%)	0.694
IVIg + intravenous corticosteroids	14 (20.9%)	84 (17.9%)	
Intravenous corticosteroids	12 (17.0%)	84 (17.9%)	
Supportive treatment	9 (13.4%)	90 (19.2%)	
Pneumonia	20 (29.9%)	88 (18.8%)	0.035
Duration of hospitalization, days	17 (13–22)	14 (11–20)	0.010
Patients with poor prognosis when discharged	39 (58.2%)	200 (42.7%)	0.017

Group 1, elderly group, that is, patients aged ≥ 60 years; group 2, nonelderly group, that is, patients aged 18 to 59 years. GBS = Guillain-Barré syndrome, IVIg = intravenous immunoglobulin.

patients in the nonelderly group received the electrophysiological examination).

3.3. Elderly patients with GBS had poorer short-term prognosis

No statistically significant difference was observed in disease severity between the elderly and nonelderly groups. Similarly, the treatment modality did not differ between the 2 groups (Table 3). However, the duration of hospitalization was significantly longer in the elderly group (17 vs 14 days, $P < 0.05$), along with higher proportion of patients with pneumonia (29.9% vs 18.8%, $P < 0.05$) and poorer short-term prognosis at discharge from the hospital (58.2% vs 42.7%, $P < 0.05$) (Table 3).

Table 4**Comparison of hospitalization and discharge conditions of nonsevere GBS between elderly and nonelderly group.**

	Group 1 (n=23)	Group 2 (n=208)	P
MRC score at nadir	48 (46–60)	48 (48–56)	0.974
HFGS score at nadir	3 (1–3)	3 (2–3)	0.957
Treatment modality			
IVIg	10 (43.5%)	77 (37.0%)	0.938
IVIg + intravenous corticosteroids	2 (8.7%)	18 (8.7%)	
Intravenous corticosteroids	5 (21.7%)	54 (26.0%)	
Supportive treatment	6 (26.1%)	59 (28.4%)	
Pneumonia	2 (8.7%)	8 (3.8%)	0.000
Duration of hospitalization, days	14 (12–19)	13 (9.25–17)	0.126
Poor prognosis after discharge from the hospital	2 (8.7%)	34 (16.3%)	0.511
HFGS score improvement ≥ 1 point	13 (56.5%)	98 (47.1%)	0.392
HFGS score improvement ≥ 2 points	4 (17.4%)	37 (17.8%)	1.000
MRC score improvement ≥ 5 points	11 (47.8%)	85 (40.9%)	0.520
MRC score improvement ≥ 10 points	6 (26.1%)	46 (22.1%)	0.727

Nonsevere GBS, HFGS score was < 4 points at nadir, group 1, elderly group, that is, patients aged ≥ 60 years; group 2, nonelderly group, that is, patients aged 18 to 59 years. GBS = Guillain-Barré syndrome, HFGS = Hughes Functional Grading Scale, IVIg = intravenous immunoglobulin, MRC = Medical Research Council sum score.

3.4. Comparable short-term prognosis of GBS patients with a HFGS < 4 points between elderly and nonelderly group

Out of the 231 patients whose HFGS score < 4 points at nadir, patients in the elderly group had higher proportion of pneumonia (Table 4); however, no statistically significant differences were observed with regard to the factors such as disease severity (MRC and HFGS at nadir), treatment modality, duration of hospitalization, and the proportion of patients with poor prognosis at discharge from the hospital. No statistically significant differences were observed in the improvement of HFGS and MRC score between the 2 groups when patients discharged from the hospital (Table 4).

3.5. Old age and lower nadir MRC serve as predictors for poor short-term prognosis in severe GBS patients

Totally, 304 patients with severe GBS were enrolled. Although no statistically significant differences were observed in disease severity, treatment modality, pneumonia, and the duration of hospitalization, the proportion of elderly patients with poor prognosis at discharge from the hospital was still higher (84.1% vs 63.8%, $P < 0.01$) (Table 5). Further analysis showed the proportion of patients with an improvement in HFGS score ≥ 2 and patients with an improvement in MRC score ≥ 10 from nadir to discharge were lower in the elderly group (Table 5). By using univariate analysis on variables including old age, antecedent infection, involvement of cranial nerve, MRC score at nadir, whether requiring mechanical ventilation, pneumonia, lymphocytopenia, hyponatremia, hypokalemia, hypoalbuminemia, hyperglycemia, abnormal liver function, axonal damage, and duration of hospitalization, we found that old age (≥ 60 years) (OR = 2.993, 95% CI: 1.284–6.979, $P < 0.05$), lack of antecedent infection (OR = 2.130, 95% CI: 1.244–3.646, $P < 0.01$), lower MRC score at nadir (OR = 0.949, 95% CI: 0.930–0.967, $P < 0.01$), pneumonia (OR = 1.840, 95% CI: 1.072–3.158, $P < 0.05$), and longer of duration of hospitalization (OR = 1.019, 95% CI: 1.001–1.036, $P < 0.05$) were associated with poor short-term prognosis. Furthermore, old age (OR = 2.906, 95% CI: 1.174–7.194, $P < 0.05$) and lower MRC score at nadir (OR = 0.948, 95% CI: 0.927–0.969, $P < 0.01$) were identified to be risk factors of poor short-term prognosis by multivariate analysis.

