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Persistent delirium after acute ischemic stroke in a patient with Parkinson's- plus syndrome: a multifactorial diagnostic challenge — case report

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Case Report

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Abstract

Background

Delirium is a frequent complication among elderly patients with acute ischemic stroke, particularly in those with underlying neurodegenerative disorders such as Parkinson's-plus syndromes. In many cases, delirium is multifactorial and may persist despite treatment of apparent precipitating causes such as infection.

Case presentation

A 75-year-old female with Parkinson's-plus syndrome and prior cerebrovascular disease presented with persistent delirium characterized by abnormal behavior, irrelevant speech, and fluctuating attention. Symptoms began approximately ten days before her first hospitalization, where encephalitis and urinary tract infection were initially suspected. Subsequent evaluation demonstrated an acute ischemic infarct in the left parietal lobe on a background of chronic cerebrovascular disease with cortical atrophy. Urine cultures grew *Enterococcus faecalis* and *Pseudomonas* species and were treated with culture-guided antimicrobial therapy. Despite improvement in inflammatory markers, delirium persisted and improved only gradually.

Conclusions

Persistent delirium after stroke often reflects the interaction of structural brain disease, neurodegeneration, infection, and systemic illness. Clinicians should evaluate multiple contributing factors rather than attributing delirium to a single cause.

Background

Delirium is an acute disorder of attention and cognition commonly encountered among hospitalized older adults [1, 2]. Patients with Parkinson's-plus syndromes are particularly vulnerable because baseline neurodegeneration reduces cognitive reserve, making them more susceptible to prolonged or refractory delirium when acute systemic or cerebral insults occur [3].

The syndrome is multifactorial and often reflects the combined effects of neurological injury, infection, metabolic disturbance, and medication exposure [4]. Delirium is associated with prolonged hospitalization, functional decline, and worse long-term cognitive outcomes [5]. It is also especially common following acute stroke [6].

The coexistence of acute stroke, chronic cerebrovascular disease, Parkinsonian neurodegeneration, and systemic infection creates a complex clinical scenario in which the causes of delirium may be difficult to disentangle.

This report highlights the diagnostic complexity of persistent delirium when acute ischemic stroke, infection, and pre-existing neurodegenerative disease coexist.

Case presentation

A 75-year-old female with Parkinson's-plus syndrome, prior cerebrovascular disease, type 2 diabetes mellitus, hypertension, osteoporosis, and tinea corporis developed abnormal behavior and irrelevant speech approximately ten days before her first hospital admission on 22 February 2026.

During that admission at an outside facility, encephalitis and urinary tract infection were suspected, although confirmatory records were unavailable. A discharge document listed encephalitis, urinary tract infection, prior stroke, diabetes mellitus, hypertension, osteoporosis, and tinea corporis among the diagnoses.

During the course of illness, the patient developed sudden onset speech difficulty and right-sided weakness while being fed, suggesting an acute cerebrovascular event. She was subsequently transferred for further evaluation.

Delirium was present from the outset of the current admission and was characterized by fluctuating consciousness, inattention, disorganized thinking, and agitation. On neurological examination she had right-sided hemiparesis with ipsilateral facial deviation. Although the facial deviation was atypical for an acute unilateral stroke, it was considered likely related to previous cerebrovascular injury and pre-existing neurological deficits.

Symptoms began around 12 February 2026 with abnormal behavior and irrelevant speech. MRI performed on 12 March 2026 confirmed an acute left parietal infarct. Urine cultures obtained on 12 and 13 March 2026 grew *Enterococcus faecalis* and subsequently *Enterococcus faecalis* with *Pseudomonas* species.

Meropenem therapy was initiated on 17 March 2026 according to culture sensitivity results. Ultrasound of the abdomen on 18 March 2026 and CT KUB on 20 March 2026 showed no evidence of obstructive uropathy.

Overall, the clinical findings were consistent with acute ischemic stroke complicated by multifactorial delirium in the setting of underlying Parkinson's-plus syndrome, chronic cerebrovascular disease, and systemic infection.

Timeline

12 February 2026 — Onset of abnormal behavior and irrelevant speech.

22 February 2026 — First hospital admission; encephalitis and urinary tract infection suspected.

During illness — Sudden onset speech difficulty and right-sided weakness suggesting acute stroke.

12 March 2026 — MRI brain confirmed acute left parietal infarct.

12–13 March 2026 — Urine cultures grew Enterococcus faecalis followed by Enterococcus faecalis with Pseudomonas species.

