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Normal Pressure Hydrocephalus Overshadowed by Traumatic and Degenerative Spinal Diseases: A New Assessment Proposal

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To watch the surgical videoclip, please visit https://www.turkishneurosurgery.org.tr/uploads/jtn-37051-video-1.mpeg

ABSTRACT

AIM: To evaluate normal pressure hydrocephalus (NPH) patients who admitted to clinic with a walking disability, depressed mood, cognitive dysfunction, and compromised quality of life in order to create a system for minimizing misdiagnosis, and inaccurate treatment of NPH at the first instance.

MATERIAL and METHODS: In this study, patients diagnosed with NPH and registered in our clinical database from 2015 to May 2019 were retrospectively screened, and a "Ste-P" formula was applied to devise a new diagnostic scoring system. Representative cases showed that some of the patients received inaccurate medical and surgical treatments before being diagnosed with NPH.

RESULTS: The study found that some of the 29 patients confused dizziness with truncal ataxia or imbalance due to gait abnormality. As the time between the onset of complaints and diagnosis increased, the diagnostic score approached zero, and diagnosis became difficult.

CONCLUSION: Every unnecessary operation carries serious risks that may threaten the life of the patient and decrease the quality of life. These surgeries and instrumentation used also mean additional financial burden to the patient. Similarly, the long-term use of Parkinson's and dementia medications has a serious economic burden on health insurance systems and is detrimental to the patient's health. Considering all these diagnoses and physiological conditions that can easily confuse one disease for another, we recommend a new diagnostic scoring system to reduce the possibility of misdiagnosis and treatment of patients with walking disorders.

KEYWORDS: Normal pressure hydrocephalus, Dementia, Parkinson disease, Lumbar spinal stenosis

ABBREVIATIONS: CSF: Cerebrospinal fluid, **LP:** Lumbar puncture, **MEP:** Motor evoked potentials, **MRI:** Magnetic resonance imaging, **NPH:** Normal pressure hydrocephalus, **PD:** Parkinson's disease

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INTRODUCTION

Gait abnormalities can be caused by a heterogeneous group of disorders. Although disturbed gait is a common condition among the elderly, the prevalence of these disorders is not well defined. Based only on a syndromic classification system, three studies involving participants from the Einstein Aging Study cohort found that 16%–20% of participants aged over 70 years suffered from neurological gait disorders (26,45–47). A community-based study showed that 32.2% of participants presented with impaired gait, 14.8% manifested neurological gait disorders, 8.2% presented non-neurological gait problems (i.e., vascular claudication), and 9.2% had a combination of neurological and non-neurological gait problems (26). Prevalence increased with age (p=0.001), although 38.3% of subjects aged 80 years or older still had a normally preserved gait (26).

Gait disorders and subsequent falls often result in the loss of independence, the use of restraints for patients and caregivers, and costs to the health care system. The most frequent diagnoses associated with gait disorder cases were stroke, Parkinson's disease (PD), and polyneuropathy, followed by multiple sclerosis and spinal disorders (26,42). Other neurological diagnoses include pain syndrome, seizures, central nervous system tumors, intracerebral bleeding, meningitis or encephalitis, non-idiopathic PD, essential tremor, polyradiculitis, Huntington's chorea, normal pressure hydrocephalus (NPH), and other rare neurological diagnoses (42).

Although abnormal gait is the main component of the classical NPH triad, only 0.5% of the patients admitted with a gait disorder are diagnosed with NPH. Thus, understanding NPH is the most important factor for accurate diagnosis. Moreover, the most important issue in NPH cases is therapeutic success. Among the aforementioned diseases, only NPH can be totally reversed, despite the associated dementia, gait disorder, and incontinence. Thus, accurately identifying the patients with NPH among those with gait disorders is crucial.

Considering the rareness of NPH and the similarity of its symptoms with several other pathologies, such as PD, lumbar spinal stenosis, and even aging, NPH is an underdiagnosed entity. However, the percentage of misdiagnosis has not been established in the literature. Therefore, we retrospectively evaluated for NPH patients admitted to our clinic with a walking disability, depressed mood, cognitive dysfunction, and compromised quality of life to create a system for minimizing misdiagnosis and inaccurate treatment of NPH at the first instance.

MATERIAL and METHODS

In this study, patients diagnosed with NPH and registered in our clinical database between 2015 and May 2019 were retrospectively screened. The screening identified 29 patients with NPH. Of these, 11 had previously been diagnosed with different diseases in other centers. Eight patients, including the example cases presented below, were diagnosed with degenerative spinal diseases and were recommended to undergo spinal operations, some of whom underwent the surgeries. The remaining three patients were diagnosed with PD (n=2) and dementia (n=1) and were prescribed pharmacologic treatments. Only two of the patients underwent treatment for NPH at our hospital. The other nine patients underwent further investigations and subsequent surgery for NPH in other centers due to insurance company agreements and due to being overseas at the time.

