DERLEME REVIEW

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Pusher Syndrome: Not a Rare, But a Little-known Phenomenon in Stroke Survivors: A Review of the Literature with Report of a Typical Case

Pusher Sendromu: İnme Hastalarında Nadir Olmayan Ancak Sık Gözden Kaçan Bir Fenomen: Literatür Derlemesi ve Tipik Bir Olgunun Sunumu

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ABSTRACT Pusher syndrome (PS) is an abnormal postural reaction characterized by actively pushing and tilting the body to the hemiplegic side in some stroke patients. Its exact etiology is unknown. However, the disruption of the neural network that processes gravitational sensation and verticality perception is accused for PS. These patients have postural instability that impairs balance, transfer, mobility, and functionality, even if they have adequate motor activity on the hemiplegic side. However, PS may be overlooked in acute care and rehabilitation, especially when dealing with pain or other motor and cognitive problems. In this paper, the current literature on the pathophysiological basis, diagnostic approaches, and rehabilitation interventions of PS will be discussed through a case example.

Keywords: Pusher syndrome; contraversive pushing; lateropulsion; stroke; posture

ÖZET Pusher sendromu (PS), bazı inme hastalarında vücudun aktif olarak hemiplejik tarafa doğru itilmesi ve eğilmesi ile karakterize, anormal bir postüral reaksiyondur. Kesin etiyolojisi bilinmemektedir. Ancak yer çekimi hissi ve dikeylik algısına ilişkin nöral bağlantıların bozulması sorumlu tutulmaktadır. Bu hastalar, hemiplejik tarafta yeterli motor aktiviteye sahip olsalar bile denge, transfer, hareketlilik ve fonksiyonelliği bozan postüral instabiliteye sahiptir. Bununla birlikte, akut bakım ve rehabilitasyonda, özellikle ağrı veya diğer motor ve bilişsel problemlerle uğraşırken PS gözden kaçabilir. Bu yazıda, PS'nin patofizyolojik temeli, tanısal yaklaşımları ve rehabilitasyon girişimleri ile ilgili güncel literatür bir vaka örneği üzerinden tartışılacaktır.

Anahtar Kelimeler: Pusher sendromu; kontraversif itme; lateropulsiyon; inme; postür

Patricia Davis first defined the Pusher syndrome (PS) in 1985 as the behavior of pushing the body towards the paretic side in stroke patients. Patients with PS lean on their contralesional side while sitting or standing and even fall due to lateral postural instability when not supported.[1] PS differs from ipsilesional lateropulsion of medullary and pontine stroke, which means an irresistible falling to one side. Beyond lateropulsion, patients with PS actively resist corrective external forces by pushing themselves with their non-paretic extremities. It is also called "contraversive lateropulsion with pushing."[2] The prevalence of PS among stroke patients has been reported to be 9-63%.[3,4] It is more common in patients with right hemisphere stroke.[5,6] Deep subcortical lesions involving the insula and thalamus seem to be associated with the PS.[3,7,8]

The exact mechanism of this peculiar behavior is unknown, but a disruption in multimodal sensory processing regarding postural control rather than a pri-

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mary somatosensory impairment is accused.[9] Studies have shown that PS patients have a deviated perception of subjective vertical regarding non-pushers.[10,11] Therefore, PS has been related to impaired gravity perception (graviception) or vestibular misperception, due to a mismatch between somatosensory information and internal postural modelling.[12] PS frequently accompanies visuospatial problems such as neglect syndrome. Although described as graviceptional neglect by some authors, its mechanism cannot be explained by the horizontal shift of the spatial midline in neglect syndrome.[4,13,14] Nevertheless, the close relationship between the PS and neglect syndrome raised curiosity about the neural networks regarding the perception of body orientation in different spatial planes.[15]

Mild forms of PS may be overlooked by inexperienced clinicians, especially when dealing with pain or other motor and cognitive problems. However, it is associated with poor mobility and functionality, delays functional gains during rehabilitation, and lengthens hospital stay.[4,16-18] In this narrative review, the neural correlates, diagnosis, clinical course, and recent rehabilitation approaches of PS will be discussed through a case of chronic stroke patient.

