Right Tegmental Hemorrhage with Urinary Retention: A Case Report

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Abstract
The upper brainstem tegmentum is dense and complex, making it difficult to localize functions to specific subregions. In particular, the precise location and possible laterality of subregions supporting basic functions like consciousness and urinary continence remain unclear. Here, we describe a patient who presented with a right pontine tegmental syndrome caused by intraparenchymal hemorrhage. Despite hemorrhage extension into the fourth ventricle and expansion of both hemorrhage and edema into a large region of the caudal midbrain and right-sided pontine tegmentum, this patient did not lose consciousness. Instead, he developed new and total urinary retention, with residual bladder volumes of more than 1,000 mL. We conclude that injury to the right pontine tegmentum is sufficient to disrupt the micturition reflex pathway.

Introduction
The upper pons tegmentum contains neurons that project to the spinal cord and allow urination. This reflex pathway was discovered in the 1920s, when Barrington made stereotaxic lesions in this region of the cat brainstem and observed total urinary retention [1]. While this effect required bilateral lesions in experimental animals, urinary retention has been reported in human patients with both bilateral and right-sided lesions in the pontine tegmentum [2, 3].
The precise location and lateralization of the human "pontine micturition center" is not completely understood, in part because injury to this region of the brainstem tegmentum frequently disrupts consciousness [4, 5]. The few patients who do not lose consciousness after lesions in this region hold valuable insights that could inform our understanding of functional localization and lateralization in the pontine tegmentum [6, 7].

**Case Report**

A 70-year-old man with hypertension and atrial fibrillation, taking rivaroxaban for embolic risk reduction, complained of sudden-onset left-sided sensory changes, imbalance, and dizziness. Initial examination at an outside hospital revealed mild gait instability. The initial head CT at that hospital (Fig. 1a) identified a small, hyperdense lesion in the rostral pons, centered at the junction of the base and tegmentum.

During ambulance transfer to our tertiary care hospital, his speech became dysarthric. He also developed a complex ophthalmoplegia (including right one-and-a-half syndrome), right ptosis, right facial paresis (upper and lower face), and contralateral sensory loss (left face, arm, and leg) with weakness in the left arm and leg. There was no ocular bobbing, vertical nystagmus, or "ping-pong" horizontal eye movements, and his pupils remained 3 mm and equally reactive to light. A second head CT at our hospital (Fig. 1b), performed 2 h after the initial scan, revealed expansion of the hemorrhage to 2.5 × 1.5 cm. Blood had dissected through the right pontine tegmentum and into the midbrain. On the following day, magnetic
resonance imaging (FLAIR images shown in Fig. 1d–g) revealed surrounding edema in the midbrain and pons, partial compression of the fourth ventricle, intraventricular blood, and blood in the interpeduncular fossa.

Although transiently somnolent on the day of transfer, he never lost consciousness and remained fully oriented with appropriate responses to questions and commands through discharge, 12 days later. His left arm and leg weakness largely recovered before discharge. However, from admission through discharge, he was unable to void his bladder. He denied previous urinary retention. Despite reporting a sensation of bladder fullness and attempting to void, he failed 2 trials of spontaneous voiding, on hospital days 2 and 9, with residual bladder volumes of 1,100 and 1,050 mL. His urinary retention was managed initially with a Foley catheter, then by intermittent straight catheterization. After discharge, 2 weeks into his stay at a rehabilitation hospital (1 month after the hemorrhage), volitional voiding recovered to the point that he no longer required catheterization. A follow-up CT one month later (Fig. 1c) revealed a hypodense 1-cm patch centered over the junction between the right pontine tegmentum and base.

**Discussion/Conclusion**

Beyond his crossed deficits (indicating damage to cranial nerve nuclei VI–VII, the medial lemniscus, and the corticospinal tract), this patient is remarkable for 2 reasons. First, despite a relatively large hemorrhage – dissecting through the right pons-midbrain tegmentum, with edema extending partly across the midline and well into the midbrain – he never lost consciousness. Hemorrhages and other lesions in the pons-midbrain tegmentum frequently produce coma [4, 5], but loss of consciousness may be more common after bilateral or left-sided lesions [8, 9].

Second, his unilateral lesion caused new and total urinary retention. Urinary retention has been reported following strokes in the pontine tegmentum that may injure neurons in Barrington’s nucleus, also known as the pontine micturition center [7]. Urinary retention may also occur after strokes in the lateral medulla that damage the axonal projections from Barrington’s nucleus to the spinal cord [10]. The ability of right-sided lesions to cause new and total urinary retention in our patient raises the possibility that the neural pathway for micturition lateralizes to the right. Supporting this possibility, total urinary retention was reported in a patient with a unilateral inflammatory lesion in the right pontine tegmentum [2], and a PET imaging study in healthy adults identified a right-lateralized change in cerebral blood flow in this region during micturition [11]. However, we cannot exclude the possibility that right- and left-sided lesions similarly disrupt voiding, and that urinary retention goes undetected in unconscious (presumably catheterized) patients after injury to the left pontine tegmentum.

In summary, we present the case of a 70-year-old man who developed new and total urinary retention with a hemorrhagic stroke involving the right pontine tegmentum. More evidence is needed to clarify localization and lateralization, but this rare case adds to previous, limited evidence suggesting a right-lateralization of micturition circuitry in the human brainstem.

**Statement of Ethics**

This research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. The patient gave written informed consent to publish this case (including images). Ethical approval was not required for this study.
Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

M.M.T. and J.C.G. drafted and edited the paper. M.M.T. and J.C.G. drafted and edited the figure. A.D.B. first identified this case and collaborated in the conceptualization and writing of this report. All authors edited and approved the final manuscript.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Further enquiries can be directed to the corresponding author.

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