Recurrent Falls Associated with Lower Limb Deep Vein Thrombosis

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Dear Editor,

Falls in elderly patients are relatively common, and their risk factors including motor weakness, history of falls, taking many types of drugs, musculoskeletal problems, depressive state, aged >80 years, gait disturbance, cognitive impairment, visual field defect, and limited activities of daily living.¹ Moreover, buckling of the knee has been known to be associated with osteoarthritis (OA) or functional gait disorder.^{2,3} However, to our knowledge, there has been no previous report of falls accompanied with knee buckling being associated with lower limb deep vein thrombosis (DVT). Herein we report a case of bilateral lower limb DVT in a patient with buckling of both knees causing frequent falls.

An 87-year-old man was admitted to our hospital because of recurrent falls, which had occurred twice on the day before and twice on the day of hospitalization. Each fall was typically followed by abrupt bending of both knees, and he also experienced transient weakness around both knees while walking. No other associated symptoms including dizziness, palpitation, or loss of consciousness was observed. He received medication at the Department of Pulmonology for chronic obstructive pulmonary disease (COPD), which had opened 2 years previously. Three months previously he had received emergency treatment for exacerbation of COPD in the same department for 10 days. Additionally, 2 weeks before his admission to our department he was prescribed drugs for watery diarrhea with abdominal pain for 5 days in the Department of Gastroenterology.

The results of a neurologic examination and routine blood tests were unremarkable. He had moderate-to-severe OA in both knees without any pain or limitation of joint range of motion. The D-dimer titer had been 402 ng/mL (normal: 0–500 ng/mL) 3 months previously, it was now notably elevated to 1,763 ng/mL. The results of neurologic evaluations including brain diffusion-weighted imaging, computed tomography (CT) angiography for the brain and neck, electroencephalogram, and whole-spine magnetic resonance imaging were unremarkable. There was no significant abnormality in cardiac evaluations including transthoracic echocardiogram, 24-hour Holter monitoring, or ankle-brachial index. The CT venography revealed typical filling defects in the bilateral femoral and popliteal veins that indicated DVT in both legs (Fig. 1). He received oral anticoagulant (apixaban, ELIQUIS) and was supplied with compression stockings. He was discharged after 10 days without any recurrent falls, at which time the D-dimer titer had normalized.

The patient didn't exhibit characteristic symptoms of DVT such as pain, edema, or heat sensation in his legs,⁴ which made it difficult to diagnose this condition. However, applying CT venography because of the elevated D-dimer level resulted in a diagnosis of bilateral lower limb DVT. Moreover, we excluded other etiologies including transient ischemic attack, stroke, seizure, spinal cord lesion and cardiogenic problems, and peripheral artery disease, which could increase the number of falls. We therefore supposed that the recurrent falls of the patient could be the presenting symptom of bilateral lower limb DVT.

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Fig. 1. Computed tomography venography findings. Intraluminal filling defects are observed in both femoral veins (A, white arrows) and both popliteal veins (B, open arrows).

It is likely that the DVT was triggered by the immobilization of the patient during previous hospitalization for treatment of COPD, and this could have been aggravated because of a recent episode of watery diarrhea inducing a considerable loss of blood volume. Although the patient had OA in both knees, recurrent falls had not occurred before developing DVT, and these falls disappeared following DVT treatment. Furthermore, the patient did not experience any recurrence of falls during a 4-month follow-up. We therefore supposed that the frequent falls with knee buckling occurred in association with OA and bilateral DVT. Considering that knee buckling disappeared with anticoagulation, the DVT

may have triggered this symptom. However, the exact mechanism underlying how DVT triggered knee buckling—which is usually caused by OA—remains uncertain. A possible explanation is venous stasis in DVT that could be caused by increased blood viscosity.⁵ The hypoxemia caused by such venous stasis in the lower extremities might induce transient weakness in both legs while walking that also results in falls.

In conclusion, the current case suggests that lower limb DVT should be considered when performing differential diagnoses of falls.

Conflicts of Interest

The authors have no financial conflicts of interest.

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