CASE REPORT

A sixty five years old male patient with hypertension and dilated cardiomyopathy for seven years was admitted to our rehabilitation clinic with left hemiparesis. He had suffered a hemorrhagic stroke 6 months ago involving the right basal ganglia and the subcortical white matter of the frontoparietal lobes (Figure 1). He could not sit unsupported and not stand unaided. Berg Balance Scale (BBS) score and Functional Ambulation Classification (FAC) level were both 0. There was a severe left shoulder pain and glenohumeral subluxation restricting passive joint movement. He could initiate but not complete flexor synergy of the left upper extremity with mild hypertonia. There was a minimal activity in his left finger flexors. He could perform isolated hip flexion, knee flexion/extension, and some ankle dorsiflexion with flexor synergy of the left lower extremity. His motor recovery stages regarding Brunnstrom evaluation corresponded to 2, 2 and 4 for the upper extremity, hand, and lower extremity, respectively. He got 31 points on the Functional Independence Scale (FIM) indicating poor functionality. He had moderate mental dysfunction with 10 points on the Mini-Mental State Evaluation and severe left visuospatial neglect behavior in activities of daily living. Obvious right-sided bias paper and pencil tests and 30 points on the Catherine Bergego Scale (CBS) supported the diagnosis of left hemispatial neglect syndrome.

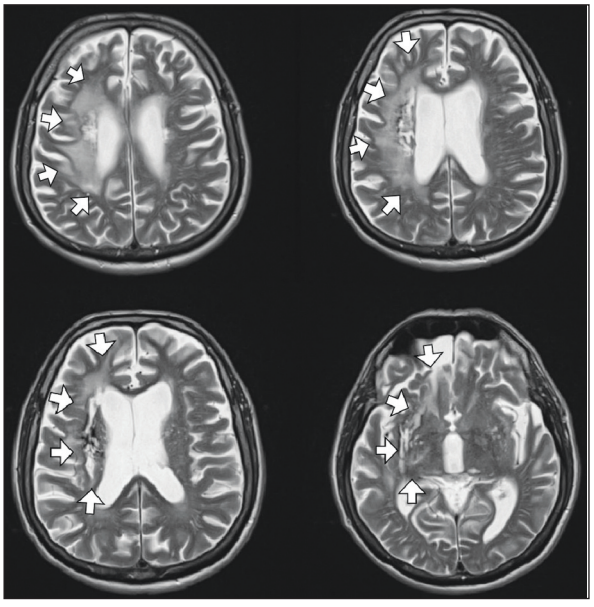

FIGURE 1: Axial T2-weighted brain MRI of the patient at 6 months after stroke (white arrows indicate brain areas affected by hemorrhagic lesion).
MRI: Magnetic resonance imaging.

From the first days of rehabilitation, he complained of intractable hemiplegic shoulder pain unresponsive to the conventional interventions. Therefore, we decided to perform a left suprascapular nerve block under ultrasonography guidance. During the procedure in supported sitting, it was noticed that the patient was leaning to the left by pushing himself with his right upper extremity and resisted being brought to the upright. This "pushing" activity with poor postural control was similar to contraversive lateropulsion. Subsequently, the patient was diagnosed as PS with 14 points on the Burke Lateropulsion Scale (BLS). His rehabilitation program was revised and weighted on posture-correcting interventions using the mirror and virtual reality-based visual feedback [The Nintendo Wii Fit Plus™ system (Nintendo Co, Ltd, Kyoto, Japan)]. Ipsilesional reaching exercises were

implemented to prevent contraversive lateropulsion. At the end of 60 sessions (in 84 days of stay), his BLS score decreased to 11. Although the Brunnstrom stages did not change, his FAC level and BBS score increased to 1 and 5, respectively. He could sit unsupported and regain the bipedal stance using a tripod cane, but his left-leaning posture persisted (Figure 2, Figure 3). He still needed physical assistance during walking and transfers, but he could walk on smooth surfaces for short distances using a tripod cane. His FIM score slightly increased to 43. Nevertheless, his severe hemispatial neglect persisted with 28 points on the CBS at discharge to home. Informed consent was obtained from the patient and his daughter to use his medical data and images in this paper.

