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Case Report

When Infection Strikes the Basal Ganglia: UTI-Triggered Parkinsonian Dystonia

Running Title: UTI-triggered dystonia in Parkinson's disease

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A B S T R A C T

Urinary tract infection is a recognized precipitant of acute neurological deterioration in Parkinson's disease and may worsen motor symptoms, functional status, and cognition. We report a 71-year-old woman with Parkinson's disease and dystonia who developed fever, dysuria, urgency, frequency, suprapubic pain, reduced urine output, vomiting, delirium, and acute worsening of painful spasms, blepharospasm, dysarthria, gait freezing, stiffness, tremor, dyskinesia, rigidity, and inability to stand. Urine culture grew multidrug-resistant Klebsiella spp. with sensitivity only to amikacin and gentamicin, and inflammatory markers improved after targeted treatment. No increase in dopaminergic medication was required, and the motor deterioration improved after treatment of the infection, supporting UTI as the trigger.

Keywords: Parkinson's disease; dystonia; Urinary tract infection; Dyskinesia; Klebsiella; Infection-triggered worsening

Introduction

Parkinson's disease is frequently complicated by motor fluctuations and non-motor worsening during systemic illness, especially infection. Among infections, urinary tract infection is one of the most common and clinically important triggers because it may present with both urinary symptoms and acute motor deterioration[1]. In older patients with Parkinson's disease, infection-related inflammation, dehydration, and delirium can amplify tremor, rigidity, dyskinesia, and gait impairment[2]. This case is presented to emphasize the importance of early diagnosis of UTI when unexplained acute worsening occurs in a patient with Parkinson's disease[3].

Case Presentation

A 71-year-old woman, known to have Parkinson's disease since 2022, presented with fever of 102 F, burning micturition, dysuria, urinary frequency, urgency, suprapubic pain, reduced urine output, vomiting, and delirium. She also developed acute worsening of dystonia/parkinsonian symptoms, including painful spasms, blepharospasm, dysarthria, gait freezing, stiffness, tremor, dyskinesia, rigidity in all limbs, and inability to stand. The motor worsening began after the fever started and progressed over approximately 5 days, before the culture report became available. She was already receiving levodopa-carbidopa combination therapy and rivastigmine, without any recent dose escalation. At presentation, her blood pressure was 100/70 mmHg and pulse rate was 106/min, and she appeared dehydrated. Neurological examination revealed bradykinesia, excessive tremor, rigidity of the limbs, increased tone, and bilateral plantar flexor responses; gait could not be assessed because she was unable to stand. NCCT brain was normal, and there was no bladder tenderness. Urinalysis showed pale straw, slightly hazy urine with acidic pH, trace protein, trace blood, and marked pyuria of 35–40 pus cells/hpf. Serum fasting glucose was 177 mg/dL, sodium 142.4 mmol/L, potassium 2.83 mmol/L, urea 22.6 mg/dL, and creatinine 1.31 mg/dL. Liver tests showed low albumin and elevated alkaline phosphatase, reflecting the patient's systemic illness and comorbidity burden. Complete haemogram showed hemoglobin 10.2 g/dL, RBC count 3.39 million/cumm, hematocrit 33%, MCV 97.3 fL, MCH 30.1 pg, MCHC 30.9%, TLC 10,210/cumm, platelets 1.57 lakh/cmm, ESR 30 mm/hr, and neutrophils 73%. CRP was 21.52 mg/L on follow-up testing, and the marker had decreased serially from 77 to 21.52 and then to 3 mg/L after treatment, paralleling clinical recovery. Urine culture grew Klebsiella spp. with colony count above 100,000/mL. The organism was sensitive only to amikacin and gentamicin and resistant to amoxicillin-clavulanate, ampicillin, cefepime, ceftazidime, ceftriaxone, cefuroxime, ciprofloxacin, co-trimoxazole, imipenem, levofloxacin, meropenem, netilmicin, nitrofurantoin, norfloxacin, ofloxacin, piperacillin-tazobactam, ertapenem, doripenem, and doxycycline. The urinary isolate was treated according to culture and sensitivity results because the antibiogram showed extensive resistance. No increase in levodopa-carbidopa dosage was made, and the neurological worsening improved after the UTI was treated. Supportive care included correction of dehydration and management of the acute systemic illness. The response suggested that the acute dystonia/parkinsonian worsening was infection-triggered rather than a primary progression of Parkinson's disease.

Discussion

This case illustrates the important and sometimes underappreciated link between UTI and acute motor deterioration in Parkinson's disease. The patient developed urinary symptoms, fever, dehydration, delirium, and then marked worsening of dystonia and other parkinsonian features, which improved after treating the infection. That temporal sequence strongly supports a reversible trigger, rather than disease progression or medication failure. The fact that her symptoms improved without increasing antiparkinsonian drugs further strengthens the causal role of UTI[4].

The review by Hogg et al. notes that UTI is a common precipitant of acute hospitalization and motoric exacerbation in Parkinson's disease, and that infection-related inflammatory stress may underlie neurological deterioration. Delirium and high fever are also known to be associated with subacute motor deterioration in PD, and this deterioration may persist in some patients. In our patient, the combination of fever, dehydration, and multidrug-resistant Klebsiella likely amplified the acute motor syndrome. The clinical picture is consistent with a systemic infection-driven worsening of basal ganglia dysfunction, manifesting as exacerbated rigidity, tremor, dyskinesia, and dystonia[5].

This case is also important from an antimicrobial perspective. The isolate was resistant to most common oral and parenteral antibiotics and remained susceptible only to amikacin and gentamicin. Such a susceptibility profile highlights the value of urine culture before definitive treatment, especially in elderly hospitalized patients with comorbidities. Early targeted therapy likely prevented progression to severe sepsis and allowed neurological recovery.

Conclusion

UTI should be considered an urgent and reversible trigger when a patient with Parkinson's disease develops sudden worsening of dystonia, dyskinesia, rigidity, tremor, or gait failure. In this patient, febrile multidrug-resistant Klebsiella UTI precipitated acute motor deterioration that improved after culture-guided treatment without increasing dopaminergic therapy. Prompt recognition of infection and early microbiological confirmation are essential to prevent unnecessary neurologic escalation and improve outcomes in Parkinson's disease.

Declarations

Author contributions

Dr. Kumar Kishlay and Girin Ray contributed equally to manuscript conception, drafting, clinical data interpretation, and critical revision of the final manuscript.

Conflict of interest

The authors declare that they have no conflict of interest.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying clinical details. Identifying information has been omitted to protect patient confidentiality.

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Data availability

All data relevant to this case report are included in the manuscript. Additional details are available from the corresponding author on reasonable request.

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