

CASE REPORT

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A case of ischemic stroke in the basal ganglia presenting with fecal and urinary incontinence as initial clinical manifestations

Houde Li¹ and Jiaxin Dai^{2*}

Abstract

Limb disorders and slurred speech are common clinical symptoms associated with acute ischemic stroke. Although urinary incontinence is a known symptom in many cases of acute ischemic stroke, the simultaneous impairment of both bowel and bladder function is relatively rare. The occurrence of fecal and urinary incontinence as the primary clinical manifestation in minor acute ischemic stroke is especially uncommon. In this study, we present the case of a 67-year-old male patient who experienced a minor acute ischemic stroke in the basal ganglia region, notably presenting with both fecal and urinary incontinence. We also review the relevant literature to explore the potential causes behind this unusual presentation.

Keywords Acute ischemic stroke, Fecal and urinary incontinence, Neurological impairment

Background

Acute ischemic stroke is a prevalent cerebrovascular disease characterized by sudden onset and potential for severe neurological impairment. Among its various clinical presentations, acute ischemic stroke in the basal ganglia presenting with both fecal and urinary incontinence is an exceedingly rare condition that significantly impacts patients' quality of life. While urinary incontinence is commonly associated with large or critical cerebral infarctions involving regions such as the brainstem or frontal lobe—both of which are closely linked to excretory function—the etiology behind the concurrent impairment of stool and urine function remains elusive. This study presents a case of minor acute ischemic stroke

in the basal ganglia with fecal and urinary incontinence as the primary clinical manifestations. We also review the relevant literature and explore the potential mechanisms underlying this rare symptom.

Case presentation

A 67-year-old male patient presented with an acute cerebral infarction in the right basal ganglia. Upon admission, he primarily exhibited fecal and urinary incontinence, left-sided limb weakness, and slurred speech. Clothing residue from urine and bowel movements was observed upon arrival. Notably, there was no numbness or cognitive dysfunction present. The patient had a history of cerebral infarction and had been on long-term treatment with aspirin and rosuvastatin calcium. Additionally, he had a history of polycythemia vera and had been receiving maintenance treatment with human interferon alpha 2b injections.

The neurological examination revealed an National Institute of Health Stroke Scale (NIHSS) score of 2 points for the patient, with 1 point attributed to facial paralysis

*Correspondence:

Jiaxin Dai

daijiaxin1001@163.com

¹Department of Neurology, The Nuclear Industry 417 Hospital, Xi'an 710600, Shaanxi Province, China

²Department of General Internal Medicine, The Nuclear Industry 417 Hospital, Xi'an 710600, Shaanxi Province, China



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and another for dysarthria. The patient's cognitive status was normal, and they were conscious with impaired speech. On examination, the left nasolabial fold was shallow, the mouth corner was deviated to the right, and the tongue was midline. The muscle strength in the left limbs was graded at level 4, while the right limbs showed normal strength at level 5. Babinski's sign was observed on the left side. No other significant positive neurological signs were noted.

Laboratory tests revealed no significant abnormalities. Craniocerebral magnetic resonance imaging (MRI) revealed new infarcts in the right basal ganglia and adjacent to the lateral ventricle, as well as residual lesions from a previous cerebral infarction in the left basal ganglia and near the lateral ventricle (Fig. 1). There was mild stenosis observed in the M1 segment of the right middle cerebral artery. Brain CT angiography (CTA) confirmed moderate lumen stenosis in the same arterial segment (Fig. 2). Urinary ultrasound identified a left renal cyst and prostatic hyperplasia. No significant abnormalities were detected in the holter electrocardiogram or echocardiography.

