Can clinical characteristics predict the length of hospital stay in vestibular neuritis?

Original Article

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Abstract

Objectives: Epidemiological and clinical characterization of the population diagnosed with vestibular neuritis (VN) over a five-year period at a tertiary hospital in Portugal. Evaluate the influence of various characteristics as risk factors for prolonged hospitalization (≥ 8 days).
Study design: Retrospective.
Materials and Methods: Review of clinical records of patients hospitalized and treated between January 2019 and December 2023 with a diagnosis of VN.
Results: Ninety-nine patients were included. The median age was 62±14.3 years, with 57.6% being women. Only 14.1% had a history of Diabetes Mellitus (DM). The median length of hospital stay was 4±3.3 days. 70.7% of patients presented with grade III nystagmus.
Conclusions: The results suggest, at a statistically significant level, that age ≥ 65 years old (p=0.038), history of DM (p=0.017) and grade of nystagmus at admission (p=0.018) may represent predictive factors for hospitalizations lasting ≥ 8 days in patients with VN.

Keywords: vestibular neuritis; unilateral acute vestibulopathy; risk factors; comorbidities; hospitalization.

Introduction

Vestibular neuritis (VN), also known as acute unilateral vestibulopathy, is a peripheral vestibular syndrome characterized by sudden unilateral loss of peripheral vestibular function without any central neurological or auditory symptoms.[1]

Acute vestibular dysfunction has been recognized since the early 20th century and received several names in clinical practice. Earlier, terms like “vestibular neuronitis” and “vestibular neuritis” were commonly used, while more neutral terms such as “vestibular neuropathy” or “acute unilateral vestibulopathy” gained prominence in

the second half of the 20th century.[1] The exact etiology of VN remains controversial. Ischemia and autoimmune mechanisms have been proposed, but the most widely-accepted hypothesis is that it is caused by the reactivation of neurotropic viruses such as herpes simplex virus (HSV) types 1 and 2 and herpes zoster.[1-4] Some histopathologic studies of the vestibular nerve have reported changes in VN similar to those found in viral infections like herpes zoster oticus.[2] Other studies have detected HSV-1 DNA in the vestibular ganglia of two-thirds of the patients who died from other causes using polymerase chain reaction (PCR).[4,5] This confirms that vestibular ganglia may be a site of latent HSV-1 infection and supports the hypothesis of latent viral infection reactivation. However, respiratory viruses responsible for upper respiratory tract infections were also shown to be associated with VN in up to 46% of cases,[2] resulting in the historical term "epidemic vertigo."[6]

VN is one of the most common causes of acute peripheral vestibular dysfunction. A population study identified it as the third most frequent cause, after benign paroxysmal positional vertigo and Ménière's disease.[1,7]

In epidemiologic studies conducted in the Japanese and Croatian populations, men and women appeared to be equally affected. Although VN can occur at any age, it is rare in children and has a higher prevalence in individuals between the ages of 30 and 50 years.[5,8] Regarding its estimated annual incidence, Sekitani et al . reported an incidence of 3.5 cases/100,000 population,[8] while Adamec et al. reported up to 15.5 cases/100,000 population.[5]

In 2022, the Committee for the Classification of Vestibular Disorders of the Bárány Society defined the diagnostic criteria for VN as the onset of moderate to severe vertigo lasting for at least 24 hours, associated with autonomic symptoms such as nausea and vomiting, oscillopsia, postural instability, and a tendency to fall toward the affected side. Objective examination shows spontaneous nystagmus, typically horizontal-torsional and with a fixed direction, which can be aggravated by the removal of visual fixation.[1] The superior division of the vestibular nerve seems to be affected in 55–100% cases, probably due to its smaller diameter and greater length within the bony canal.[2,9] Clinically, this presents as spontaneous horizontal-torsional nystagmus during conjugate gaze, with the fast phase directed away from the affected side and an upbeat component. If the inferior division of the nerve is affected, the nystagmus is downbeat, while involvement of both divisions results in contralateral horizontal-torsional nystagmus.[1,9,10] According to Alexander's law, during the acute phase of VN, the intensity and amplitude of the fast phase of nystagmus increase when the gaze is directed toward the fast phase, decreasing when directed toward the slow phase.[11] The head impulse test (HIT) is usually abnormal on the same side as the affected ear, and patients often exhibit ipsilateral segmental and axial deviations, which are evident on conducting the modified Romberg and Fukuda tests.[1,12,13] Otoscopy and audiometry are typically normal.[1]