17 March 2026 — Intravenous meropenem initiated according to culture sensitivity.

18 March 2026 — Ultrasound abdomen showed no obstructive uropathy.

20 March 2026 — CT KUB confirmed no obstruction and demonstrated a right renal cortical cyst.

Investigations

MRI of the brain demonstrated an acute infarct in the left parietal lobe with additional chronic infarcts in the left parietal and right parietotemporal regions, together with gliosis, encephalomalacic change, and generalized cortical atrophy. Representative MRI findings are shown in Fig. 1, and diffusion-weighted imaging findings are shown in Fig. 2.

Urine microscopy demonstrated significant pyuria and hematuria. Sequential urine cultures confirmed infection with *Enterococcus faecalis* and *Pseudomonas* species.

Ultrasound of the whole abdomen showed normal-sized kidneys with preserved corticomedullary differentiation, increased cortical echogenicity, mild cortical thinning, a right parapelvic or exophytic cyst, grade I left renal medullary nephrocalcinosis, and mild echogenic debris within the urinary bladder.

CT KUB confirmed normal kidney size without hydronephrosis or obstruction and demonstrated a right renal cortical cyst measuring approximately 37 × 34 mm. Coronal CT KUB findings are shown in Fig. 3.

Treatment

The patient was started on intravenous meropenem on 17 March 2026 according to culture sensitivity results. Supportive care included delirium precautions, renal monitoring, and continuation of baseline antiparkinsonian therapy.

Intermittent antipsychotic medication was administered for agitation.

No significant adverse effects related to antimicrobial therapy were observed during hospitalization.

Outcome and follow-up

Despite improvement in inflammatory markers and appropriate antimicrobial therapy, the patient's delirium persisted for a prolonged period and improved only gradually during the subsequent hospital course. Cognitive fluctuations remained present but became less severe over time.

Discussion

This case highlights the multifactorial and persistent nature of delirium in elderly patients with neurological vulnerability. The earliest available records indicated that symptoms began before the confirmed stroke diagnosis and were initially attributed to possible encephalitis and urinary infection.

Acute stroke is a recognized trigger for delirium, particularly when cortical injury occurs in patients with pre-existing cerebrovascular disease [6, 7]. In this patient, delirium persisted after the acute ischemic event, suggesting that infection alone could not explain the clinical picture.

Neuroimaging demonstrated multiple chronic infarcts and generalized cortical atrophy, both of which suggest reduced cognitive reserve and increased susceptibility to prolonged delirium [1, 5].

The catheter-associated urinary tract infection was microbiologically significant, with sequential cultures demonstrating *Enterococcus faecalis* followed by *Enterococcus faecalis* with *Pseudomonas* species. Although both organisms were sensitive to meropenem and appropriate therapy was initiated, delirium persisted. This temporal dissociation suggests that infection contributed to but did not solely account for the neuropsychiatric syndrome [1, 4, 8, 9].

Renal dysfunction during the acute illness may also have contributed to metabolic vulnerability. Although imaging did not demonstrate obstructive uropathy or advanced chronic kidney disease, transient kidney injury during systemic illness can worsen delirium in older adults [1, 4, 10].

In addition, medications used during treatment—including dopaminergic therapy and antipsychotics—may influence delirium in patients with Parkinson's-plus syndromes, as certain agents may exacerbate or prolong cognitive disturbance in susceptible individuals [3, 4, 11].

Overall, the most plausible explanation for this patient's persistent delirium is an interaction between acute stroke, chronic structural brain disease, Parkinsonian neurodegeneration, infection, and systemic illness.

This case illustrates how persistent delirium in elderly patients often results from the interaction of structural brain disease, neurodegeneration, and systemic illness rather than a single identifiable cause.

Patient perspective

A direct patient perspective could not be obtained because the patient remained cognitively impaired during the acute illness. The patient's legal guardian reported concern regarding the prolonged confusion

and behavioral changes but noted gradual improvement following treatment and supportive care.

Strengths and limitations

The strength of this case lies in the integration of clinical, radiological, and microbiological findings across a complex and evolving clinical course. It highlights the diagnostic challenge of persistent delirium in the presence of multiple contributing factors. Limitations include incomplete documentation from the initial outside-hospital admission, absence of standardized delirium severity scoring, and the inherent difficulty in establishing causality in a multifactorial condition. Despite these limitations, the temporal association of stroke, infection, and neurodegeneration supports a multifactorial etiology.