In terms of treatment, the patient was administered aspirin (100 mg) in combination with clopidogrel (75 mg) for antiplatelet aggregation, and rosuvastatin calcium tablets to regulate blood lipids, improve circulation, and eliminate oxygen free radicals. The patient was also

advised to urinate regularly, adjust his diet, and undergo defecation training. By the 5th day of hospitalization, the patient's fecal and urinary incontinence had improved, although some incontinence persisted. Notably, the symptoms of limb weakness and speech were significantly improved. On the 10th day, the patient's symptoms had improved markedly, leading to his discharge. At discharge, the patient's fecal and urinary incontinence were largely resolved. The neurological examination at discharge revealed an NIHSS score of 1 (due to facial paralysis) and modified Rankin Scale (mRS) score of 1. The left nasolabial fold was shallow, the mouth corner was deviated to the right, and the tongue was midline. The left limb muscle strength was graded at level 4+, while the right limb muscle strength was level 5. Babinski's sign was observed on the left side. No other significant positive neurological signs were noted. Two weeks after discharge, the patient had essentially regained control of his bowel and bladder functions, with no further episodes of incontinence. However, he still reported some lack of flexibility in the movement of his left limb.

Discussion

Acute ischemic stroke typically manifests with symptoms such as unilateral limb weakness or numbness, slurred speech, facial distortion, staring, dizziness, and disturbances in consciousness. Patients in the severe category

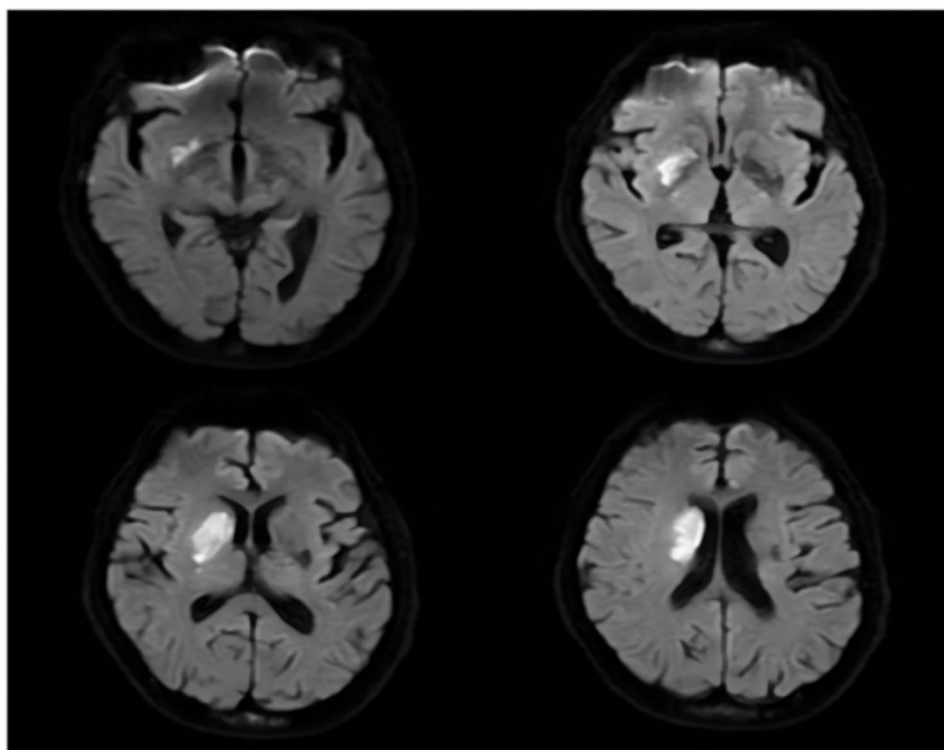


Fig. 1 Diffusion-weighted magnetic resonance imaging shows hyperintensity over the right basal ganglia and beside the lateral ventricle, while hypointensity is observed over the left basal ganglia and beside the lateral ventricle (Residual lesions of previous cerebral infarction)

