

Higher-level gait disorders: a population-based study on prevalence, quality of life, depression and confidence in gait and balance

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ABSTRACT

Background Higher-level gait disorders (HLGDs) are slow, unsteady neurological GDs in older people. GDs can reduce quality of life (QoL) and cause depression. This has not been investigated in HLGD even though some HLGD causes are treatable, potentially affecting associated problems. We aimed to investigate gait and balance confidence, depressive symptoms and QoL in HLGD.

Methods In a population (n=3769, 65–84y), 798 reported gait impairment (questionnaire) and were clinically examined together with 249 age- and sex-matched controls. Gait property groups were formed: 'HLGD', 'other neurological GD', 'non-neurological GD' or 'no GD'. Swedish Falls Efficacy Scale (FES(S)), Modified Gait Efficacy Scale (mGES), Euro Quality of Life 5-Dimension 5-Level index, Euro Quality of Life Visual Analogue Scale (EQ VAS) and Geriatric Depression Scale-15 (GDS-15) were compared.

Results In the general population, 38% had GDs, of which 16% (n=87/561) were HLGDs, giving an HLGD prevalence of 5.8%; 26% (n=145/561) were other neurological GDs; and 59% (n=329/561) non-neurological GDs. HLGD had more depressive symptoms than non-neurological GD and no GD (GDS-15 HLGD, 3.9 ± 3.4 ; non-neurological GD, 2.5 ± 2.8 ; no GD, 1.4 ± 2.0 ; $p < 0.05$), lower EQ VAS (HLGD, 63 ± 17 ; non-neurological GD, 71 ± 18 ; no GD, 82 ± 14 ; $p < 0.001$), lower gait confidence (mGES HLGD, 60 ± 22 ; non-neurological GD, 74 ± 21 ; no GD, 90 ± 13 ; $p < 0.001$) and lower balance confidence (FES(S) HLGD, 93 ± 32 ; non-neurological GD, 111 ± 25 ; no GD, 124 ± 13 ; $p < 0.001$).

Conclusions HLGDs are common and associated with reduced QoL, reduced confidence in gait and balance, and depressive symptoms, emphasising awareness of mental health among older people with slow unsteady gait.

INTRODUCTION

In neurological¹ and outpatient clinics, older people often present with a slow, symmetrical, unsteady gait of unknown cause. Sometimes the cause is identified, but often, the condition is considered normal ageing and does not elicit further investigation. Some seek medical care, while others accept the impairment as an untreatable phenomenon of age.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Higher-level gait disorder (HLGD) is a category of disorders characterised by a slow, symmetrical, broad-based gait, with a previously reported prevalence of 2% in older people. Several aetiologies exist, including normal pressure hydrocephalus, nascler parkinsonism and progressive supranuclear palsy, but many patients and clinicians might consider this clinical gait syndrome part of normal ageing, and therefore, it does not elicit further investigation in many situations.

WHAT THIS STUDY ADDS

⇒ One problem might be that the concept of HLGDs is fairly unknown to physicians, along with their impact on individuals. This large population-based study investigates the prevalence of HLGDs and their effect on older individuals through evaluation of quality of life, symptoms of depression, and confidence in gait and balance among older people with HLGDs, compared with age and sex matched controls from the general population.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Since this study shows that HLGD was more common than previously reported and that affected individuals have more depressive symptoms, lower QoL and lower confidence in gait and balance than individuals with normal gait and non-neurological gait disorders, it should raise awareness of HLGD among clinicians and researchers as an important clinical syndrome to consider in future clinical work and research.

However, gait disorders (GDs) may cause falls,^{2,3} fear of falling,⁴ injuries⁵ and increased mortality,⁶ and some GDs that may seem age-related are caused by treatable neurological syndromes.^{7–9}

One-third of people >60 years have a GD, about half have neurological causes.^{10,11} Categorisation of GDs through a topical diagnosis such as due to dysfunction in the higher-

middle- or lower-levels of the nervous system has been suggested.⁹ By definition, higher-level GDs (HLGDs) cannot be explained solely by lower- or middle-level (peripheral nervous system, corticospinal tract, cerebellum and basal ganglia) dysfunction; they are unsteady, symmetrical and often broad-based.⁹ Examples of HLGDs are normal pressure hydrocephalus (NPH), progressive supranuclear palsy and vascular parkinsonism, diseases that may be difficult to diagnose in an early stage and often remain undiagnosed.⁹ Even though HLGD is common (2% of older people^{10 11} and potentially treatable), it is surprisingly poorly and insufficiently described in the literature.⁹

Knowledge about the effects of HLGD on patients' well-being is needed, both to improve their care and to highlight the importance of awareness of symptoms indicative of treatable conditions. Slow walking speed has been associated with institutionalisation and high mortality rates^{12 13} and abnormal gait with reduced confidence in one's balance (falls efficacy), which in turn is associated with depressive symptoms and low quality of life (QoL).⁴ Since reduced gait velocity is a cardinal feature of HLGD, it is reasonable to hypothesise that these problems affect individuals with HLGD; however, population-based studies investigating these important matters are currently lacking.