Conclusions

Persistent delirium following stroke in elderly patients is often multifactorial. Structural brain disease, neurodegenerative disorders, infection, and systemic illness may interact to produce prolonged neuropsychiatric symptoms. Clinicians should consider multiple contributing factors when evaluating persistent delirium after stroke.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent for publication of this case and the accompanying images could not be obtained from the patient due to impaired decision-making capacity related to persistent delirium. Therefore, written informed consent was obtained from the patient's next of kin (son). A copy of the written consent form is available for review by the Editor-in-Chief of this journal on request.

Availability of data and materials

All data generated or analyzed during this study are included in this published article. Additional details are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Khandaker Abdul Sadib was responsible for patient care, data collection, and manuscript preparation. Yeaomoon Fabiha contributed to clinical interpretation and critical revision of the manuscript. All authors approved the final manuscript.

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Figures

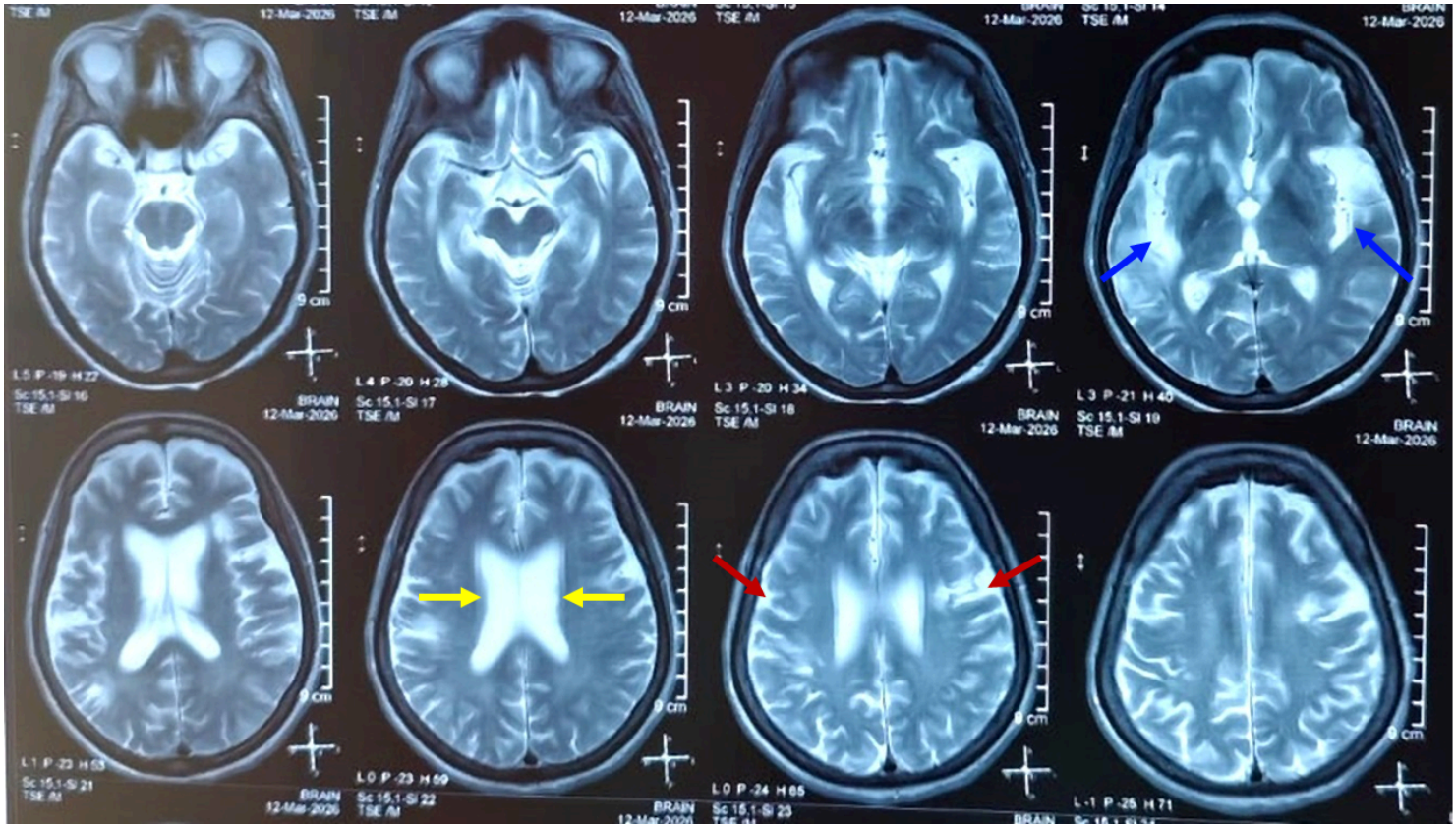


Figure 1

Axial T2-weighted MRI demonstrating cortical atrophy and chronic cerebrovascular changes. Red arrows indicate atrophied gyri with widened cortical sulci. Yellow arrows demonstrate compensatory symmetrical dilatation of the lateral ventricles. Blue arrows highlight symmetrically widened Sylvian fissures.