VN is primarily a diagnosis of exclusion, although ancillary diagnostic methods can reveal vestibular dysfunction. Videonystagmography (VNG) shows a reduced or absent response on the affected side, except in cases where only the inferior division of the vestibular nerve is affected. Vestibular evoked myogenic potentials (VEMPs) can help in identifying which division is affected; in superior division involvement, ocular VEMPs are abnormal and cervical VEMPs remain normal, whereas in inferior division involvement, the opposite pattern is observed. Video-HIT (v-HIT) demonstrates reduced vestibulo-ocular reflex (VOR) gain of less than 0.7, with a difference of at least 0.3 between the two sides, and helps to differentiate between peripheral and central lesions because a normal v-HIT is inconsistent with VN.[1]

Acute VN often improves within the first three days after symptom onset, followed by progressive improvement over the next few weeks.[1,10] Depending on the severity of the

disease, some patients recover spontaneously, while others require outpatient medication or hospitalization. The treatment of VN consists of three main components: symptomatic therapy, specific therapy, and vestibular rehabilitation.[11] Symptomatic therapy includes fluid replacement, antiemetics (e.g., metoclopramide or ondansetron) for patients with total food intolerance, and vestibular suppressants (e.g., betahistine, dimenhydrinate, or diazepam).[11] Although a viral etiology is widely accepted in the scientific community, antiviral agents such as valacyclovir have not been shown to influence disease activity.[14] Use of corticosteroid therapy in VN remains controversial. Some studies have reported no evidence of clinical improvement,[15] while others found a short-term improvement in canal paresis as demonstrated by caloric testing.[16] Vestibular rehabilitation is a safe and effective treatment for unilateral peripheral vestibular dysfunction, with medium-term symptom relief and functional improvement.[12] This study aimed to characterize the population diagnosed with VN at a tertiary hospital in Portugal over a 5-year period, along with evaluating the role of various clinical characteristics as risk factors for prolonged hospitalization (≥ 8 days)

Materials and methods

In this retrospective cohort study, the electronic clinical records of patients diagnosed with VN and admitted to the otolaryngology (ORL) department of a tertiary hospital in Portugal between January 2019 and December 2023 were analyzed. The inclusion criterion was patients diagnosed with the International Classification of Diseases - Tenth Revision (ICD-10) diagnostic code H81.2: Vestibular Neuronitis. The exclusion criteria were concomitant otological signs and symptoms, such as hypoacusis, otorrhea, or tinnitus; or neurological signs. Epidemiological (sex, age, month, and quarter of onset) and clinical (laterality, recurrence, comorbidities, and degree of nystagmus on admission and discharge) parameters were analyzed. Additionally, the diagnostic methods used; VOR gain on admission for patients undergoing v-HIT within the first four weeks after onset; treatment; and length of hospital stay were recorded. The time to reassessment and presence of symptoms at that point were also analyzed. Categorical variables were reported as frequencies and percentages, while quantitative variables were described as medians and standard deviations.

Patients were categorized based on the length of stay as less than eight days or eight days and longer (prolonged stay). A factorial analysis of variance (ANOVA) was conducted to evaluate the impact of epidemiological and clinical characteristics on prolonged stay. Statistical analysis was performed using the IBM SPSS® software for Windows® version 26.0 (IBM Corp., *2017,* Armonk, NY, USA). Statistical significance was set as $p \leq 0.05$.