To diagnose NPH, computed tomography or magnetic resonance imaging studies should be performed. Cerebral atrophy, obstruction of cerebrospinal fluid (CSF) pathology, and cerebrovascular ischemia are important differential diagnoses. Supplementary testing is also performed. These tests include consecutive lumbar puncture (LP), external lumbar drainage, or measures of CSF outflow resistance. Tests can increase the prognostic accuracy. The method of choice depends on the clinician's experience.

All participants gave written informed consent. All investigations were carried out in accordance with the principles expressed in the Declaration of Helsinki.

Case 1

A 78-year-old man was admitted to our clinic in February 2017 with complaints of walking disability and urinary incontinence. Neurological examination showed that he was able to stand up from the wheelchair with the help of two people but unable to walk independently and had truncal ataxia. Assessment of medical history revealed that he fell at home and broke his L2 vertebra, for which T12, L1, L3, and L4 pedicle screw instrumentation and fusion were performed in 2014 at another institution (Figure 1). After the operation, the patient started PD treatment due to difficulty in walking. Despite medical treatment of PD, his walking condition worsened. He

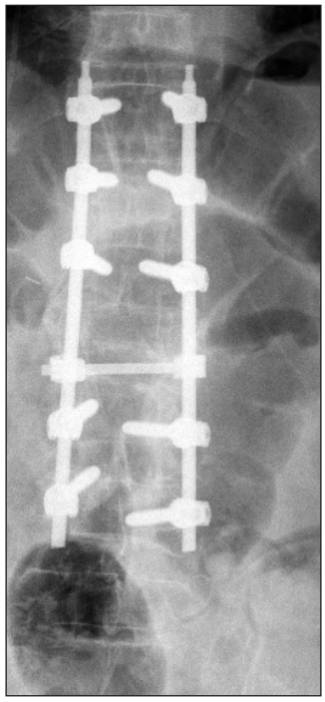

Figure 1: PA plain X-ray of "Case 1" showing T12, L1, L3, and L4 pedicle screw instrumentation.

consulted another center and was diagnosed with cervical canal stenosis. Posterior cervical instrumentation and fusion with C3–C6 decompressive laminectomies were performed in 2015 (Figure 2A, B). After the operation, the patient's walking difficulty worsened daily. However, when careful anamnesis was performed, the family stated that 5 to 6 years before the fall accident and lumbar operation, he was diagnosed with PD because of difficulty in walking, and various medications were started. During follow-up, the doctors changed the medications several times, which were eventually discontinued because the patient's condition did not improve.

After cervical and lumbar instrumentations were checked through plain X-rays and magnetic resonance imaging (MRI), a new brain MRI was recommended. Brain MRI showed chronic atrophic changes and ischemic gliotic lesions, and periventricular white matter T2A hyperintensity due to transependymal CSF transition (Figure 3). In coronal sections, the subarachnoid distance of convexity was constricted (Figure 3B). CSF-flow MRI was also performed and showed a correlation with NPH.

After informing the patient and his family and obtaining written informed consent, operation was performed, and a programmable ventriculoperitoneal Codman–Hakim shunt system was inserted. On the third month, the patient was able to stand up easily, sit down, and walk without support (Video 1). The patient also displayed improved mental capacity and speech.

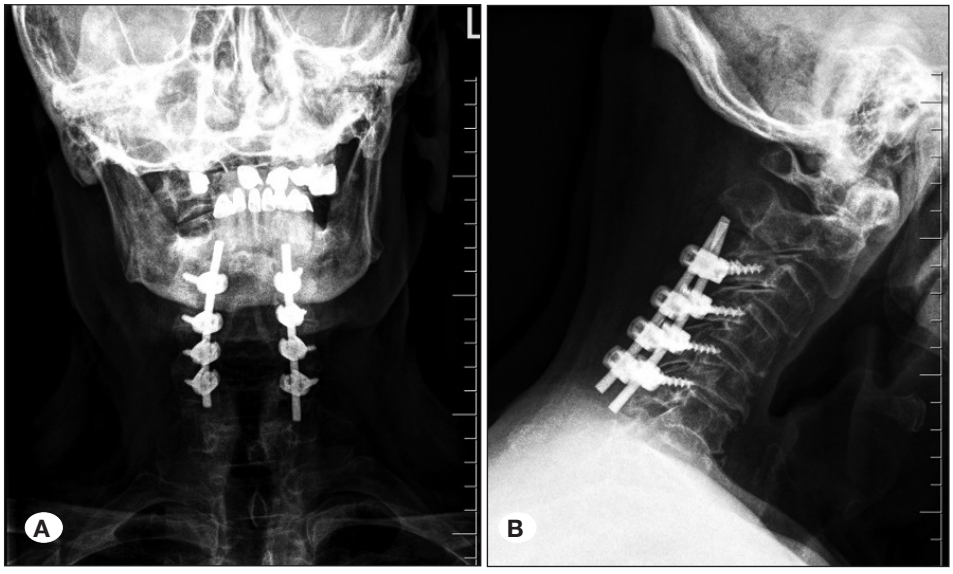

Figure 2: A) PA and **B)** lateral plain X-rays of Case 1 showing posterior cervical instrumentation with lateral mass screws and C3–C6 decompressive laminectomies.