Table 5**Comparison of hospitalization and discharge conditions of severe GBS between elderly and nonelderly group.**

	Group 1 (n = 44)	Group 2 (n = 260)	P
MRC score at nadir	30 (12–41.5)	30 (16–36.75)	0.786
HFGS score at nadir	4 (4–5)	4 (4–4.75)	0.524
Treatment modality			
IVIg	22 (50.0%)	133 (51.2%)	0.658
IVIg + intravenous corticosteroids	12 (27.3%)	66 (25.4%)	
Intravenous corticosteroids	7 (15.9%)	30 (11.5%)	
Supportive treatment	3 (6.8%)	31 (11.9%)	
Pneumonia	18 (40.9%)	80 (30.8%)	0.183
Duration of hospitalization, days	18 (15–23.5)	16 (12–22)	0.114
Patients with poor prognosis when discharged	37 (84.1%)	166 (63.8%)	0.008
HFGS score improved ≥ 1 point	28 (63.6%)	180 (69.2%)	0.460
HFGS score improved ≥ 2 points	10 (20.7%)	105 (40.4%)	0.026
MRC score improved ≥ 5 points	32 (72.7%)	216 (83.1%)	0.101
MRC score improved ≥ 10 points	22 (50.0%)	179 (68.8%)	0.015

Nonsevere GBS, HFGS score was < 4 points at nadir; group 1, elderly group, that is, patients aged ≥ 60 years; group 2, nonelderly group, that is, patients aged 18 to 59 years. GBS = Guillain-Barré syndrome, HFGS = Hughes Functional Grading Scale, IVIg = intravenous immunoglobulin, MRC = Medical Research Council sum score.

4. Discussion

In our study, we found that the elderly patients had slower disease progression, lower incidence of antecedent infection, higher incidence of abnormal laboratory tests, and poorer short-term prognosis. Further, old age and lower MRC score at nadir were found to be risk factors for poor short-term prognosis in severe GBS.

In general, GBS is a monophasic disease, which usually reaches the nadir within 4 weeks. GBS could occur at any age. In this study, we found that the disease progression in the elderly patients was slower, which was contradictory to Winner et al.^[7] and Peric et al.,^[9] who found that the time from onset of disease until admission/nadir was similar in the elderly patients and nonelderly patients. However, the disease severity of patients in the 2 groups showed no significant difference in the MRC score, HFGS score, severe type, and proportion of mechanical ventilation in our study. This finding was consistent with the results of Winner et al.^[7] in UK, which found that the severity of disease was similar between elderly and nonelderly adults.^[7] However, our finding was contradictory to the results of Peric et al.^[9] in Eastern Europe, which found that elderly patients had more severe disease. In this study, the disease progression and the severity of disease of elderly GBS patients were different from other countries' findings, and it may be the peculiar feature of elderly GBS in China. Further, the epidemiologic studies of the different provinces in China are warranted to confirm this hypothesis.

GBS is considered as a disease caused by dysimmunity after infection, and there is evidence of antecedent infections in about two-thirds of the patients.^[1] It is of noteworthy that some other studies considered that immunosuppression may play an important role during the course of the disease.^[3,18] and patients with noninfectious triggers may be associated with the immunocompromised states.^[1] In this study, the proportion of patients without antecedent infection and with lymphocytopenia in the elderly group was significantly higher than that in the nonelderly group, implying that immunosuppression may be associated with the occurrence of GBS in the elderly patients. These results of this study were consistent with the results of an earlier related study that elderly patients had less antecedent infection.^[19] In addition, we found that the elderly patients were prone to develop pneumonia during the course of the disease,

which may be related to lymphocytopenia because lymphocytopenia would lessen the resistance to infection thereby increasing the risk of pneumonia. Some viral infections may induce lymphocytopenia; however, we could not confirm the relationship between lymphocytopenia and some certain viral infections in this study because we did not perform microbial or serological analyses. Meanwhile, classification of lymphocytes, detection of cytokines, and dynamic observation of changes in lymphocytes in our study were not done. We could not comprehensively evaluate the change of immune function for the patients. And further studies are required to further elucidate the immune mechanisms.