FIGURE 2: Bipedal stance posture of the patient on discharge.

FIGURE 3: Unsupported sitting posture of the patient on discharge.

LITERATURE REVIEW

PATHOPHYSIOLOGICAL UNDERPINNINGS OF PUSHER BEHAVIOR

The pathophysiology of PS is controversial. Abnormal postural response accompanying misinterpretation of sensorial inputs such as somatosensory, vestibular, and visual perception may be responsible for the lateropulsion in PS.[9,19,20] Barra et al. found that the representation of the longitudinal body axis deviates to the contralesional side in stroke survivors. The degree of tilt was higher in patients with neglect, lateropulsion, and right hemisphere stroke.[21]

The mismatch between the physical vertical (gravity vector or true vertical) and the behavioral (perceptional) vertical may be responsible for the abnormal pushing response.[22] Behavioral vertical can be evaluated by various methods. One of these, the subjective visual vertical (SVV), is determined using a linear target on a background that eliminates other visual-spatial cues. The subject/patient observes the target manipulated by himself or the examiner and adjusts its upright position. The SVV is closely related to the perception of head orientation as an indicator of vestibular function.[23] Saj et al. reported that SVV deviates 7.2 degrees to the ipsilesional side in pushers with neglect syndrome, while 6.6 degrees to the contralesional side in non-pushers with neglect syndrome.[24] Dai et al. found that contralesional deviation of the SVV was more frequent in PS and the de-

viation degree was correlated with the severity of lateropulsion.[14] In another study, Baier et al. showed that not only right but also left hemispheric lesions cause the contralesional tilt of the SVV. But they did not find any difference between pushers and non-pushers regarding the severity of SVV tilt.[19] However, Karnath et al. proposed that SVV change in stroke patients could not be attributed to PS. They noted that the contraversive lateropulsion is associated with a misperception of the somatosensory rather than the vestibular information of the head position.[12] Another concept, subjective postural vertical (SPV) is the perception of body verticality based on proprioceptive somatosensory. It is determined using a mechanism that tilts the body slowly and smoothly enough not to stimulate semicircular canals while sitting or standing. Most studies agree that the variability of SPV deviation is higher in PS patients than in non-pushers or healthy individuals. However, the results concerning the direction of the SPV deviation are contradictory. Studies have emphasized that SPV tends to deviate to the contralesional side in stroke patients with PS.[25-27] However, Karnath et al. found that PS patients had an ipsilesional deviation of postural vertical compared to non-pusher stroke survivors.[12] Bergmann et al. also reported an association between ipsilesional SPV error and BLS score.[28] Methodological variations such as eliminated vision or contralesional vs ipsilesional starting position may affect the SPV outcomes.[26] Fukata et al. reported that SPV deviates ipsilesionally or contralesionally regarding midline arrival from the ipsilesional or the contralesional tilt positions, respectively.[27] Subjective haptic vertical (SHV) which means the determination of verticality of a target just by touching, was not addressed as extensively as SPV and SVV in pusher behavior. Pérennou et al. reported a contralesional SHV deviation correlated with lateropulsion severity in PS patients.[25] The common point of the findings above is that the verticality judgement, determined by the integration between internal representation and perception, is impaired in PS patients.

LESION CHARACTERISTICS IN PS

Our patient's brain magnetic resonance imaging revealed a large hemorrhagic lesion extending from the lateral border of the thalamus to the subcortical area of the frontoparietal cortex involving the insula and lentiform nucleus in the right hemisphere (Figure 1). Identifying the lesion characteristics of PS patients may provide insight into the neural networks responsible for the perception of verticality.[3] Studies in stroke patients suggest that the neural networks related to perception of verticality and vestibular information show hemispheric lateralisation.[25,29] Abe et al. investigated the prevalence of PS in 1,660 cerebral hemispheric stroke patients and showed that it was 1.8 times higher in those with right hemisphere lesions.[6]