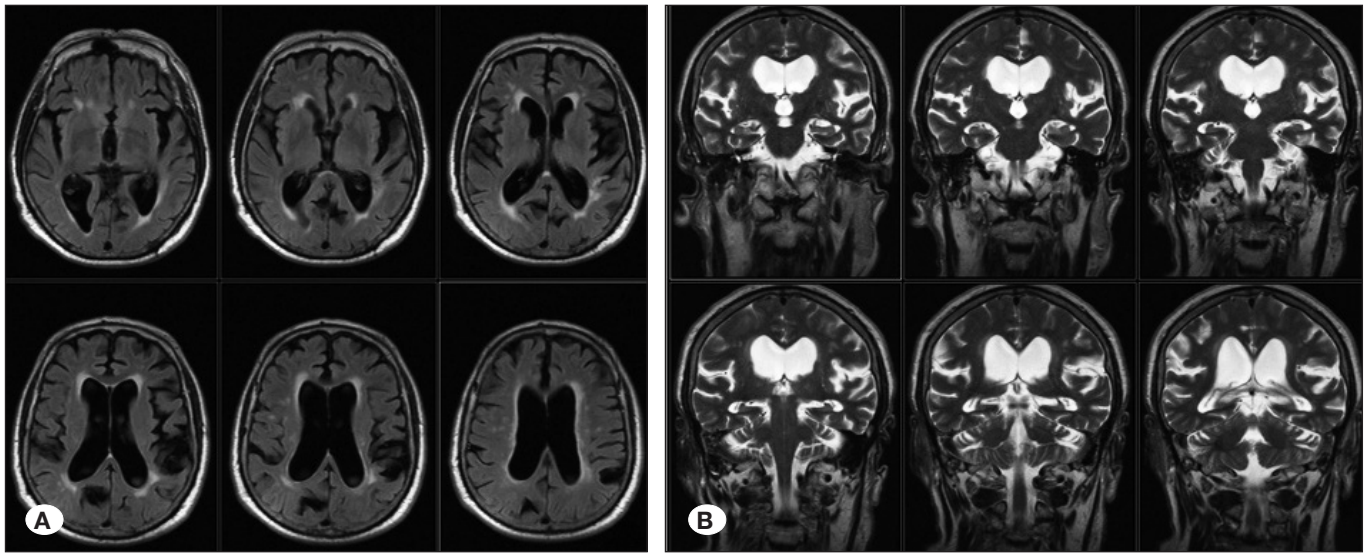

Figure 3: A) Axial fluid-attenuated inversion recovery brain magnetic resonance imaging (MRI) sequences showing chronic atrophic changes, ischemic gliotic lesions, and periventricular white matter T2A hyperintensity due to cerebrospinal fluid transition; and **B)** coronal T2-weighted brain MRI sequences showing tight high-convexity and medial (interhemispheric) surface subarachnoid spaces, enlarged ventricles, and Sylvian fissures.

Case 2

A 69-year-old woman was admitted for a longstanding history of lower back pain accompanied with dizziness, lower back and leg numbness, and walking difficulty. Upon assessment, she also mentioned having neurogenic claudication and experiencing night cramps on both legs. She was offered spinal stenosis surgery at another hospital for both cervical and lumbar spine. Her lumbar MRIs showed multiple-level disk protrusions, spondylolisthesis with facet fracture at the L4–L5 level, and foraminal stenosis (Figure 4A, B). Cervical MRI showed cervical stenosis without myelomalacia at the C4–C6 levels (Figure 5A, B), whereas motor evoked potentials (MEP) and somatosensory evoked potentials (SSEP) revealed no deficits. On examination, she had a positive Lasegue test on the right side. She exhibited a slow, short-stepped, broad-based, and unstable gait. Brain MRI showed mild ventriculomegaly, ischemic gliotic changes, and, on T2-weighted fluid-attenuated inversion recovery MRI, periventricular hyperintensity (PVH) in the parenchymal tissue directly adjacent to the frontal and occipital horns of the lateral ventricles (Figure 6A, B). Evan's ratio was 0.32 (borderline). As the walking pattern was quite typical for NPH, she was hospitalized for 3 days, and LP was performed twice, which resulted in 12 cmH_2O pressure and significant gait improvement. The patient and her family were informed that lumbar and cervical spinal surgeries with instrumentation and