In this population-based study, one-third of the older population in a Swedish town was invited for participation. The objective was to determine the prevalence of HLGD in the normal population and whether individuals with HLGD have lower QoL, more depressive symptoms and lower confidence in gait and balance than older people of the population.

METHODS

This study is part of the 'Ventriculomegaly and gait disturbance in the senior Population in the Region of Vasterbotten' (VeSPR) study, which was a prospective population-based case-control study conducted in 2018–2020. Questionnaires were mailed to one-third of the older population in a Swedish middle-sized town to identify individuals with subjective gait impairment. These, and a control group without subjective gait impairment, were invited for clinical examinations. Thereafter, everyone was assigned to one of four groups according to the cause of the GD (or lack thereof).

Study population

Figure 1 shows patient and control flow. Table 1 shows demographics.

One-third¹⁴ of the individuals aged 65–84 years who lived in the area were randomly selected from a population registry¹⁵ and invited to participate through a mailed-out questionnaire regarding self-perceived gait impairments (n=6412). Individuals reporting 'subjective gait impairment' (definition in table 2) through the questionnaire were invited for examination by a physician

(n=1510). For each subject in the 'HLGD' group, four age- and sex-matched controls were invited (n=513), randomly selected from the survey respondents who had reported no subjective gait impairment. All who accepted the invitation for clinical examination (798 with subjective gait impairment and 249 controls) were classified according to their clinical gait status: (1) 'HLGD', (2) 'other neurological gait disorder', (3) 'non-neurological gait disorder' and (4) 'no gait disorder'.

Exclusion criteria for clinical examinations were death at the time of the examination.

Questionnaire

Researchers and physicians specialised in neurological mobility impairments designed the questionnaire. Its purpose was to gather demographic information, screen for and characterise GDs. It was tested on 144 participants, thereafter, revised and used on the entire cohort (response rate: 59% n=3769/6467; see table 1). Participants answered on paper or online, by themselves or with the aid of a relative or home care personnel. Maximum walking distance, pooled walking distance on a normal day, subjective walking speed in relation to others of the same age, progression of gait impairment over time (fast or slow) and duration of GDs were assessed. Characteristics such as freezing or shuffling, difficulties turning around, need for walking device, balance impairment and vertigo were noted. The individual's knowledge about the cause of their GD was obtained. One reminder was sent to non-responders.

Falls efficacy and gait efficacy

The questionnaire incorporated two well-established scales: the Swedish modification of the Falls Efficacy Scale (FES(S))¹⁶ and the Modified Gait Efficacy Scale (mGES).¹⁷ Both assess self-efficacy: FES(S) in avoiding falls in 13 daily activities and mGES in 10 walking tasks. Both rate confidence on a numerical analogue scale; in FES(S), it ranges from 0 (not confident at all) to 10 (completely confident) with a total score range of 0–130, and in mGES, it ranges from 1 to 10 (total score range: 10–100).

Definition of gait disorders

Following real-time observation of gait patterns, the physician classified them (see section study population), based on clinical appearance and suspected underlying cause (table 3). When multiple causes were identified, the one considered the most influential contributor was used for analysis in this study. To reduce the risk of inter-rater disagreement, all causes were reviewed after the visit by at least two physicians, through inspection of the gait video recording and all clinical data. In cases where disagreement occurred, observed signs and symptoms were discussed until a sign-based decision could be reached in agreement.

HLGD was defined as an abnormal, symmetrical gait, with reduced cadence, and/or shuffling gait, and/or