Imaging studies have shown that certain brain regions were frequently affected in PS patients. One of them is the thalamus, which acts as a junction between cortical, cerebellar, and spinal pathways. The posterolateral regions of the thalamus commonly involved in PS patients are closely associated with verticality perception and have intense neural connections with the sensorimotor and parietal cortex.[30,31]

The insular cortex, operculum and superior temporal gyrus, involved in the multisensory vestibular cortical network, are associated with PS.[19,32] It has been suggested that the inferior parietal gyrus and primary sensorimotor cortex, which play an important role in spatial orientation, are also related to the perception of verticality. Parietal cortex lesions may result in coexistence of PS and neglect syndrome.[8,32] Lee et al. found that PS severity is related to lesion size rather than any specific lesion localization.[7] In other words, larger lesions affecting more neural connections, as in our patient, result in more severe lateropulsion. These findings suggest that the cause of PS is the disruption of the neural network operating verticality perception, vestibular information, and postural reactions.

DIAGNOSIS AND DETERMINATION OF THE SEVERITY OF PS

Studies reported inconsistent results about the frequency of PS in stroke patients due to the heterogeneity of both patients included and the evaluation methods used.[4] Certain examination methods for post-stroke lateropulsion were found to be valid and reliable.[33] One of them, the BLS, is a semi-quantita-

tive scale that evaluates the severity of lateropulsion in different positions and activities such as supine lying (log roll test), sitting, standing, transfers, and walking. In each activity, lateropulsion and pushing severities are scored between 0 and 3, except for the standing situation, which gets a maximum of 4 points. An extra 1 point is added if there is resistance in both directions during the supine log roll test. The total BLS score ranges from 0 to 17 points, with an increased value corresponding to more severe lateropulsion.[34] The score for diagnosis of Pusher behavior is ≥2 points.[35]

Scale for Contraversive Pushing (SCP) includes three items that are tilt in spontaneous body posture, abduction and extension activities of non-paretic extremities, and resistance to passive correction of tilted posture. Each item is evaluated during both sitting and standing and scored as minimum 0 and maximum 2. A score of >1 for each domain indicates lateropulsion.[36]

Modified SCP (M-SCP) differs from the SCP in certain aspects. It addresses lateropulsion with pushing activities of non-paretic extremities during 2 static (sitting and standing) and 2 dynamic (sitting transfer and standing transfer) phases. Each part is assessed separately, scored as 0 (no pushing) to 2 (severe pushing). In total, zero corresponds to no sign of lateropulsion while a value of 8 indicates severe pushing behavior.[33]

In recent years, the four-point pusher score has been proposed as a valid and reliable method to evaluate PS. The severity of contralesional tilt and pushing behavior is evaluated as a whole on levels 0 to 3. Therefore, it is simple to administer in busy clinical settings.[37]

Koter et al. have suggested that the BLS is superior to other scales, as it includes various domains of functional testing positions and has less uncertainty concerning cross-cultural validity. BLS also allows the detection of minor changes in PS severity.[38] Therefore, we preferred to use the BLS to evaluate contraversive lateropulsion in our patients.

TIME COURSE OF PS AND REHABILITATION INTERVENTIONS TARGETING LATEROPULSION

Our patient was diagnosed with PS after admission to our rehabilitation clinic in the 6 month of the stroke. However, reliable information about the onset and severity of the pushing behavior in the early phases of stroke could not be obtained. Mild PS usually resolves in the first weeks after the stroke.[11] Complete recovery has been reported in 18.8-69.4% of patients regarding the initial severity of lateropulsion.[18] Persisting PS is more common in right hemisphere stroke and is associated with older age, greater admission lateropulsion severity, and lower cognitive FIM scores.[6,16] Moreover, accompanying disorders such as hemianopia, motor, proprioceptive, and visuospatial deficits are negative predictors of lateropulsion recovery.[5] Our patient had severe left hemispatial neglect with a large right hemisphere lesion and cognitive impairment accompanying persistent lateropulsion.