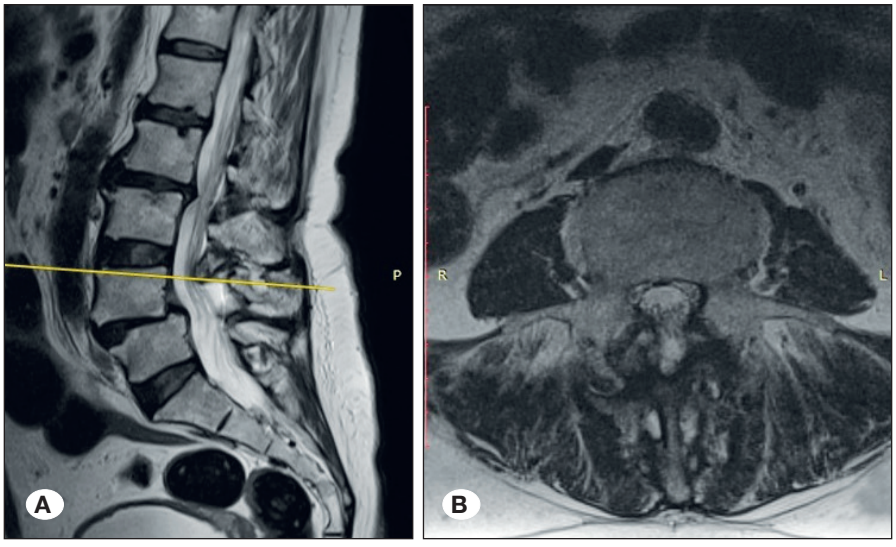

Figure 4: A) Sagittal and **B)** axial T2-weighted lumbar magnetic resonance imaging sections showing multiple-level disk protrusions, spondylolisthesis with facet fracture at the L4–L5 level, and foraminal narrowing.

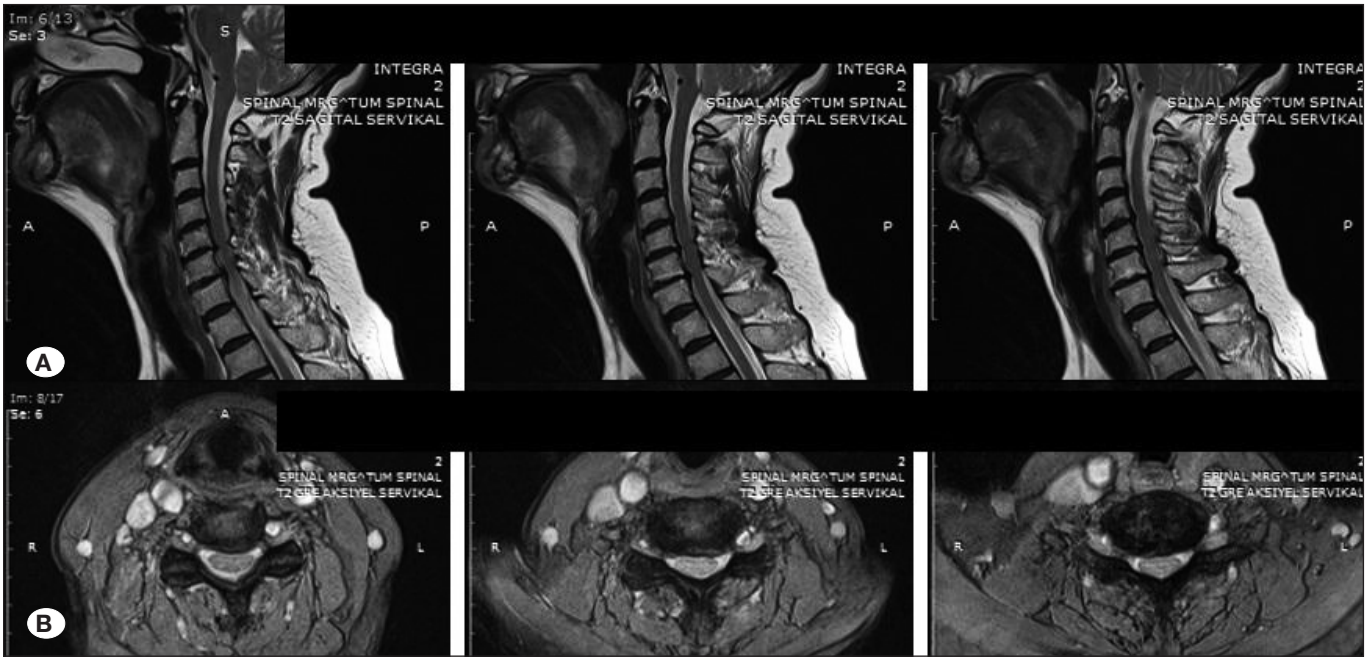

Figure 5: Sagittal **(A)** and axial **(B)** T2-weighted cervical magnetic resonance imaging sections showing cervical stenosis at C4–C5, C5–C6, and C6–C7 levels without myelomalacia.