Results

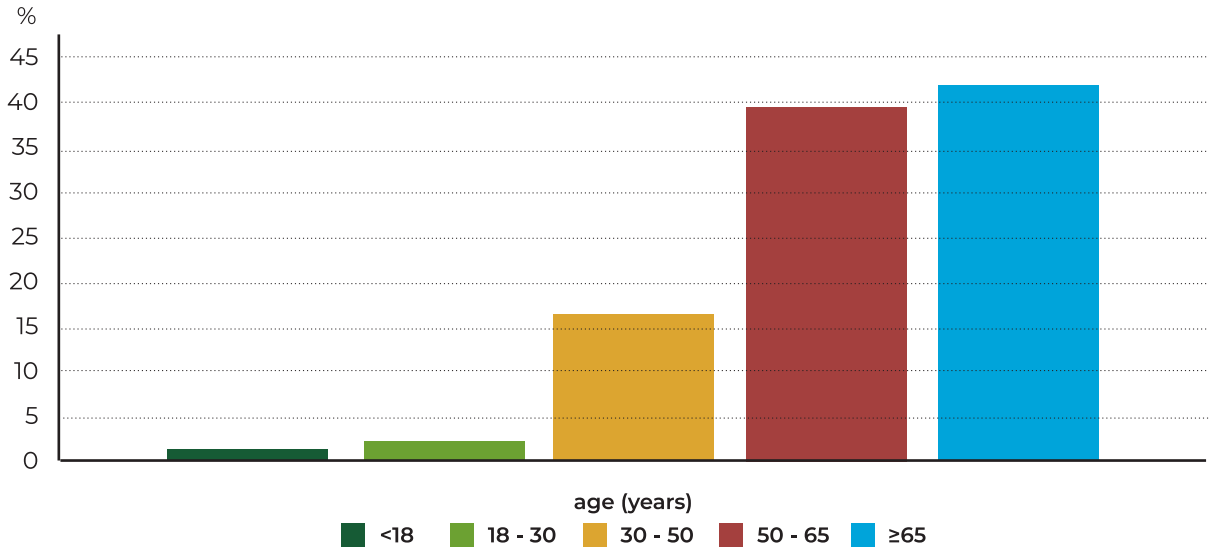
The study included 99 patients diagnosed with VN who were admitted to the ORL department for medical treatment between January 2019 and December 2023. The majority of the patients were women (n = 57, 57.6%) (Table 1). The median age at diagnosis was 62 ± 14.3 years (13–89 years), and 41 (41.4%) patients were over the age of 65 years (Graph 1). Age >65 years was significantly associated with the need for prolonged hospitalization ($p = 0.038$). The highest incidence of VN episodes was observed in September, October, and November (34.3%), with November and December recording the highest number of VN episodes (Graph 2 and Table 2).

Regarding laterality, most episodes were reported on the left side (n = 52, 52.5%). Most patients (90.9%) had no history of otologic conditions. Recurrence was recorded in seven (7.1%) patients. With regard to personal history, 51 (51.5%) patients had hypertension (HT), 14 (14.1%) had diabetes mellitus (DM), 40 (40.4%) had dyslipidemia, 21 (21.2%) had anxiety or depression, and seven (7.1%) had coronary disease. There was a statistically significant relationship between DM and prolonged

Table 1
Demographic characteristics

Demographic characteristics	N	%	p-value
Sex			0.215
Male	42	42.4	
Female	57	57.6	
Age subgroup			0.038
< 18 years	1	1	
18–30 years	2	2	
30–50 years	16	16.2	
50–65 years	39	39.4	
≥ 65 years	41	41.4	
Total	99	100%	

Figure 1
Distribution by age



hospitalization (p = 0.017). No statistically significant relationships were observed between the other parameters (Table 3).

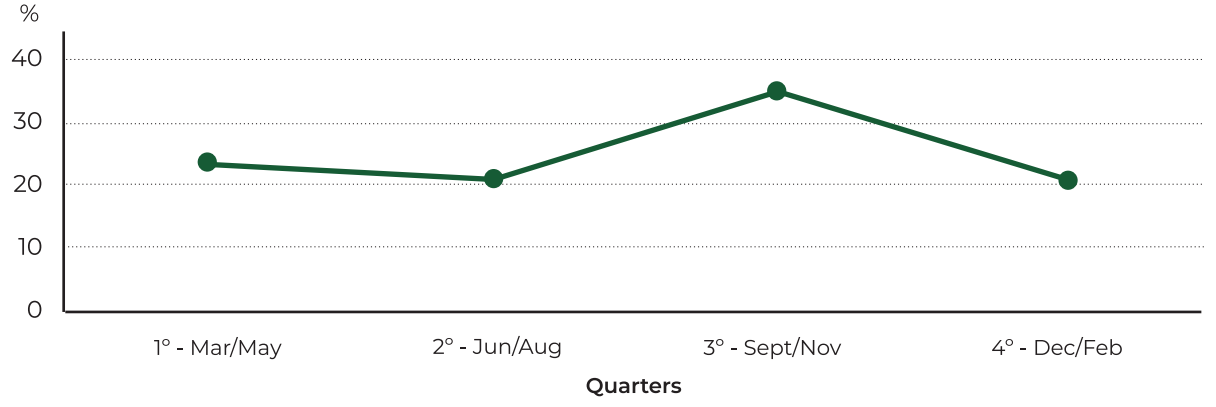
On objective examination, all patients exhibited spontaneous horizontal-torsional nystagmus. The majority had grade III nystagmus according to Alexander's laws (n = 70, 70.7%), while 25 (25.3%) patients had grade II, and only four (4%) had grade I (Table 4). The degree of nystagmus on admission was significantly associated with the need for prolonged hospitalization (p = 0.018). Most patients (n = 64, 64.6%) were assessed by a neurologist. Most patients (n = 77, 77.8%) needed ancillary imaging tests in the emergency room or during hospitalization, with 61 (61.6%) undergoing computed tomography (CT) and 16 (16.2%) undergoing both CT and magnetic resonance imaging (MRI). Diagnosis was based on the clinical findings in only 22 (22.2%) patients. The 29 patients who underwent *v*-HIT in the first four weeks after onset demonstrated a median VOR gain of 0.4797 ± 0.05 in the affected ear, which was not related to prolonged hospitalization (p = 0.605) (Table 5).