Our patient had an FIM gain of 12 points and a relatively low FIM efficiency of 0.14 at the end of inpatient rehabilitation. Despite sufficient motor recovery of his left lower extremity, his transfer and mobility abilities were still poor. Persistent pushing behavior negatively affects the stroke rehabilitation process.[17] PS patients have poorer FIM efficiency and longer length of stay than non-pusher, probably related to poor postural control independent of motor recovery.[4,18] Therefore, rehabilitation interventions that reduce lateropulsion are the priority in PS patients. On the other hand, the optimal therapeutic approach for contraversive lateropulsion is unclear. We provided our patient with reaching exercises to reduce pushing behavior, and postural correction training under visual feedback to facilitate upright posture. A contradiction between the internal modelling of body-midline and perceptions of the visual and proprioceptive vertical is thought to lead to abnormal postural responses in PS patients. Improving the disrupted perceptions of verticality and increasing postural orientation using visual and somatosensory cues may be a reasonable goal in PS rehabilitation.[11,39] Pardo et al. reported that treatment focused on regaining the sense of midline during mobility, and neuro re-education activities may be useful in PS patients with subacute right hemisphere stroke.[40] Karnath et al. suggested that the visual vertical perception is relatively preserved despite the proprioceptive vertical being impaired in stroke patients with con-

traversive lateropulsion. Therefore, PS patients can be taught to correct their posture based on visual information from vertical objects in their environment.[12] Indeed, Zhang et al. reported an instant improvement in pushing behavior with the application of the visual deprivation on the contralesional eye in 2 PS patients and suggested that visual input modification may be effective in PS rehabilitation.[41] Broetz et al. reported the positive results of visual feedback therapy in the first 3.5 weeks of stroke in 8 PS patients. Their method consisted of 3 domains: raising awareness of tilted posture, promoting upright posture by orienting to the visual feedback from vertical objects, and reaching activities with non-paretic hand by shifting weight towards the ipsilesional side.[42] However, few randomized controlled trials are addressing visual feedback interventions in PS patients with chronic stroke. Yang et al. compared the effects of 20-minute computer-based visual feedback vs. mirror visual feedback therapy on contraversive pushing, motor control and balance in 12 chronic PS patients. They found favorable results in both groups.[43]

Another approach in PS rehabilitation is to improve postural control via modulating proprioceptive verticality perception. Sophisticated methods have been proposed to improve the somatosensory of body verticality. An et al. showed that whole-body tilting training was more effective than conventional postural training on BLS scores and balance in subacute stroke patients with PS. In this study, the Spine Balance 3D (CyberMedic Co., Iksan, Korea), a device that performs postural training via multidirectional tilting in predefined angles with visual feedback, was used. The device allows bipedal postural tilting exercises in patients who cannot stand unsupported via stabilizing the pelvis and lower extremities. It has been suggested that trunk muscles can be better activated than conventional therapy by providing multidimensional gravitational inputs.[44]

Mobility assisting methods including robot-assisted gait training that stabilizes the patient's posture and allows walking on paretic extremities may be beneficial in PS rehabilitation. Krewer et al. showed that a single session of Lokomat (a driven gait orthosis) training can reduce lateropulsion severity in subacute stroke patients.[45] Similarly, 2 randomized controlled trials reported that approximately 15 sessions of Lokomat therapy in PS patients with subacute stroke were superior to conventional physiotherapy in improving BLS score and balance.[46,47] These methods are supposed to improve the somatic graviception and verticality perception via simulating walking in a safe and upright posture in patients with PS.

CONCLUSION

PS is a phenomenon characterized by impaired perception and internal modeling of body verticality resulting in abnormal postural response to gravity. It is more common in right hemisphere lesions involving deep brain regions such as the thalamus and insula. PS is associated with a longer length of stay, poor functionality, and delayed response to rehabilitation. It negatively affects posture, balance, and mobility, independent of motor recovery. Both BLS and SCP are valid and reliable tests to evaluate PS in clinical settings. Various therapeutic interventions aimed at improving contraversive pushing with lateropulsion in subacute stroke patients have been suggested to be beneficial. However, there is no consensus on the optimal rehabilitation method for chronic PS patients. Randomized controlled studies are needed to reveal more effective rehabilitation methods.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

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