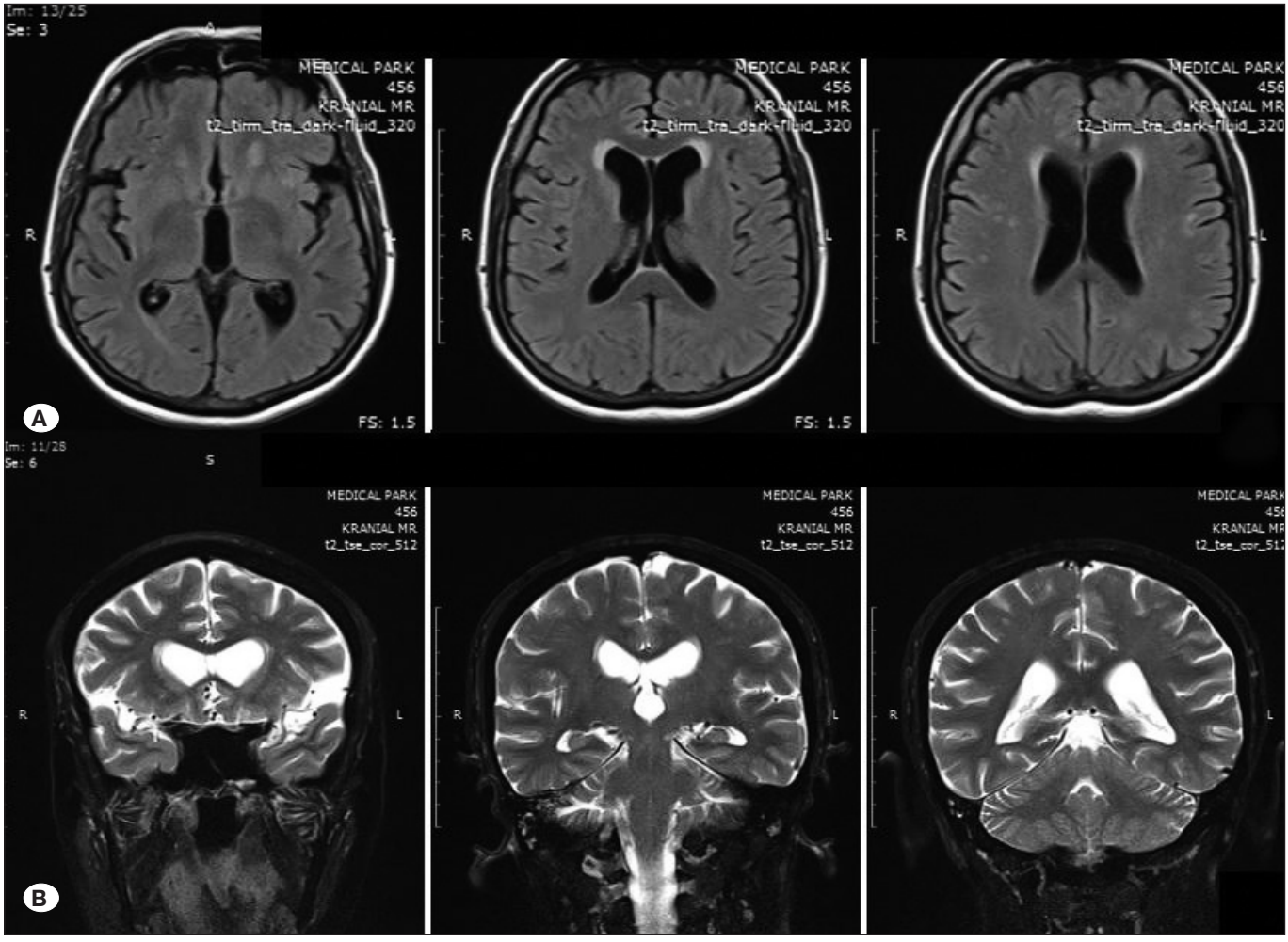

Figure 6: A) Axial fluid-attenuated inversion recovery brain magnetic resonance imaging (MRI) sequences showing ischemic gliotic lesions and periventricular white matter T2A hyperintensities; **B)** coronal T2-weighted brain MRI sequences showing tight high-convexity and medial (interhemispheric) surface subarachnoid spaces with mild ventriculomegaly and enlarged Sylvian fissures.

fusion are also needed. She refused the ventriculoperitoneal shunt because she did not want to undergo brain surgery. Although the patient was informed that a problem may occur in the shunt system when lumbar surgery is needed in the future, she preferred a lumboperitoneal shunt. Upon obtaining the required consent forms, a Miethke dual switch 10/30 lumboperitoneal shunt was implanted. After 15 months of follow-up, both gait and cognitive status were still in perfect condition. The patient no longer complained of numbness in her hands after the shunt procedure. The cervical narrow canal and lumbar spondylolisthesis were monitored with follow-up clinical examination, spinal X-rays, MRI, and SSEP and MEP.

RESULTS

Considering that similar diagnoses and physiological conditions can be easily confused with one another, we have devised and recommend a new diagnostic scoring system to reduce the possibility of misdiagnosis and inaccurate treatment in patients with walking disorders. The following questions should be asked of every elderly patient presenting with a walking disorder or who had experienced a fall accident. The formula in Table I can be used to assess gait disorders. The eight questions listed on the symptoms of patients must be asked briefly. The **C**omp**R**e**S**sion or **D**e**M**en**SIA** mnemonic can also be used: C for claudication (–1), R for Romberg's sign (–1), S for stooping (–1), D for dizziness (+1), M for memory (+1), S for symmetry (+1), I for incontinence (+1), and A for apraxia (+1). Negative values indicate a likelihood of a spinal pathology, whereas positive values indicate correlation to NPH (e.g., spinal stenosis < 0 < NPH). As the score approaches zero, suspicion for PD should be raised (Table II). Even if the patient comes with a post-fall and spinal traumatic problem, these questions should be asked and recorded as belonging to the period before the fall. If complaints are suggestive of NPH and PD, the patient should be monitored closely after spinal surgery. If possible, the pre- and postoperative status should be evaluated by a neurologist. This formula is not a 100% diagnostic evaluation. Many evaluation tools for walking, dementia, etc. are available, but the process is long and detailed and takes a long time. As most symptoms were