Importantly, we found that the elderly patients had poorer short-term prognosis than nonelderly patients, especially patients with severe GBS. This finding was further proved by the multivariate regression analysis, which identified old age and lower MRC score at nadir were independent risk factors for poor short-term prognosis in severe GBS patients. This result was consistent with previous studies which found that old age was a factor in predicting adverse prognosis of GBS.^[10–12,20,21] The poorer short-term prognosis of the elderly patients in our study might be due to the higher incidence of complications during hospitalization, such as lymphocytopenia, hyponatremia, hypoalbuminemia, hyperglycemia, and pneumonia. The proportion of hyponatremia in the elderly group was higher than that in the nonelderly group, which is consistent with previous studies.^[22,23] The hyponatremia may be due to the syndrome of inappropriate secretion of antidiuretic hormone, renal salt-wasting syndrome as part of dysautonomia, use of immunoglobulin, etc.^[23] However, we could not confirm the certain cause of hyponatremia in the study because we had not examined the antidiuretic hormone, the urine volume, and blood volume of the patients. And hyponatremia was found to increase the risk of death in a study.^[22] A higher proportion of hypoalbuminemia was also found in the elderly patients, which might increase the probability of infection and existing venous thrombosis.^[24] In addition, a higher proportion of hyperglycemia was found in the elderly patients. A previous study have suggested that diabetes might influence the prognosis of patients with GBS for 3 months.^[25]

As to the severe GBS, except for old age and lower nadir MRC score, the univariate regression analysis showed that lack of antecedent infections, pneumonia, and longer duration of hospitalization were also relevant to poor short-term prognosis in patients with severe GBS. Lack of antecedent infection was

related to poor short-term prognosis, which is consistent with the previous result that lack of antecedent infection is a risk factor for poor short-term prognosis in GBS patients with mechanical ventilation,^[26] as well is consistent with the results of Lin et al^[27] in Taiwan. Pneumonia was related to poor short-term prognosis, which is consistent with the result of a previous study that pneumonia was associated with duration of mechanical ventilation for GBS patients admitted to the intensive care unit.^[28] It may be an reason that the severity of illness prolonged the time of hospitalization, and longer duration of hospitalization might be related to poor short-term prognosis with severe GBS. McKhann et al^[29] found that the clinical features and prognosis of AMAN was different from acute inflammatory demyelinating polyneuropathy in 1993, while in this study we did not find the correlation between the short-term prognosis and the axonal damage (AMAN) in severe GBS patients. Electrophysiological findings might have prognostic relevance, but the results of different studies show variation.^[31] Also serial electrophysiological examinations will find some AMAN are characterized by reversible conduction failure,^[30] and these patients will recover good usually. Further studies are required to investigate the relationship between electrophysiological findings and prognosis in GBS by sequential electrophysiological examinations.

This study has following limitations. It was a retrospective study, especially the prognosis was done mainly on the patients of the hospital, lacking of follow-up observations to study the long-term prognosis. Also the detail data about autonomic nervous system involvement was not recorded in this study. We did not perform microbial or serological analyses in this study so that we could not confirm particular antecedent infection and might omit some subclinical antecedent infections. As we aimed to investigate the clinical features of elderly patients with GBS and we just made a retrospective study in The First Hospital of Jilin University, the incidence of GBS in nearby provinces was not investigated in the current study.

In summary, the elderly patients may have slower disease progression, lower incidence of antecedent infection, higher incidence of abnormal laboratory tests, and poorer short-term prognosis. Further, old age and lower MRC score at nadir are risk factors for poor short-term prognosis in severe GBS.

Acknowledgements

The authors thank the National Natural Science Foundation of China (No. 81271294) for the support.