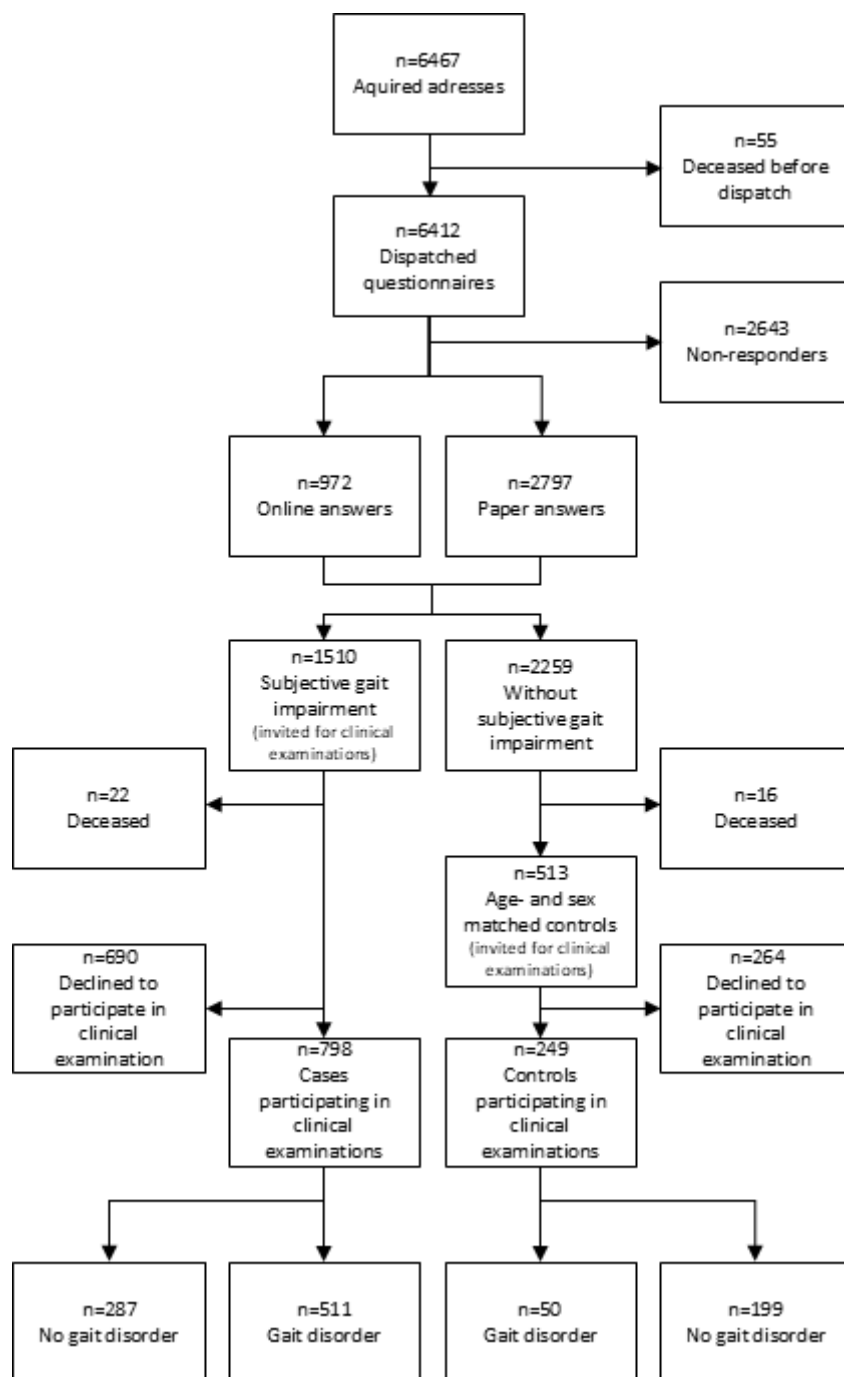


Figure 1 Patient and control recruitment flow chart. Number of dispatched questionnaires, participants invited for clinical evaluations and participants with subjective and objective gait disorder along with non-responders and deceased.

reduced balance when turning, with a medical history and physical examination that could not elicit an apparent underlying lower- or middle-level dysfunction.⁹

Clinical examinations

A physician obtained a comprehensive medical history and performed a neurological and musculoskeletal examination, according to a standardised protocol. Muscle strength and tone, signs of muscular atrophy, deep-tendon reflexes, parkinsonian signs including tremor, bradykinesia, rigidity, coordination and signs of cerebellar ataxia in lower extremities were examined. The sensory system was

evaluated through differentiation of sharp and dull pain and sensibility for vibration (128 Hz tuning fork). Balance and gait functions were tested with the Tinetti balance test¹⁸ and the Timed Up and Go test (TUG)¹⁹ at maximum speed. Starting in a halted position, maximum gait velocity (m/s; mean of three attempts on a 10-metre distance) was measured. A gait video recording including sideways and frontal views, TUG, and 180- and 360-degree turns was made. Gait patterns were described using the terms broad-based, shuffling, reduced cadence, impaired balance when turning, symmetrical, asymmetrical or hemiparetic.

Table 1 Demographics and questionnaire response rate

Invited to answer questionnaire, n	6412
Age, years, mean±SD (range)*	74±5 (65–86)
Female sex, n (%)	3380 (53)
Questionnaire answers, n (%)	3769 (59)
Paper, n (%)	2797 (74)
Online, n (%)	972 (26)
Demographics of participants invited to answer the questionnaire and questionnaire response rates.	
*Age at time for questionnaire dispatch.	
n, number of individuals; SD, Standard Deviation.	

No additional diagnostic tests or radiological examinations aimed to find specific diagnoses were performed in this stage of the study.

Depressive symptoms and quality of life

At the visit to the clinic, depressive symptoms were assessed with the Geriatric Depression Scale 15 (GDS-15),²⁰ ≥5 points were regarded as ‘symptoms of depression’. QoL was assessed with the Euro Quality of Life 5 Dimension 5 Level (EQ5D5L) index, measuring QoL across five dimensions (mobility, usual activities, pain/discomfort, self-care and anxiety/depression) using five levels (no, slight, moderate, severe or extreme) and the Euro Quality of Life Visual Analogue Scale (EQ VAS).²¹

Data management

Data were collected and managed using Research Electronic Data Capture tools hosted at Umeå University.^{22 23}

Table 2 Definition of subjective gait impairment

Question	Subjective gait impairment
How fast do YOU YOURSELF think that you walk, in comparison to others your own age?	‘A little bit slower’ ‘Much slower’
How do YOU YOURSELF perceive your gait- and balance abilities?	‘I have a gait impairment’ ‘I have a gait impairment that debuted in a slowly progressing manner’
Three questions were answered on a scale ranging from 1 (not confident at all) to 10 (completely confident), responding to the question ‘How much confidence do you have that you would be able to safely...’	
‘...Walk on grass’	Scored ≤7
‘...Walk over an obstacle in your path’	Scored ≤7
‘...Walk a long distance such as 1 kilometre’	Scored ≤7
Subjective gait impairment was defined as present if any of the answers listed in the ‘subjective gait impairment’ column was given in the questionnaire used for inclusion.	