Table I: Ste-p Formula (**C**omp**R**e**S**sion / **D**e**M**en**SIA**) Describing Predisposition of Gait Disorders Needed Surgical Evaluation

	Mnemonic	
neurogenic **Claudication**	C	-1
Romberg test positive	R	-1
forward lean while walking - **Stoop**	S	-1
Dizziness	D	+1
short term **Memory** deficit	M	+1
Symmetry in complaints and/or gait disorder	S	+1
urinary **Incontinence**	I	+1
gait **Apraxia**	A	+1
	Comp**R**e**S**sion / **D**e**M**en**SIA**	
Spinal Stenosis < 0 < NPH	**-1, -1,-1, < 0 < +1, +1, +1, +1**	

Table II: Example of Calculation for Parkinson's Disease

	Mnemonic	
neurogenic **Claudication**	C	No
Romberg test positive	R	-1
forward lean while walking - **Stoop**	S	-1
Dizziness	D	+1
short term **Memory** deficit	M	+1
Symmetry in complaints and/or gait disorder	S	No
urinary **Incontinence**	I	No
gait **Apraxia**	A	No
	Spinal Stenosis < 0 < NPH	
Result		0

The romberg test positivity and forward lean while walking are together a total of -2 for PD, and the symptoms will be asymmetrical (S: No), but for memory disorders and orthostatic dizziness there will be a total of +2, and the total result is zero. So as we approach zero, PD should be kept in mind.

common in these diseases, we conducted a large literature review and devised this simple formula. This makes the Ste-P formula easy to remember, easy to apply, and will provide a prompt for the many similar diseases (Table III).

In the study period (2015 to May 2019), the medical records of the 29 patients (including the 11 patients who were previously misdiagnosed and inaccurately treated prior to NPH diagnosis) admitted to our clinic and diagnosed as NPH were retrospectively scanned, and the Ste-P formula was applied (Table IV). If the questions in the formula are not answered in the records, the case is scored as 0. All patients had positive values, except one male patient who had a total score of 0. Some of the patients confused dizziness with truncal ataxia or imbalance due to gait abnormality. As the time between the onset of complaints and diagnosis increased, the diagnostic score approached zero, and diagnosis became difficult. If the patient was misdiagnosed and received imprecise medical and/or surgical treatment, the diagnosis time was considered prolonged, and the score was also closer to zero. Despite the abovementioned diagnostic difficulties, none of the patients with NPH were evaluated with negative scores.

Table III: Example of Calculation for Case-2

	Mnemonic	
neurogenic **Claudication**	C	-1
Romberg test positive	R	No
forward lean while walking - **Stoop**	S	-1
Dizziness	D	+1
short term **Memory** deficit	M	+1
Symmetry in complaints and/or gait disorder	S	+1
urinary **Incontinence**	I	No
gait **Apraxia**	A	+1
	Spinal Stenosis < 0 < NPH	
Result		+2

Neurogenic claudication and forward lean while walking are together -2 for spinal canal stenosis, and the other symptoms (dizziness, memory disorder, symmetry in complaints, and gait apraxia) are total of +4, and the total result is +2. So NPH should be kept in mind, even if the radiological results mostly indicated spinal canal stenosis.

Table IV: Characteristics and Ste-P Formula Values of the NPH Patients

	Number of patients	**Age (year)**	**Ste-P Formula**
Male	13	72.92 ± 6.37	2.33 ± 1.30
Female	16	71 ± 8.98	2.88 ± 1.09

DISCUSSION

Gait disturbance is one of the main complaints of patients in many branches of medicine. It may develop due to neurologic, vascular, musculoskeletal, or cardiorespiratory problems. Notably, aging is the most important reason associated with gait disturbance. Furthermore, gait patterns are affected by

age, sex, and sociocultural factors. For example, patients with depression have a slow walking speed. Among the elderly, step width and speed decrease. Gait disorders reduce quality of life and are associated with falls, bone fractures, and brain trauma in the elderly. Doctors need to have the necessary knowledge of the physiopathology of the various diseases, carefully examine patients, and obtain detailed anamnesis with well-planned radiological examinations and appropriate instrumental investigations before deciding on the diagnosis.

Clinical treatment for gait disorders can improve physical condition, which allows patients to walk for distance, such as 10 meters. Doctors should also observe how patients stand up from a chair, such as by using the timed up-and-go test (2). This test measures the time it takes for patients stand up, walk 3 meters, turn around, and sit back. It is helpful in determining a patient's risk for falls. Doctors should also conduct a neurological examination. In order to make a differential diagnosis, clinical findings of other diseases should be ruled out. Of all the walking disorders, three diseases that are often confused with each other: NPH, PD, and degenerative spinal diseases.