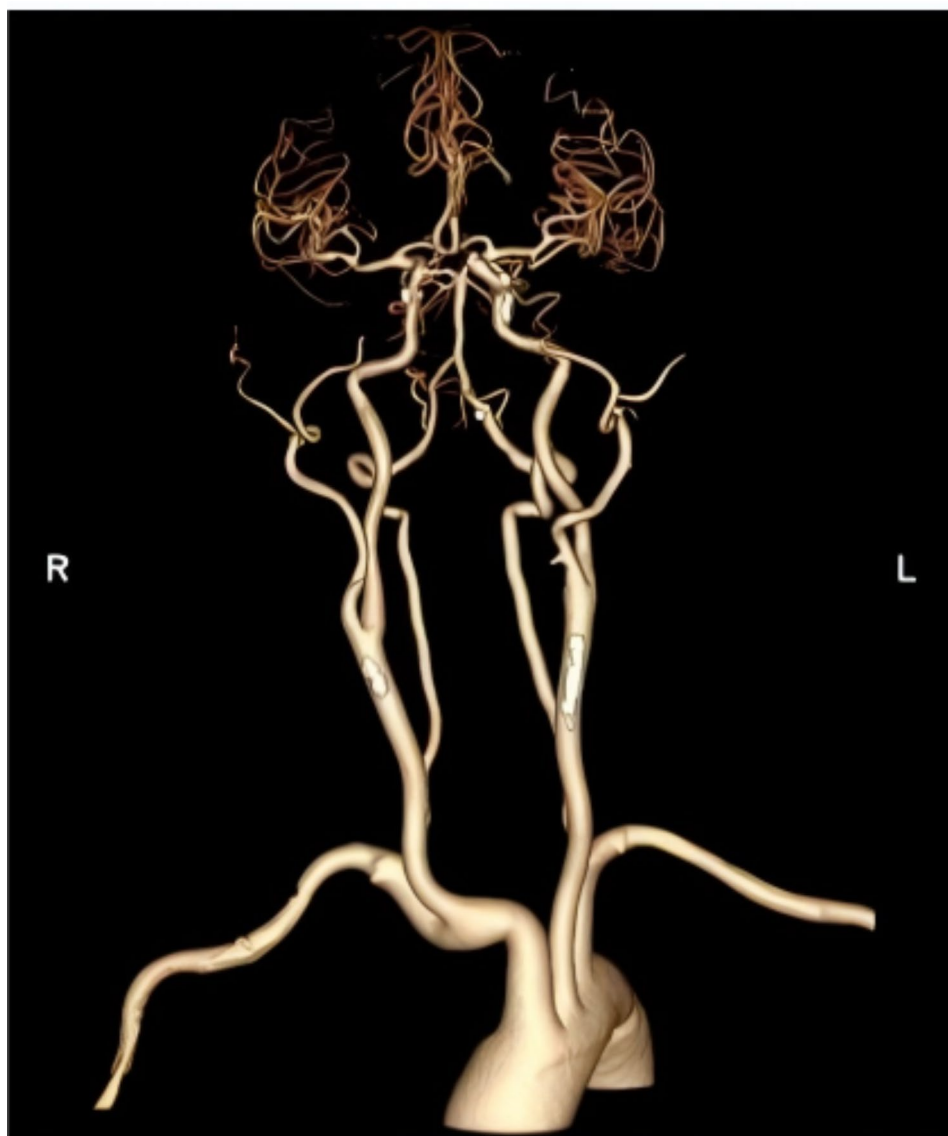


Fig. 2 CTA imaging shows moderate stenosis in the M1 segment of the right middle cerebral artery

often exhibit fecal and urinary incontinence. It is relatively rare for patients with minor acute ischemic stroke in the basal ganglia to present with fecal and urinary incontinence as their initial clinical signs. In this study, the patient had a history of polycythemia vera, which should be taken into consideration when making the differential diagnosis of the cause of ischemic stroke. Previous literature has pointed out that hematological diseases are a commonly unrecognized cause of ischemic stroke, which may be overlooked in clinical practice and need to be taken seriously by clinicians [1]. The prevalence of urinary incontinence among stroke patients is approximately 38%. Factors such as age, lesion location (parietal, frontal, and temporal lobes), high NIHSS scores, and Oxfordshire Community Stroke Project (OCSP) grades (total anterior circulation infarction) have been identified