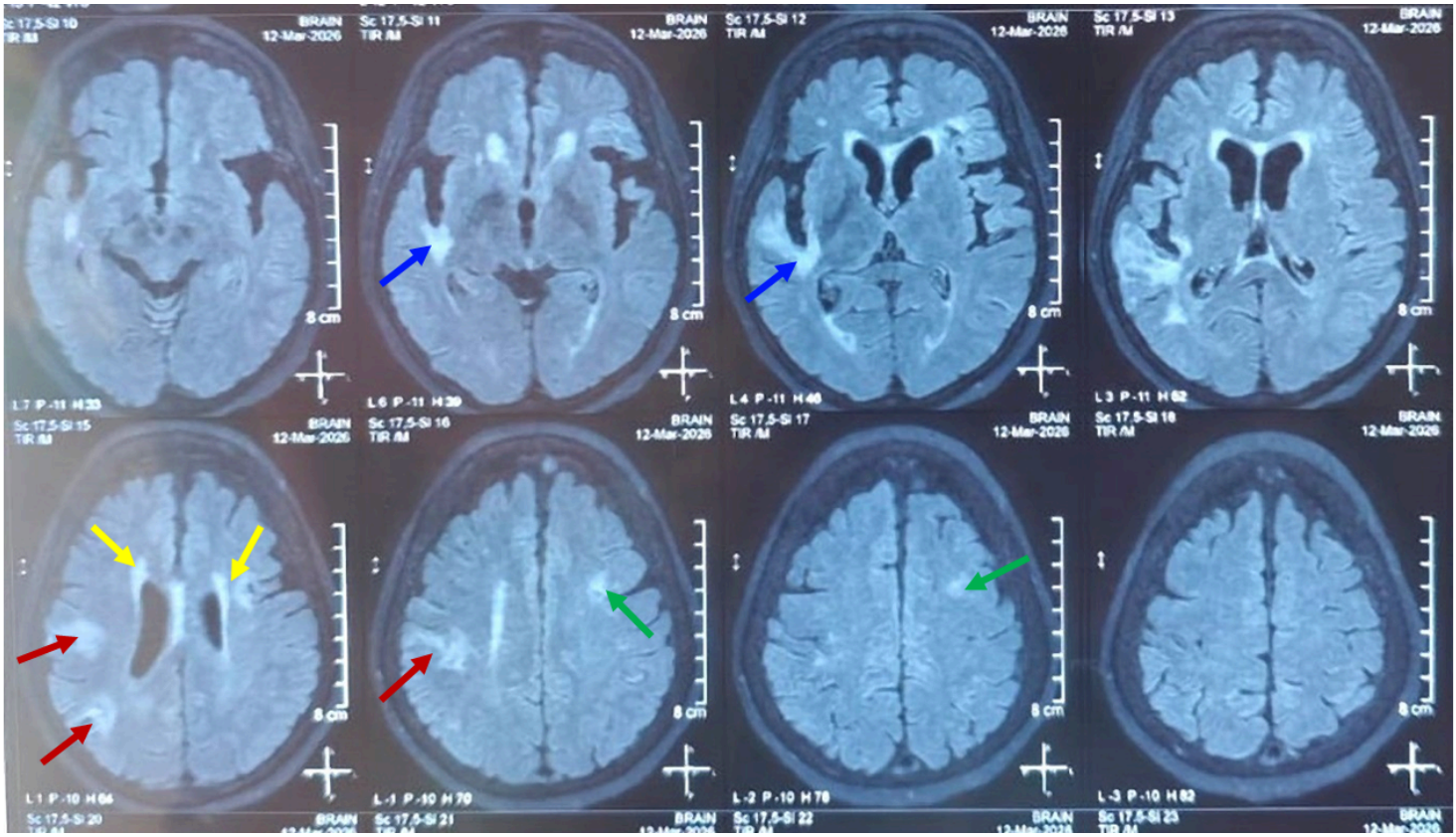


Figure 2

Diffusion-weighted imaging demonstrating multifocal ischemic changes. Red arrows indicate an acute infarct in the left parietal lobe. Blue arrows highlight areas of restricted diffusion in the temporal region. Yellow arrows indicate periventricular hyperintense foci representing chronic infarcts with gliosis. Green arrows denote additional chronic infarcts.