Table 3 Type of gait disorders in the population

Type of gait disorder*	Number of individuals
Neurological gait disorder	232
Higher-level gait disorder	87
Unknown cause	76
Parkinson's disease/parkinsonian disorders	5
Idiopathic normal pressure hydrocephalus	2
Other†	4
Other neurological gait disorder	145
Polyneuropathy	47
Stroke	37
Myelopathy/radiculopathy	23
Parkinson's disease/parkinsonian disorders	9
Other‡	29
Non-neurological gait disorder	329
Arthrosis	226
Musculoskeletal disorder	65
Vertigo/vestibular dysfunction	10
Arthritis	6
Other‡	22
No gait disorder	486
Number of individuals within each gait disorder group and diagnoses causing gait disorders determined through clinical examination by a physician.	
*Type of gait disorder determined through clinical examinations.	
†One case per diagnosis.	
‡Five cases or less per diagnosis.	

Ethics

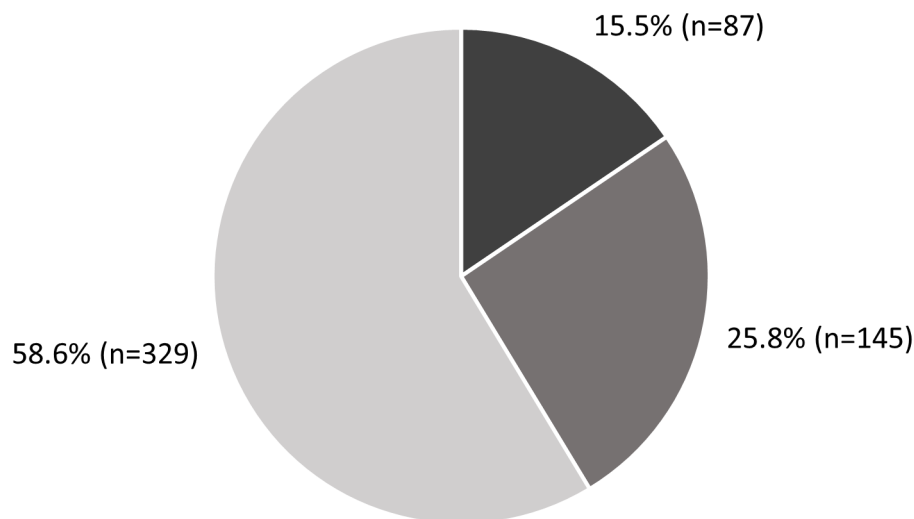
The Regional Ethical Review Board in Umeå (Dnr 2017/335-31) approved the study. It was registered at clinicaltrials.gov (NCT05204745) and conducted in accordance with the Declaration of Helsinki. Written and oral informed consent was obtained from participants.

Statistical analyses

IBM SPSS Statistics 27 (IBM Corp. 2020) was used. Significance level was $p \leq 0.05$. Participants with missing data were excluded from the analyses.

A two-tailed independent samples t-test was used for comparisons of means between the groups with and without GDs. One-way analysis of variance with post hoc Games-Howell analysis was used for comparisons of means between multiple groups. The χ^2 test and Fisher's exact test were used for comparisons of frequencies between dichotomous variables.

The prevalence of GDs in the whole population that answered the questionnaire was estimated based on the proportion of true positive (TP%) questionnaire answers among participants with subjective gait impairment and



■ Higher-level gait disorder ■ Other neurological gait disorder ■ Non-neurological gait disorder

Figure 2 Prevalence of gait disorders in the study population. Pie chart of the prevalence of higher-level gait disorders, other neurological gait disorders and non-neurological gait disorders within the study population, presented in percent and number of individuals.

the proportion of false negative (FN%) answers among participants without subjective gait impairment per number of survey respondents:

$$\text{GD Prevalence} = (\text{TP}\% \times 1510 + \text{FN}\% \times 2259) / 3769.$$

The prevalence of HLGD in the population was estimated through multiplication of the proportion of HLGD among participants with GDs with the GD prevalence:

$$\text{HLGD prevalence} = \text{proportion HLGD} \times \text{GD Prevalence}.$$

The 95% CIs of the GD prevalence and HLGD prevalence were calculated based on the estimated effective sample sizes used to determine the calculated prevalence. Estimated effective sample size = (identified number of GD or HLGD cases) / (estimated GD or HLGD prevalence).

The accuracy of the questionnaire to detect clinically verified GDs was calculated:

$$(\text{TP}\% \times \text{number with subjective gait impairment}) + (\text{TN}\% \times \text{number with no subjective gait impairment}) / 3769$$

RESULTS

Prevalence

In the population, the GD prevalence was 38% (95% CI 36 to 40), and the HLGD prevalence was 5.8% (95% CI 4.6 to 7.0%).