The median length of hospital stay was 4 ± 3.3 days, with 81 (81.8%) patients hospitalized for <8 days and 18 patients hospitalized for ≥8

Table 2
Seasonal characteristics

Demographic characteristics	N	%	p-value
Month			
January	7.0	7.07	
February	5.0	5.05	
March	9	9.09	
April	5	5.05	
May	10	10.1	
June	6	6.06	
July	7	7.07	
August	5	5.05	
September	11	11.11	
October	10	10.10	
November	12	12.12	
December	12	12.12	
Quarter			0.810
1st - March, April, May	23	23.2	
2nd - June, July, August	21	21.2	
3rd - September, October, November	34	34.3	
4th - December, January, February	21	21.2	
Total	99	100%	

Figure 2
Seasonal distribution of vestibular neuritis episodes



days. Regarding treatment, 97% of patients were treated with intravenous corticosteroid therapy (dexamethasone 4 mg once or twice a day), 78.8% with benzodiazepine, and 77.8% with betahistine (16 mg three times a day) (Table 6 and Graph 3). None of these therapies or combinations of therapies were significantly associated with prolonged hospitalization.

At discharge, 46.5% of patients had no nystagmus, 45.5% had grade I nystagmus, and only 8.1% had grade II nystagmus (Table 4). The median time to follow-up was 26 ± 26.4 days (6–150 days), and 48.5% of patients were asymptomatic at that time (Table 6).

Table 3
Clinical characteristics

Demographic characteristics	N	%	p-value
Laterality			0.425
Left	47	47.5	
Right	52	52.5	
ORL comorbidities			0.643
Yes	9	9.1	
No	90	90.9	
Recurring events			0.052
Yes	7	7.1	
No	92	92.9	
Personal history			
HT	51	51.5	0.887
DM	14	14.1	0.017
Dyslipidemia	20	40.4	0.701
Anxiety/depression	21	21.2	0.454
Coronary disease	7	7.1	0.784
Total	99	100%	

Table 4
Characteristics of nystagmus

	N	%	p-value
Degree of nystagmus on admission			0,018
0	0	0	
I	4	4	
II	25	25,3	
III	70	70,7	
Degree of nystagmus at discharge			0,970
0	46	46,5	
I	45	45,5	
II	8	8,1	
III	0	0	
Total	99	100%	

Discussion

The analysis of demographic characteristics revealed some differences in the sex and age distribution of patients with VN compared to the findings in the existing literature.[1,5,17] Adamec et al. analyzed 79 patients with VN and found no significant difference between the sexes (men-to-women ratio of 1:1.1). In contrast, our study identified a higher incidence of VN in women (n = 57, 57.6%).[5] According to previous studies, the age group most often affected by VN is 30–50 years, which represented only 16.2% of patients in our study.[1,5,8,17] The median age at diagnosis

Table 5
Diagnostic characteristics

	N	%	p-value
Neurologic evaluation			
Yes	64	64,6	
No	35	35,4	
Imaging test	N	%	
CT	61	61,6	
CT + MRI	14	14,2	
No	22	22,2	
v-HIT	N	VOR	0,605
Yes	29	0,4797±0,05	
No	70	-	
Total	99	100%	