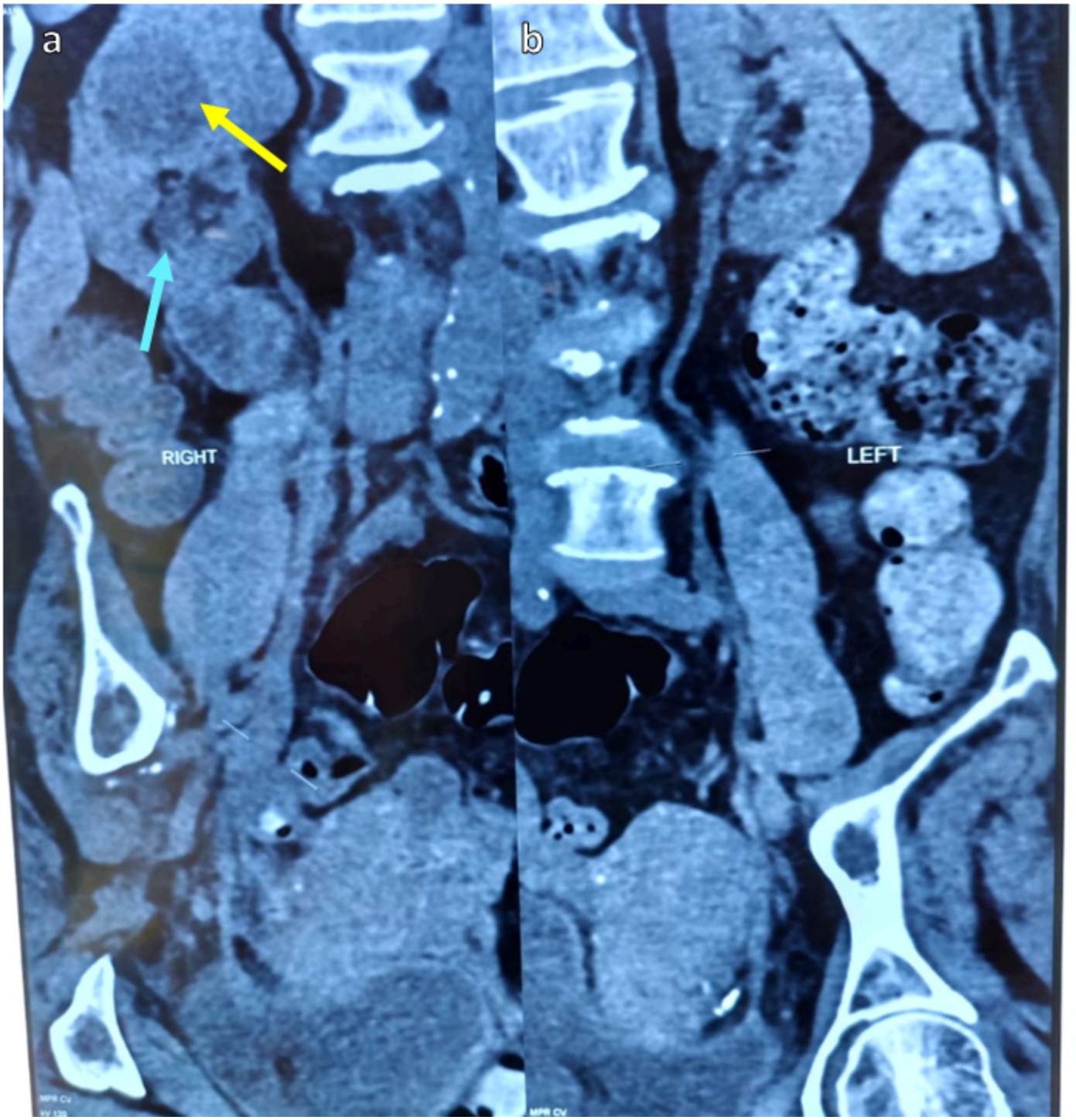


Figure 3

Coronal CT KUB demonstrating renal findings without obstructive uropathy. Yellow arrows indicate a right renal cortical cyst, while the pale blue arrow highlights an additional small hypodense lesion likely representing a small renal cyst.