NPH is a rare disease among elderly people. The triad of the disease consists of gait disorder, dementia, and urinary incontinence. Ventriculomegaly with normal CSF pressure is observed. However, the clinical triad is not necessary for diagnosis. No standard clinical tests have been established except examination and anamnesis because brain atrophy and ventricular dilatation are common in the elderly (48). Thus, NPH can be easily confused with other pathologies causing gait disturbance. Dementia and urinary incontinence are most commonly associated with age, and the latter may be due to disability (12). PD may be easily confused with NPH, as it can cause dementia as well. In addition to performing cognitive tasks after LP, gait assessment is very helpful in distinguishing NPH from comorbidities causing these symptoms. Abnormal gait is the most improved symptom after LP; however, response to dementia is very limited (1). NPH can be classified into two groups. The etiology of the first group is not known, whereas that in the second group is usually associated with some intracranial problems, such as head trauma, subarachnoid hemorrhage, meningitis, and tumors. The pathophysiology of NPH is not yet well known, but CSF absorption resistance in the arachnoid villi may be involved (28). Gait disorders are typically the first symptom in patients with NPH. A slow, short-stepped, broad-based, and unstable gait is usually observed, and patients have difficulty in turning back (12). Dementia is subcortical and frontal in nature (16). Attention deficit and memory disorder can also manifest, indicating the need for performing differential diagnosis. Patients also exhibit pollakiuria and urgency, and fecal incontinence occurs with the progression of the disease (39). Nevertheless, these symptoms can also manifest in PD and other neurodegenerative diseases.

If the medical history recorded from the patient and relatives, symptoms, and examination results indicate NPH, cranial MRI must be performed. MRI, PVH, ventricular dilatation (Evan's index > 0.3), dilated temporal horns, absence of any obstructions to CSF, and a corpus callosum angle > 40° are the criteria for NPH imaging (49). Evan's index is the ratio of the maximum width of the frontal horns of the lateral ventricles and the maximal biparietal internal diameter of the skull at the same level on axial computed tomography and MRI. A ratio greater than 0.3 indicates ventriculomegaly. However, no association was found between clinical recovery relationship after shunting and Evan's index (4,29,38). Moreover, since the shape of the ventriculomegaly is more vertical in NPH, the Evan's index may be borderline, which is not a determinant for hydrocephalus in these patients. The periventricular signal undergoes a change, which may be due to subcortical vascular encephalopathy, but it does not represent a negative response to shunting (22,44). The CSF-flow void in the aqueduct can be used in diagnosing NPH, with a void greater than 18 mL/min indicating NPH (25). It is also effective in evaluating shunt response (5).

Shunting is the only effective treatment for NPH (49). A ventriculoperitoneal shunt is the standard; however, in some cases, ventriculoatrial or lumboperitoneal shunts are used. Programmable shunts are now a gold standard. Complications of shunting include subdural hematoma, catheter malposition, infection, pump failure, proximal or distal catheter obstruction, subdural hygroma, and headache.

PD is a neurodegenerative disease that occurs mostly in people more than 60 years old. Degeneration is observed in dopamine-secreting cells of the substantia nigra of the basal ganglia. The etiology of PD is not known; however, viral infections and heavy metal intoxication, especially manganese and 1 methyl-4-phenyl-1,2,3,6,-tetrahydropyridine, have been associated with the development of the disease (24). Genetic predisposition is also an important factor (36,37).

Tremor, postural instability, rigidity, and bradykinesia are the major symptoms. Tremor is the most common symptom noted during the onset of PD. It generally occurs unilaterally at rest, mostly in the upper limb. The frequency of the tremor in PD is 3–5 cycles/second in a drawing pill-rolling motion. Usually, tremor is accompanied by other symptoms, such as bradykinesia and rigidity. PD is usually asymmetrical at first. Bradykinesia can be an early sign of the disease. It is characterized by distal muscle incompetence, which is evident in shirt buttoning and handwriting. The time to do manual tasks is longer in PD. Rigidity indicates increased muscle tone and resistance to passive motion. Patients have more difficulty initiating walking than sustaining it and also experience stiffness and reduced arm swing. Gait disturbance in PD causes slow, short, and shuffled steps. Postural instability is associated with bradykinesia and rigidity (18). Extensor muscles are weaker than flexor muscles; therefore, stooping is pronounced. When healthy persons need to walk carefully, they restore their posture first. In Parkinsonian gait, restoration of the posture gets disrupted, and patients freeze when they are approaching narrowed parts or obstacles on the road. Freezing regresses with anti-Parkinsonian medications.

Symptoms of dysautonomia are observed in PD and include constipation, urinary problems, orthostatic hypotension, thermoregulation problems, sexual dysfunction, and neuropsy-

chiatric disorders such as cognitive impairment, dementia, depression, and psychosis (7). PD is a clinical diagnosis. If at least two of the four major symptoms and response to dopaminergic medications are present, PD will be diagnosed. MRI is helpful in ruling out other diagnoses, as it is normal in PD (34).