References

- Wakerley BR, Yuki N. Infectious and noninfectious triggers in Guillain-Barré syndrome. *Expert Rev Clin Immunol* 2013;9:627–39.
- Steiner I, Rosenberg G, Wirguin I. Transient immunosuppression: a bridge between infection and the atypical autoimmunity of Guillain-Barré syndrome. *Clin Exp Immunol* 2010;162:32–40.
- van den Berg B, Walgaard C, Drenthen J, et al. Guillain-Barré syndrome: pathogenesis, diagnosis, treatment and prognosis. *Nat Rev Neurol* 2014;10:469–82.
- Sejvar JJ, Baughman AL, Wise M, et al. Population incidence of Guillain-Barré syndrome: a systematic review and meta-analysis. *Neuroepidemiology* 2011;36:123–33.
- Cheng Q, Wang DS, Jiang GX, et al. Distinct pattern of age-specific incidence of Guillain-Barré syndrome in Harbin, China. *J Neurol* 2002;249:25–32.
- Chen Y, Ma F, Zhang J, et al. Population incidence of Guillain-Barré syndrome in parts of China: three large populations in Jiangsu province, 2008–2010. *Eur J Neurol* 2014;21:124–9.
- Winner SJ, Evans JG. Guillain-Barré syndrome in Oxfordshire: clinical features in relation to age. *Age Ageing* 1993;22:164–70.
- Franca MC Jr, Deus-Silva L, de Castro R, et al. Guillain-Barré syndrome in the elderly: clinical, electrophysiological, therapeutic and outcome features. *Arq Neuropsiquiatr* 2005;63:772–5.
- Peric S, Berisavac I, Stojiljkovic TO, et al. Guillain-Barré syndrome in the elderly. *Journal of the Peripheral Nervous System* 2016;21:105–10.
- González-Suárez I, Sanz-Gallego I, Rodríguez de Rivera FJ, et al. Guillain-Barré Syndrome: Natural history and prognostic factors: a retrospective review of 106 cases. *BMC Neurol* 2013;13:95.
- Durand MC, Porcher R, Orlikowski D, et al. Clinical and electrophysiological predictors of respiratory failure in Guillain-Barré syndrome: a prospective study. *Lancet Neurol* 2012;11:58–63.
- van Koningsveld R, Steyerberg EW, Hughes RA, et al. A clinical prognostic scoring system for Guillain-Barré syndrome. *Lancet Neurol* 2007;6:589–94.
- Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. *Ann Neurol* 1990;27:S21–4.
- Hadden RD, Cornblath DR, Hughes RA, et al. Electrophysiological classification of Guillain-Barré syndrome: clinical associations and outcome. *Ann Neurol* 1998;44:780–8.
- Hughes RA, Newsom-Davis JM, Perkin GD, et al. Controlled trial prednisolone in acute polyneuropathy. *Lancet* 1978;2:750–3.
- Kleyweg RP, van der Meché FG, Schmitz PI. Interobserver agreement in the assessment of muscle strength and functional abilities in Guillain-Barré syndrome. *Muscle Nerve* 1991;14:1103–9.
- Reisin RC, Pocięcha J, Rodríguez E, et al. Severe Guillain-Barré syndrome in childhood treated with human immune globulin. *Pediatr Neurol* 1996;14:308–12.
- Hardy TA, Blum S, McCombe PA, et al. Guillain-Barré syndrome: modern theories of etiology. *Curr Allergy Asthma Rep* 2011;11:197–204.
- Sridharan GV, Tallis RC, Gautam PC. Guillain-Barré syndrome in the elderly. A retrospective comparative study. *Gerontology* 1993;39:170–5.
- Walgaard C, Lingsma HF, Ruts L, et al. Early recognition of poor prognosis in Guillain-Barré syndrome. *Neurology* 2011;76:968–75.
- Soysal A, Aysal F, Caliskan B, et al. Clinico-electrophysiological findings and prognosis of Guillain-Barré syndrome – 10 years' experience. *Acta Neurol Scand* 2011;123:181–6.
- Wang YH, Liu JY. Hyponatremia is a predictor for poor outcome in Guillain-Barré syndrome. *Neurol Res* 2015;37:347–51.
- Hiew FL, Winer JB, Rajabally YA. Hyponatraemia in Guillain-Barré syndrome revisited. *Acta Neurol Scand* 2016;133:295–301.
- Hayama M, Akahani S, Michiba T, et al. Significant factors for surgical site infection: analysis of 203 head and neck surgeries. *Nihon Jibiinkoka Gakkai Kaiho* 2014;117:103–10.
- Bae JS, Kim YJ, Kim JK. Diabetes mellitus exacerbates the clinical and electrophysiological features of Guillain-Barré syndrome. *Eur J Neurol* 2016;23:439–46.
- Wu X, Li C, Zhang B, et al. Predictors for mechanical ventilation and short-term prognosis in patients with Guillain-Barré syndrome. *Crit Care* 2015;19:310.
- Lin JH, Tu KH, Chang CH, et al. Prognostic factors and complication rates for double-filtration plasmapheresis in patients with Guillain-Barré syndrome. *Transfus Apher Sci* 2015;52:78–83.
- Dhar R, Stritt L, Hahn AF. The morbidity and outcome of patients with Guillain-Barré syndrome admitted to the intensive care unit. *J Neurol Sci* 2008;264:121–8.
- McKhann GM, Cornblath DR, Griffin JW, et al. Acute motor axonal neuropathy: a frequent cause of acute flaccid paralysis in China. *Ann Neurol* 1993;33:333–42.
- Uncini A, Kuwabara S. Electrodiagnostic criteria for Guillain-Barré syndrome: a critical revision and the need for an update. *Clin Neurophysiol* 2012;123:1487–95.