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Letter to Editor

Post-Stroke Incomplete Evacuation of Urinary Bladder: Are we Missing Something?

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DEAR EDITOR,

A 68-year-old diabetic man with benign hypertrophy of the prostate presented with acute stroke involving the right cerebellar hemisphere, right lateral pons, and medulla. He had ataxia, dysarthria, and a mild sensory deficit (NIHSS of 4). He was beyond the window for acute stroke intervention when he attended the emergency room. Standard stroke medications along with stroke rehabilitation were started. We regularly mobilised the patient out of bed and planned for his discharge with an NIHSS of 2. We established large vessel atherosclerosis as the stroke mechanism (vertebral and basilar arteries were diseased). A final metabolic parameter detection just before discharge revealed a sharp rise of creatinine from 0.9 milligrammes/decilitre to 3.8 milligrammes/decilitre over six days. We catheterized him, and 1700 millilitres of urine were evacuated within the next 12 hours, leading to a fall of creatinine from 3.8 milligrammes/decilitre to 1.2 milligrammes/decilitre within a span of two days. He was not on any nephrotoxic drugs or had any convulsive episodes in the hospital, which could have caused the sudden creatinine rise. There was no evidence of a urinary tract infection, either. We concluded that although he passed urine regularly, there used to be incomplete urinary evacuation, and some amount of urine used to accumulate on each occasion, leading to this unfortunate situation. He used to have satisfactory urodynamic studies before admission, and renal function used to be stable on his regular drugs for his obstructive uropathy, which were continued post-admission. He did not have any cognitive impairment anytime post-stroke and never complained of symptoms related to urinary retention or had symptoms of fluid overload in his body. We should have been more vigilant regarding this posterior fossa stroke and checked post-void urine routinely to avoid this unavoidable situation.

Stroke may cause some changes in the body, which in turn may influence the stroke outcome. One of them is sphincter dysfunction, a common but often underrecognized incident. The incidence of urinary retention ranges from 29% to 56% in patients with acute stroke [1].

Most of the older people who come in for a stroke already have problems with their sphincters because they have dementia, previous strokes, neurodegenerative diseases like parkinsonism, or bladder outlet obstructions like benign hypertrophy of the prostrate/ urethral stricture. Stroke directly involving any part responsible for sphincter control (the pontine micturition centre in the pons, the periaqueductal grey in the midbrain, higher brain centres like the prefrontal cortex and cingulate cortex) or oedema extending to these areas or increased intracranial pressure may all cause urinary retention.

Due to less space for accommodating anything extra, even smaller strokes involving the posterior fossa (like in our case) may contribute to raised intracranial pressure and dysfunction of the pontine micturition centre/periaqueductal grey matter secondary to compression of the brainstem. Immobility post-stroke further contributes to urinary retention; constipation, which is not uncommon in these patients, also contributes to secondary urinary retention due to

compression from loaded bowels/rectum. An incompletely evacuated bladder, on the other hand, also contributes to bowel retention and causes poor outcomes because of raised intracranial pressure secondary to straining. Many of these elderly patients either cannot appreciate bladder fullness due to afferent sensory neuropathy or secondary to cognitive impairment, making the situation worse.

Now incomplete urinary evacuation or retention not only invites infection but also contributes to raised intracranial pressure secondary to retrograde pressure and less venous return from the brain, and also causes fluid imbalance. These, along with straining to evacuate bowel/bladder, may cause hypertensive surges, thereby jeopardising the autoregulatory mechanism of CNS or causing rebleeding in patients with intracerebral haemorrhage, both contributing to worse stroke outcomes. Hence, in all non-lacunar strokes, especially in the elderly, we need to have a very low threshold for Foley catheterization to avoid all these complications.

Though Foley catheter may precipitate bacteriuria at a rate of 3% to 7% daily, it can prevent infection by avoiding urinary retention [2].

We also need to remember acute stroke patients present with urinary tract infections in 10% to 19% of cases, sometimes leading to incomplete urinary evacuation, and Foley catheterization in them may alleviate clinical symptoms [3].

Sometimes we may need to convince conscious stroke patients (like in our case) who may be reluctant to give consent for catheterization, as it will not only help in achieving better stroke outcomes but also ensure maintaining a proper fluid balance by recording exact fluid intake and output. There were studies where the decision for catheterization was taken based on post-void residual urine guided by ultrasound [4].

But such an approach may be individualised because stroke is a dynamic phenomenon, and the situation may change on a day-to-day basis. The initial residual urine may not remain the same throughout the course of this acute illness and during the hospitalisation period. It may also not be feasible and practically possible in all centres to perform ultrasound on a daily basis for such decision-making.

Hence, it is justified to put Foley catheter in stroke patients where brain centres commonly associated with sphincter functions, patients with considerable raised intracranial pressure, those with cognitive impairment, and those with bladder outlet obstructions. Our case highlights how an incomplete urinary bladder evacuation (not an obvious and classical urinary retention) can silently cause deterioration in a stroke patient and influence the outcome.

To avoid catheter-related infections, which become more common over time, we need to make sure that the catheterization is done in an aseptic way, that the drainage system stays closed, that the urobag is emptied regularly (before it gets full). We need to remove the catheter as soon as possible, preferably within five days. We need to judge clinically and brain imaging-wise and do a post-void residual urine volume detection after the Foley catheter is removed prior to discharging the patient from the hospital to avoid future complications.

Support: NIL.

Conflicts of Interest: NIL.

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