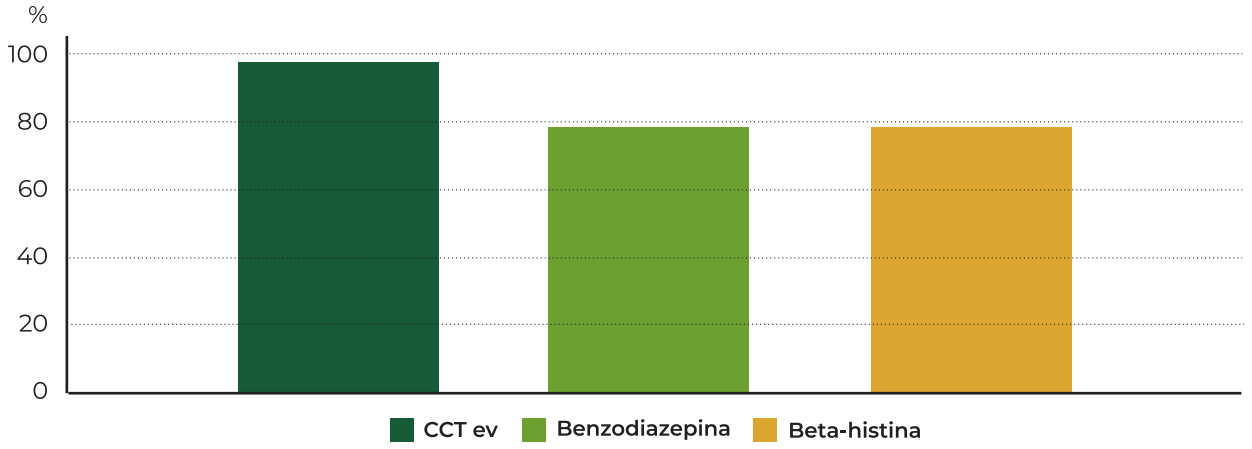
Table 6
Characteristics of clinical progression

			p-value
Length of hospital stay	N	%	
< 8 days	81	81.8	
≥ 8 days	18	18.2	
Therapy			
IV corticosteroids	97	97	0.857
Benzodiazepine	78	78.8	0.618
Betahistine	77	77.8	0.214
Time to follow-up			
< 1 month	61	61.6	
1–2 months	20	20.2	
> 2 months	12	12.1	
No follow-up	6	3	
Symptoms on reevaluation	N	%	
No	48	48.5	
Yes	45	45.5	
No follow-up	6	3	
Total	99	100%	

was 62 ± 14.3 year, and 41.4% of patients were older than 65 years. Our study included only patients who required hospitalization, which may have resulted in a selection bias due to their older age. A study demonstrated that vestibular function declines with age due to the loss of neurons and hair cells in the otolith organs and semicircular canals.[18] Patients older than 65 years, due to a preexisting vestibular deficit, may require more time to recover from residual symptoms of vertigo and imbalance during a VN crisis and may be at greater risk of prolonged hospitalization (p = 0.038).

Theoretically, a viral infection of the upper respiratory tract may be associated with VN.[2] However, an increase in the seasonal incidence of VN has not been reported n the literature. In our study, the fall quarter

Figure 3
Treatments used



(September, October, and November) had the highest incidence of VN (34.3%), with November and December being the months with the highest number of VN episodes (n = 12, 12.1%), although no statistical significance was identified, corroborating with the results in the literature.[1,5,19]

Regarding laterality, previous studied have not reported a predominant side, which is consistent with the results of this study, where both sides were equally affected. Most patients (90.9%) had no history of otological conditions such as infection, tinnitus, or hearing loss. The recurrence rate reported in the literature ranges between 1.9%[20] and 10.7%,[21] which aligns with the rate of 7.1% found in this study.

As for the personal history, 51.5% of patients had HT, 14.1% had DM, 40.4% had dyslipidemia, 21.2% had a history of anxiety or depression, and 7.1% had coronary disease. These results are comparable to those reported by Coronel-Touma et al., who analyzed the prevalence of cardiovascular risk factors (CVRF) in a population of Spanish patients with VN. The existing literature indicates a relationship between CVRF (HT, DM, and dyslipidemia) and a history of depression in patients with VN. However, the results were not consistent .[1,5,17]