Based on clinical appearance and underlying cause, individuals with GDs were classified in the following groups: 16% (n=87) HLGDs, 26% (n=145) other neurological GDs and 59% (n=329) non-neurological GDs (figure 2). Thus, 41% (n=232) had a neurological cause (ie, HLGD or other neurological GD).

Of those with HLGD, 87% (n=76/87) had an unknown cause, 64% (n=56/87) of HLGD were male and 36% (n=31/87) were female (OR for sex: 2.1 95% CI 1.3 to 3.2, p=0.002).

Quality of life

The QoL scores are shown in figure 3A,B. In summary, HLGDs were more associated with low QoL than non-neurological GDs and normal gait.

Individuals with HLGD had lower EQ VAS scores (figure 3A) and EQ5D5L index (figure 3B) than those without GDs (p<0.001). EQ VAS scores were lower in HLGDs than in non-neurological GDs (p<0.001). There was no difference in EQ VAS scores between HLGDs and other neurological GDs (p=0.724) and no difference in EQ5D5L index between HLGDs and other neurological GDs or non-neurological GDs (HLGD vs other neurological GD p=0.991; HLGD vs non-neurological GD p=0.273).

Compared with individuals without GDs, those with any type of GD had lower QoL (EQ5D5L index mean±SD, 0.693±0.184, n=559 vs 0.840±0.126, n=485 p<0.001; EQ VAS mean±SD, 68±18, n=558 vs 82±14, n=485 p<0.001).

Symptoms of depression

Differences in GDS-15 scores are shown in figure 4. In summary, HLGDs were more associated to depressive symptoms than non-neurological GDs and normal gait.

Individuals with HLGDs had higher GDS-15 scores than those with non-neurological GDs and no GDs (p<0.05). No difference was observed between HLGDs and other neurological GDs (p=0.98).

Among individuals with HLGD, 33% (n=28) had symptoms of depression (GDS-15 score ≥5): an equivalent prevalence to that observed in other neurological GDs (28%, n=40; p=848) but higher than the prevalence in non-neurological GDs and no GDs (non-neurological GD, 17%, n=54, p=0.015; no GD, 7%, n=33, p<0.001).

Individuals with any type of GD had higher GDS-15 scores than those with no GDs (mean±SD, 3.0±3.1,

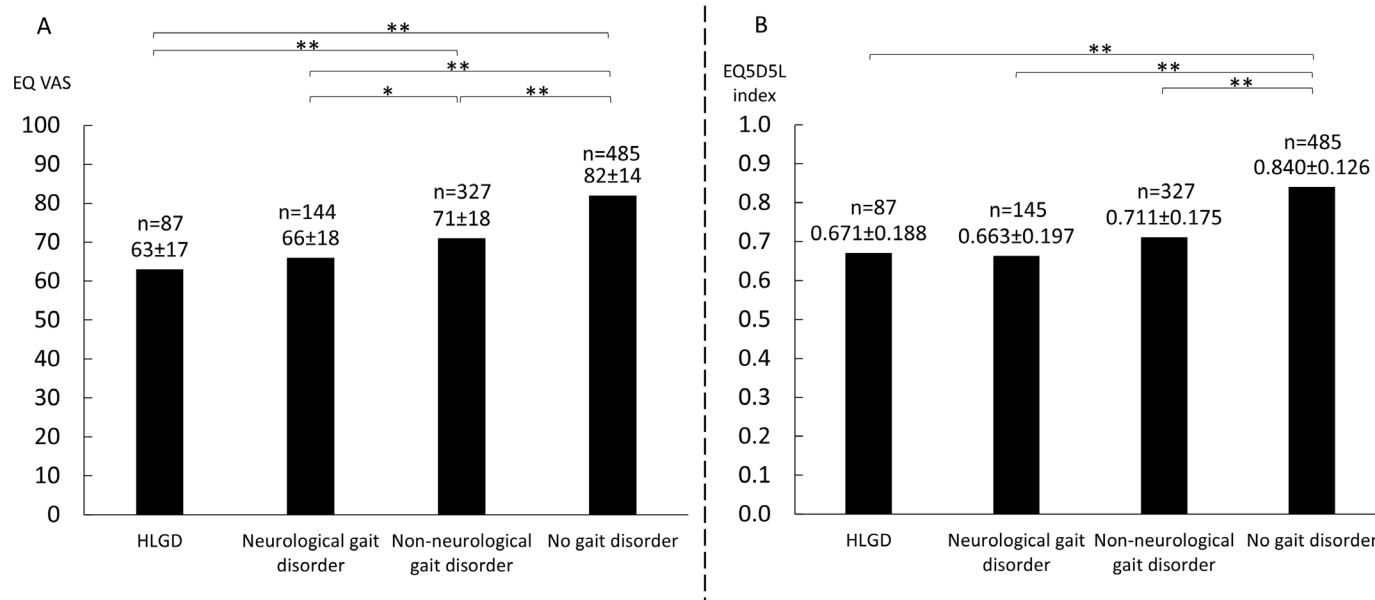


Figure 3 Quality of life. Mean quality of life scores compared between gait disorder groups. The mean±SD and number of participants analysed within each group are indicated above bars. (A) Mean Euro Quality of Life Visual Analogue Scale (EQ VAS) score. (B) Mean Euro Quality of Life 5-Dimension 5-Level Index (EQ5D5L). * $p<0.05$; ** $p<0.001$. HLGD, higher-level gait disorder.