Lumbar spinal stenosis is a common disease observed in people aged over 60–70 years. It is one of the most common reason for spinal surgery in the elderly (3) and can be classified as congenital or acquired depending on the etiology and severity of stenosis. Acquired lumbar stenosis is more common. Degenerative, traumatic, secondary to surgery, or metabolic diseases, such as Paget and rheumatoid arthritis, may cause lumbar stenosis. Disc degeneration is mostly seen and is the main cause of lumbar spinal stenosis in the elderly. Congenital narrow spinal canal can occur due to achondroplasias, spinal dysraphism, and hereditary causes (3). Patients complain of weakness in the lower extremity and neural claudication. When walking, patients typically stop because of leg pain, numbness, and weakness (19,33). Interestingly, the degree of stenosis on MRI does not correlate with the symptoms. Therefore, one person can be symptomatic due to the canal size but another can be asymptomatic with a similar canal size (33).

The inability to provide the necessary care and attention while making a differential diagnosis can lead to inaccurate medical treatment and unnecessary surgery. Long-term inappropriate drug treatment and unnecessary surgeries carry great risk to the patient's health. In addition, these also create a financial burden on the patient and the health insurance system. As seen in Case 1, when the patient was diagnosed with PD, a long-term treatment process had been initiated in changing the doses and times of administration of different drugs.

In terms of degenerative spinal diseases, lumbar surgery in Case 1 was necessary due to compression fracture of the vertebrae. However, in Case 2, it was considered as the cause of gait disorder, and surgery was recommended. The complication rate reported for surgical techniques performed on the lumbar narrow channel varies significantly between 4% and 45%. Studies have reported complications ranging from temporary urinary retention to cerebrovascular events. In general, reoperation rates were between 3% and 28%. Higher complication rate (31%, 13%) and reoperation rate (10%, 3%) were reported in cases with both decompression and fusion than in those with decompression only.

The reported incidence of postoperative respiratory compromise varies from 0% to 14% (6,8,27,35,51). Recurrent injuries to the laryngeal, superior laryngeal, and hypoglossal nerves and tracheal, esophageal (0.2%–1.15%) (13,30), carotid, and vertebral arteries (0.3%–0.5%) (14,41) have been reported for the anterior cervical approaches. Thoracic duct injury and cervical sympathetic chain injury, as well as resultant ipsilateral Horner's syndrome (miosis, ptosis, and anhidrosis) are rare (4.2%) (6,10). The overall morbidity risk associated with corpectomy is 11%–27% (6). Despite the high rate of complications, mortality is low (0.1%) (6,11). Unlike discectomy, corpectomies carry a greater risk of graft migration, strut graft dislodgement, infection, and pseudoarthrosis due to a larger destabilization of the anterior column (6,15). Other complications include graft pistoning, mortise penetration, inadequate deformity correction and fixation failure, and neurological compromise (6). Late complications include graft fracture, collapse or subsidence, and non-union (6). In surgery for ossification of posterior longitudinal ligament, the reported postoperative incidence is 2%–10% and 5%–17% for quadriplegia and root injury, respectively (6,50).

For the posterior approaches, the risk of durotomy during laminectomy is 0.3%–13% and can increase to 18% with revision surgery (6,9,40). Screw malposition varies from 0%–4% in the atlas and 0%–7% in the axis (6,31,43). Transarticular C1–C2 screws or Magerl screws pose an additional risk of vertebral artery injury, neurological deficit, or inadequate bony purchase. Subaxial lateral mass screws carry a risk of nerve root injury (1.3%) and lateral mass fracture (6,21). The incidence of kyphosis after multilevel laminectomy is 20% (20). Furthermore, up to 15% of anterior cervical discectomy and fusion cases (6,17,23) and 9% of all posterior surgeries develop adjacent segment degeneration (6,32).

The literature review shows that every unnecessary operation carries serious risks that may threaten the life of the patient and decrease the quality of life. Apart from surgical risks, the cost of these surgeries and instrumentation materials used in spinal surgery also means serious financial loss. Similarly, the long-term use of Parkinson's and dementia medications has a serious economic burden on the health insurance system and is detrimental to the patient's health. Thus, NPH is still an underestimated diagnosis for which many patients are diagnosed and treated inaccurately and too late to benefit from shunt surgery. Furthermore, instrumentation and fusion procedures performed in the lumbar region can make it impossible for the patient to undergo LP for the diagnosis of NPH.

The limitations of the study present study are; the preliminary diagnostic scoring system developed in this study should be evaluated in a large number of elderly patients presenting with walking disorders, and the adequacy of the assessment should be tested by a larger-scale, multicenter study.

CONCLUSION

Especially in elderly patients with a history of falls during walking or standing, a thorough history needs to be obtained and a careful examination conducted before planning surgical and conservative treatment. Following the correction of the spinal problems identified and keeping in mind other pathologies that may cause gait disturbance and falls, long-term follow-up of the patients and planning of the necessary examinations will prevent many misdiagnoses and unnecessary, risky, and expensive treatments.

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AUTHORSHIP CONTRIBUTION

Study conception and design: HD, PO

Data collection: HD, EE, SK

Analysis and interpretation of results: PK, MBS

Draft manuscript preparation: PK, OS

Critical revision of the article: HD, PK, PO

Other (study supervision, fundings, materials, etc...): PO, MBS, AS

All authors (HD, PK, EE, AS, OS, MBS, SK, PO) reviewed the results and approved the final version of the manuscript.

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