as risk factors for urinary incontinence in these patients [2]. Approximately 6.4% of patients are diagnosed with fecal incontinence within 72 ± 24 h post-stroke onset. Fecal incontinence is more prevalent in hemorrhagic strokes and in cases of more severe strokes; patients with an NIHSS score of 12 or higher are at an increased risk of developing fecal incontinence [3]. In this particular case, the patient suffered an acute ischemic stroke in the basal ganglia, with an NIHSS score of only 2 points. According to existing literature, this patient should have been at a low risk for developing fecal and urinary incontinence. What factors might account for the presence of fecal and urinary incontinence in this patient?

The International Urinary Incontinence Association defines urinary incontinence as an objectively verifiable, involuntary transurethral urinary leakage [4]. Gelber et

al. analyzed three primary mechanisms of urinary incontinence following stroke: (1) disruption of neural pathways involved in urination; (2) urinary incontinence due to cognitive and language impairments related to stroke; and (3) neuropathy or medication side effects [5]. Urinary incontinence can occur in stroke patients, particularly those with lesions in the basal ganglia region. Recurrent cerebral infarcts may lead to pseudobulbar syndrome, characterized by symptoms such as dysarthria, dysphagia, and mimic disturbances, and is often associated with involuntary urinary urgency or incontinence [6]. Fecal incontinence is defined as any degree of involuntary intestinal leakage, and its mechanisms in stroke patients are not yet fully understood [7, 8]. The maintenance of bowel function is a complex process that involves the coordination of pelvic motion, visceral, and sensory functions. Fecal incontinence may result from anal sphincter dysfunction, abnormal rectal compliance, reduced rectal sensation, or a combination of these factors [9, 10]. The basal ganglia, a critical deep-brain region, plays a significant role in motor control, cognitive function, and emotion regulation. Damage to the basal ganglia can potentially disrupt the nerve pathways associated with bowel control, leading to bowel dysfunction. Similarly, the basal ganglia exert a substantial influence on motor control, including that of the anal sphincter. Damage in this area may result in reduced bowel control, potentially causing fecal incontinence. Currently, there is no definitive knowledge regarding the specific cortical region responsible for fecal incontinence (the “incontinence center”), necessitating further anatomical and radiological functional studies.

Treatment options for urinary incontinence encompass a variety of approaches, including scheduled urination, pelvic floor muscle training, acupuncture, and pharmacological therapy. Post-stroke urinary incontinence and associated symptoms can be mitigated through structured nursing assessments, professional management, and specialized incontinence care [11]. Upon diagnosing fecal incontinence, treatment strategies may involve lifestyle modifications, dietary advice, basic behavioral guidance (like toilet/bowel training), the use of stool dilators and/or anti-diarrheal medications, pelvic floor muscle exercises, absorbent products for containment, and skin-care products to address anal skin irritation [12].

Acute stroke in the basal ganglia presenting with fecal and urinary incontinence as the initial clinical signs is relatively uncommon, and the underlying pathogenesis warrants further investigation. In terms of treatment, not only is the physician's diagnosis and intervention necessary, but also the collaboration of the nursing team is essential. Increased attention should be given to patients experiencing fecal and urinary incontinence due to stroke.

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Author contributions

Houde Li and Jiaxin Dai wrote the main manuscript text and Jiaxin Dai prepared Figs. 1 and 2. All authors reviewed the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The informed consent to participate was obtained from all participants and written informed consent in accordance with the Declaration of Helsinki. This study was performed in accordance with the appropriate guidelines and regulations. The study protocols were approved by the ethics committees of The Nuclear Industry 417 Hospital.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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