$n=553$ vs 1.4 ± 2.0 , $n=481$; $p<0.001$). Additionally, 22% ($n=123/553$) of individuals with GDs had symptoms of depression, compared with 7% ($n=33/481$) of those without GDs (OR 3.9 95% CI 2.6 to 5.8, $p<0.001$).

Assessments of confidence in balance and gait

FES(S) and mGES scores are shown in figure 5A,B. Individuals with HLGD had lower FES(S) and mGES scores than those with non-neurological GDs and no GDs. There was no difference in FES(S) and mGES scores between HLGDs and other neurological GDs (FES(S), $p=0.796$; mGES, $p=0.432$).

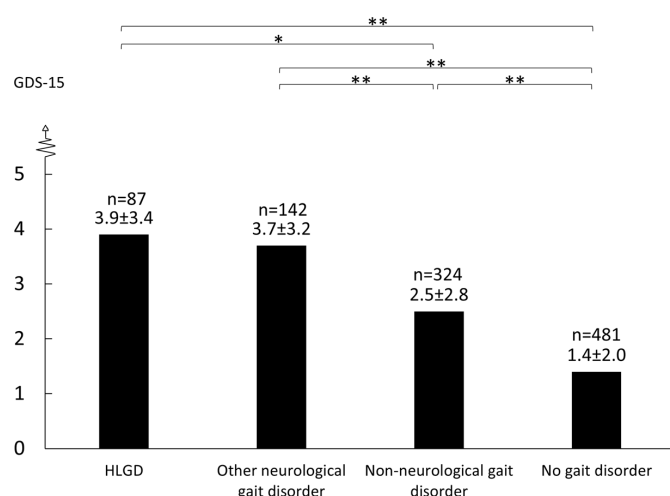


Figure 4 Symptoms of depression. Mean geriatric depression scale 15 scores compared between GD groups. The mean±SD and number of participants analysed within each group are indicated above the bars. * $p<0.05$; ** $p<0.001$. GDS-15, Geriatric Depression Scale 15; HLGD, higher-level gait disorder.

Total FES(S) and mGES scores were lower in participants with any type of GD than in those without GDs (FES(S) mean±SD, 105 ± 28 , $n=554$ vs 124 ± 13 , $n=486$, $p<0.001$; mGES mean±SD, 69 ± 22 , $n=554$ vs 90 ± 13 , $n=485$, $p<0.001$). A similar difference was seen between individuals with subjective gait impairment (according to survey answers) and those without GDs (FES(S) mean±SD, 108 ± 29 , $n=1440$ vs 129 ± 5 , $n=2194$ ($p<0.001$); mGES mean±SD, 72 ± 23 , $n=1432$ vs 97 ± 5 , $n=2193$ ($p<0.001$)).

Gait disorder prevalence and questionnaire evaluation

Of questionnaire responders, 40% ($n=1510/3769$) had a subjective gait impairment.

The positive predictive value of the questionnaire for GD according to clinical examination by a physician was 64%, and the negative predictive value was 80%. After group size corrections for application to the population, the questionnaire had 69% sensitivity, 76% specificity and 73% accuracy.

Men were more likely to answer than women (61%, $n=1855/3064$ vs 56%, $n=1914/3403$; OR 0.84 95% CI 0.76 to 0.93; $p<0.001$). Responders had slightly lower age than non-responders (mean±SD: 73 ± 5.2 y, $n=3769$ vs 74 ± 5.8 y, $n=2698$; $p<0.001$).

Comparing individuals with subjective gait impairment who chose to participate in clinical examinations and those who declined or died before the examination, those who participated had lower age (mean±SD: 74 ± 5.3 y, $n=798$ vs 76 ± 5.7 y, $n=712$; $p<0.001$) and higher mGES scores (mean±SD, 73 ± 21 y, $n=790$ vs 70 ± 24 y, $n=642$; $p=0.007$), but there was no difference in FES(S) (mean±SD: 109 ± 26 , $n=791$ vs 106 ± 33 , $n=649$; $p=0.081$).