Our study revealed a statistically significant relationship between DM and the need for prolonged hospitalization (p = 0.017), while the other parameters showed no association with the need for prolonged hospitalization. The relationship between DM and the audio-vestibular system has been investigated for over a century. Most studies on individuals with DM have reported slow and progressive sensorineural hearing loss, and a significant impact of DM on the peripheral auditory system. In contrast, other studies have demonstrated no impact of DM on vestibular function. DM affects the microvasculature and neurons at a histopathological level, but its effects on the inner ear have not been confirmed in animal or human models. DM can affect the retina, kidneys, and skeletal muscles, leading to retinopathy, nephropathy, and neuropathy. These effects may be mediated by oxidative and nitrosative stress due to hyperglycemia, which damages the microvascular endothelium and cellular DNA, and may lead to DM-associated complications, such as vascular ischemia in neural tissue resulting in atrophy and demyelination.[22,23] These findings support the theory of microvascular ischemia as the cause of VN, classifying patients with DM as a high-risk group.

In the objective examination, all patients exhibited spontaneous, horizontal-torsional nystagmus, with 70.7% presenting grade III nystagmus according to Alexander's laws. The severity of nystagmus at admission was correlated with the need for prolonged hospitalization (p = 0.018). It is worth noting

that this study included only hospitalized patients, which may have biased the sample toward patients with VN of higher severity.

Most patients (64.6%) were evaluated by a neurologist, with 77.8% undergoing imaging tests and 22.2% receiving only a clinical diagnosis. VN diagnosis is a process of exclusion, and patients with atypical symptoms such as cardiovascular risk factors, neurological signs, or headaches for more than 48 hours should undergo imaging examination (CT and/or MRI) to rule out central vertigo.[3,11]

In patients who underwent *v*-HIT within the first four weeks of symptom onset, the VOR gain in the affected ear had a median value of 0.4797 ± 0.05, and was not associated with prolonged hospitalization.

A limitation of this study is that only a subset of patients (n = 29) had their vestibular function measured during the acute phase. Considering that a definitive decline in VOR is a diagnostic criterion for VN, it is crucial to evaluate vestibular function in all patients, ideally within the first few days after the onset of symptoms.[1,17]

The median length of stay was 4 ± 3.3 days, with 18.2% patients requiring hospitalization for a minimum of 8 days. The treatment of VN comprises symptomatic therapy (fluid therapy, antiemetics, and vestibular inhibitors), etiological therapy (corticosteroids, antivirals, and vasodilators), and vestibular rehabilitation.[11,12] In our study, 97% patients were treated with intravenous corticosteroid therapy (dexamethasone 4 mg once or twice a day), 77.8% with betahistine (16 mg three times a day), and 78.8% with benzodiazepine. As observed in the literature, no correlation was identified between the treatments or treatment combinations and the need for prolonged hospitalization.[15] During hospitalization, all patients began vestibular rehabilitation as soon as they were able to stand. Consequently, the impact of this treatment on the length of hospital stay was not assessed, despite its reported benefits in the literature.[12] No patients received antiviral treatment.

At discharge, 46.5% patients exhibited no nystagmus. According to the literature, acute symptoms of VN typically improve within 3–7 days, while recovery from the vertigo sensation may take several weeks.[3,12] Okinaka et al. reported a subjective improvement of approximately 40% after three months. In our study, 48.5% of the patients were asymptomatic after a median of 26 ± 26.4 days.[24]

Conclusion

VN is one of the most common causes of vertigo, often requiring hospitalization for symptomatic and etiological treatment, with or without vestibular rehabilitation. Our results revealed that in patients aged ≥ 65 years, a history of DM and grade III nystagmus at admission may be predictors of hospital stay lasting at least 8 days. The other parameters analyzed showed no association with the length of hospital stay. While the presence of CVRF and age have been identified as risk factors for delayed recovery in VN in previous studies, no relationship between DM and degree of nystagmus has been established yet. Additionally, to our knowledge, no previous studies have analyzed the relationship of clinical factors with the length of hospital stay in VN.

Conflict of Interests

The authors declare that they have no conflict of interest regarding this article.

Data Confidentiality

The authors declare that they followed the protocols of their work in publishing patient data.

Human and animal protection

The authors declare that the procedures followed are in accordance with the regulations established by the directors of the Commission for Clinical Research and Ethics and in accordance with the Declaration of Helsinki of the World Medical Association.

Privacy policy, informed consent and Ethics committee authorization

All the processed data were based in published reports that fulfilled privacy policy and ethical considerations.

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Scientific data availability

There are no publicly available datasets related to this work.

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