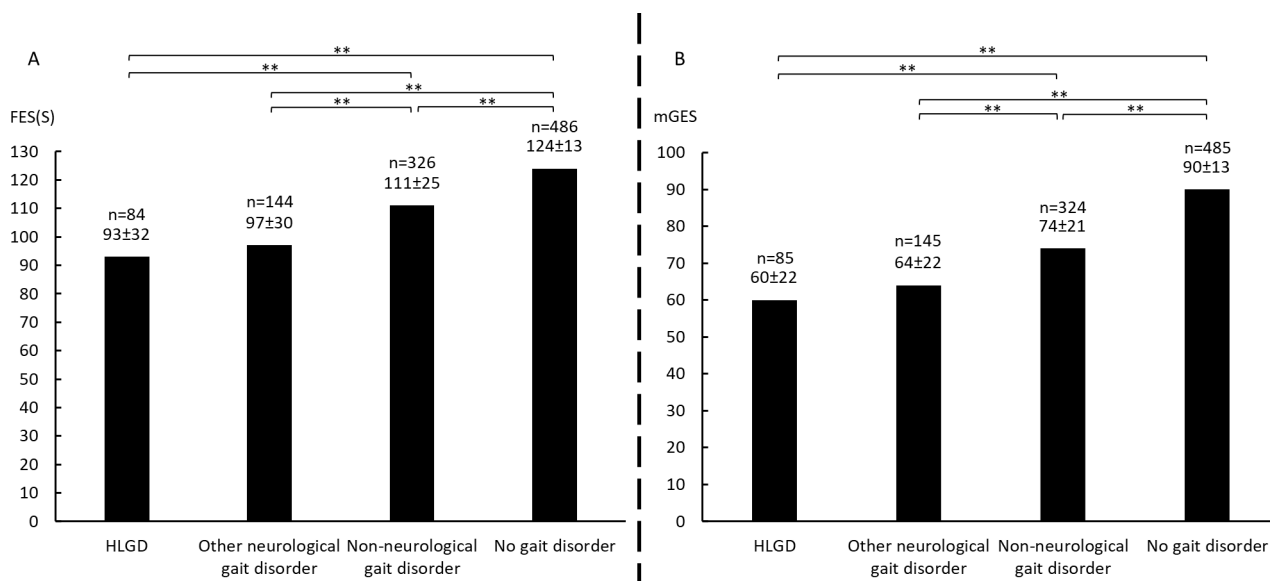


Figure 5 Gait and balance confidence. Mean Modified Gait Efficacy Scale (mGES) and Swedish Falls Efficacy Scale (FES(S)) scores compared between the different groups with GD and no GD. The mean±SD and number of participants analysed within each group are indicated above the bars. (A) mean FES(S) score; (B) mean mGES score. ** $p<0.001$. HLGD, higher-level gait disorder.

DISCUSSION

This prospective population-based study showed that HLGD was more prevalent in the general older population than previous research has implied (5.8% vs 2%).^{10 11} HLGD was characterised by more symptoms of depression, lower QoL and reduced confidence in gait and balance compared with the groups ‘no GD’ and ‘non-neurological GD’. The HLGD profile resembled that of ‘other neurological GD’; however, in contrast, very few individuals with HLGD had an identified cause for the GD, even though treatable causes exist. Hence, awareness of HLGD should be raised, and targeted investigative algorithms should be developed, especially for primary care physicians.

Prevalence of higher-level gait disorder (HLGD)

Research on the prevalence of HLGD in the population is scarce.^{10 11} The higher prevalence in this study compared with previous studies^{10 11} could be due to slight differences in definitions of the symptom. For example, one study concluded that 50% of neurological GDs in the general population were due to an ‘unsteady gait’,¹⁰ equivalent to 7% of the whole population. ‘Unsteady gait’ was not regarded in this study, and some with mild HLGD may have been considered to fit into the ‘unsteady gait’ category by other investigators. However, we showed considerable differences between HLGD and individuals without GDs, suggesting that attention should be given to all cases of HLGD, including mild ones. Our study also included twice as many participants as the previous epidemiological studies,^{10 11} strengthening the accuracy of our results. Since HLGD was common, it should be regarded as a substantial cause of reduced QoL and depressive symptoms among older people, not disregarded as a

non-specific group of GDs that warrants no further investigation.

Psychological features associated with higher-level gait disorder (HLGD)

The reduced QoL, symptoms of depression and reduced confidence in gait and balance in HLGD are distressing, especially since 76 out of 87 individuals had an unknown aetiology to the symptom, indicating they had not been in contact with healthcare professionals for their gait-related problems, or the cause could not be identified.

Functional disability is a risk factor for depression,²⁴ which may be mirrored in the association between HLGD and symptoms of depression. Symptoms of depression may also cause functional disability.²⁵ Evaluation of psychomotor symptoms is part of a standard psychiatric evaluation, but if the gait impairment is non-equivalent to the psychiatric symptoms, other causes should be considered. Those with HLGD in primary care should be screened for symptoms of depression. Depressive symptoms should elicit an in-depth anamnesis directed towards depression and, if applicable, initiation of treatment. However, the problem is complex since the use of anti-depressant drugs in old age may also contribute to higher mortality rates²⁶ and falls.^{26 27}

Impaired gait function, falls and fear of falling are associated with reduced QoL and depressive symptoms.⁴⁻⁶ However, some associated problems may be reduced by interventions (physical therapy and home-safety improvements),²⁸ which emphasise the importance of awareness of HLGD and a thorough anamnesis of associated problems.

Higher-level gait disorder (HLGD) in clinical care

The severity of the GDs in this study varied from pronounced with very slow gait velocity to milder symptoms. Additional symptoms might have emerged in a follow-up visit, making a final diagnosis possible in some individuals. However, the definition of HLGD is broad, and symptoms originating from the lower or middle levels of the nervous system may overlap with higher-level findings, introducing challenges in the diagnostics. In a previous study investigating clinical characteristics of the 'cautious gait of unknown origin', defined similar to HLGD in the present study, the cases had frontal lobe, postural and extrapyramidal disturbances; more fear of falling; incontinence; sleep disturbances; and lower cognition than normal walkers. In these individuals, more extensive investigations were performed, including neuroimaging; still, no known diagnosis could explain the syndrome.²⁹

Whether the differences between HLGD of unknown origin and normal gait represent an entity or an early stage of some previously known disease, such as idiopathic normal pressure hydrocephalus (INPH) or a parkinsonian disorder, is unclear. It was interesting that male sex was more common in the HLGD group (64% vs 36%). A similar sex imbalance can be seen among patients shunted for INPH (60% vs 40%),³⁰ individuals that should be represented in the HLGD population. INPH has been associated with hyperlipidaemia, diabetes and obesity,³¹ and ventricular enlargement (mandatory for INPH) has been associated with hypertension.³² HLGD could also encompass cases with large-vessel strokes and microinfarcts, and HLGD has been associated with white matter lesions on MRI.⁹ Hence, the higher prevalence of HLGD in men may reflect the higher cardiovascular disease incidence rate in men (13.0 vs 7.7%).³³

Due to the multiple and varying underlying conditions that may present as HLGD,⁹ it is a daunting task to present an all-encompassing diagnostic work-up. We do however suggest that radiology (CT or MRI) of the brain and spine is performed, providing structural information on white matter lesion burden, ischaemic strokes and spinal lesions that may warrant prophylactic medication or indication for operation. It would also provide information on regional atrophy, aiding in differential diagnostics, or in the case of ventriculomegaly and INPH, a possible final diagnosis.^{7,8} Polyneuropathy was common, and we humbly acknowledge the clinical overlap concerning the balance impairment of polyneuropathy and the disequilibrium of HLGD. Thus, blood tests for common polyneuropathy causes, including diabetes, vitamin deficiencies and alcohol overuse, should also be included in GD investigations. Individuals with less pronounced HLGD should likely be monitored for symptom progression. In cases with rapid progression, or when other neurological symptoms coexist, referral to a clinic specialising in movement disorders should be prioritised.

Questionnaire evaluation

Aimed to reduce the size of the population to a manageable number for physical examinations, the questionnaire-based screening of subjective gait impairment in the population filled its purpose well, with sufficient sensitivity and accuracy for the detection of GDs. The mGES and FES(S) associated with GDs and subjective gait impairment also indicate that people who experience lower confidence in performing tasks related to balance and walking in activities of daily living were identified.

Survey response rates may vary and depend on many different factors.³⁴ The 59% rate was higher than anticipated since the survey was administered to a random selection of older people with no previous relationship to the sender. Responders were slightly younger than non-responders, which is in accordance with previous research showing that response rates decline in older ages.³⁵

Future research

Pathophysiology, interventions and treatments for HLGD and associated problems, such as falls,¹¹ depression and low QoL should be investigated. Known causes of HLGD (INPH, small vessel disease and parkinsonian disorders⁹) may be difficult to differentiate between and to diagnose. For example, recent studies on the prevalence of INPH claim that it is a highly underdiagnosed syndrome;^{36–38} however, these studies did not exclude GDs that were unrepresentative for INPH (ie, not HLGD), possibly causing overestimations. Hence, standardised examination algorithms that can aid in differentiation between different disorders causing HLGD and between HLGDs and other GDs should be developed. Biomarkers for and the association between HLGD and small vessel disease should also be investigated.

Limitations

The strengths of this study include its large population-based sample, few exclusion criteria and systematic clinical evaluations of GDs, while non-response to the survey and other dropouts may introduce selection bias. Individuals who declined participation in the clinical examinations had lower confidence in their gait. Since low gait confidence was associated with GDs, it is possible that the GD prevalence was underestimated, making the results even more important. However, in a previous study investigating the reliability of information at different response rates for college surveys, the reliability was excellent (correlation to full-sample mean 0.95–1.00) at 35% response rate, and administrations with ≥1000 respondents yielded more reliable results (0.99–1.00) than smaller surveys.³⁹ Since our study had 3769 respondents and a 59% response rate, the chance for reliable results was high. Additional measures taken to reduce the risk of bias were aimed at enhancing the response rate,⁴⁰ for example, keeping the survey short and concise and sending reminders with enclosed return envelopes.

CONCLUSIONS

HLGD is common in the older population (5.8%). After a standard outpatient clinic visit, the cause often remains unknown. Therefore, clinical investigations and research should focus on measurable connections between HLGD and its underlying causes (ie, hydrocephalus, small vessel disease, extrapyramidal syndromes). Attention should also be given to depressive symptoms, QoL, and confidence in gait and balance in HLGD, as interventions targeting these issues can be beneficial for the patient.

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Data availability statement Data are available upon reasonable request. Raw anonymised data can be made available upon reasonable request from a qualified investigator.

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