

Parkinsonism-hyperpyrexia syndrome due to gastroparesis treated with subcutaneous foslevodopa: a case report

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BACKGROUND

Delayed gastric emptying and **gastroparesis**, occurring as part of the broader spectrum of autonomic dysfunction in Parkinson's disease (PD), can lead to erratic levodopa absorption, resulting in unpredictable effectiveness of oral medications and contributing to motor fluctuations and "on-off" phenomena [1].

Severely compromised absorption of dopaminergic drugs can lead to life-threatening conditions like **Parkinsonism-hyperpyrexia syndrome** (PHS), characterized by acute or subacute onset of severe rigidity and akinesia, hyperthermia, altered mental status, autonomic instability and elevated serum creatine phosphokinase levels [2].

The new formulation of **continuous subcutaneous infusion of foslevodopa/foscarbidopa** (LDp/ CDp) has been approved for advanced PD, offering a non-oral route of administration that circumvents the gastrointestinal tract [3].

1. CLINICAL HISTORY

A 71-year-old woman with a 20-year history of PD, H&Y Stage of 4 on therapy with levodopa/carbidopa 875 mg/day and transdermal rotigotine 12 mg/day, presented with severe vomiting over 20 days, complete intolerance of both solids and liquids ingestion, constipation. Past medical history of atrial fibrillation.

1. CLINICAL HISTORY

2. EMERGENCY DEPARTMENT

Alert but somnolent, marked bradykinesia, resting tremors, abdominal distension and reduced peristalsis. Gastric dilatation with air-fluid levels and fecal loading, but no evidence of mechanical obstruction at X-rays and CT. A barium meal RX confirmed complete gastric stasis (*fig. A, B*). At first neurological consultation, gastric atony was likely related to PD dysautonomia. Levodopa was administered via NGT (although an ongoing NGT reflux) and rotigotine was increased to 16 mg/day.

3. INTERNAL MEDICINE DEPARTMENT

Within 48 h, the patient developed signs of PHS:

- Hyperthermia (up to 39 °C)
- A marked decline in consciousness (GCS of 8)
- CPK levels from 30 UI/L to 1170 UI/L
- Tachycardia (from 80 to 120 bpm, sinus rhythm)
- Sudden increase in axial and appendicular rigidity
- No sign of infection (normal laboratory tests, negative blood and urine cultures, no pneumonia at chest CT scan, no endocarditis at echocardiogram). Empirical antibiotics gave no benefit.

2. EMERGENCY DEPARTMENT

3. ADMISSION

5. OUTCOME

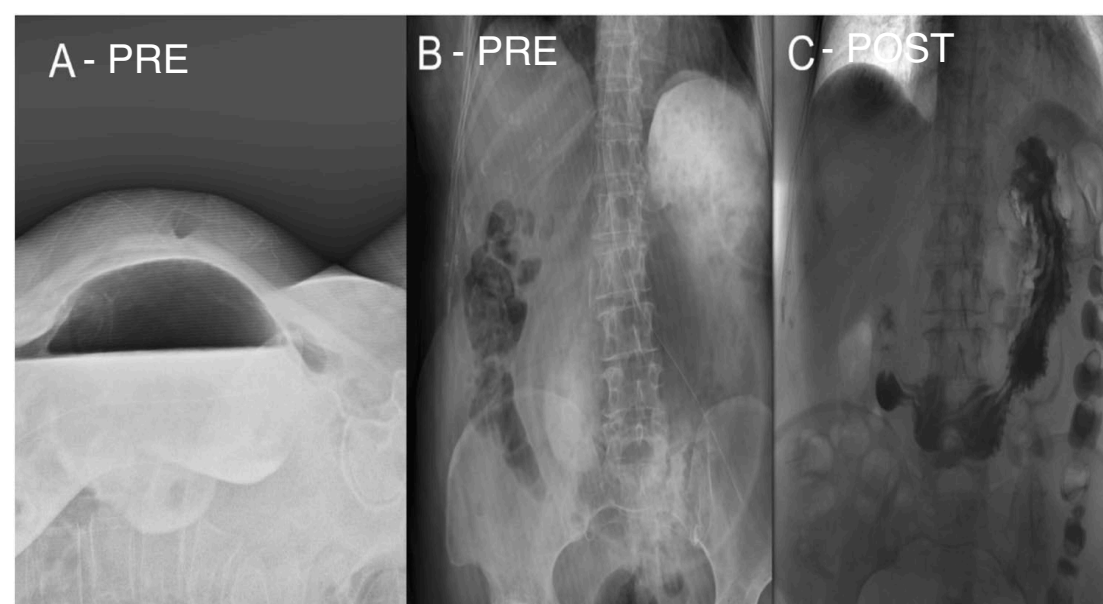
GI FOLLOW-UP: no mechanical obstruction at endoscopy and oral contrast study, confirming the functional nature of the gastric atony (*fig. C*). She resumed oral intake in about 15-20 days.

NEUROLOGICAL FOLLOW-UP: one month after admission, the patient was discharged to a rehabilitation facility, with a final LDp/CDp infusion rate of 0.34 ml/h during daytime and 0.15 ml/h overnight, and reduced rotigotine to 8 mg/die. At three-month follow-up, she had returned to near-baseline independence, able to eat, dress, and walk with minimal assistance. Her motor fluctuations are in pretty good control, present for less than 1 h a day.

5. OUTCOME

4. NEW NEUROLOGICAL CONSULTATION

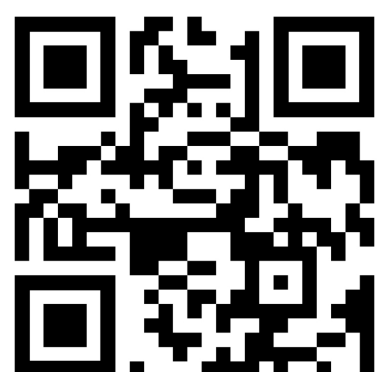
After 72h of resistant fever, given the possible ineffective levodopa absorption and the history of motor fluctuations, subcutaneous LDp/CDp infusion was initiated to bypass the compromised GI route, at infusion rate set to 0.32 ml/h during daytime and reduced to 0.15 ml/h overnight. Within 24 h, the patient's temperature normalized (37 °C), tachycardia resolved (80 bpm), and consciousness improved significantly (GCS of 14). CPK levels returned to baseline. The patient was alert, partially oriented and verbalizing, although sometimes confused, with a gradual improvement in rigidity, tremor, and bradykinesia.



Complete gastric stasis shown by barium contrast radiography (A, B) with subsequent resolution shown by water-soluble iodinated contrast radiography (C).

KEY POINTS AND CONCLUSION

- * Our case provides a novel perspective by identifying a well documented severe gastroparesis as the possible **primary mechanism** of dopaminergic deprivation, leading to **Parkinsonism-hyperpyrexia syndrome**.
- * The unexpectedly rapid improvement using subcutaneous LDp/CDp to bypass the compromised GI system, suggests that it may be a **viable therapeutic option** in clinical scenarios where gastrointestinal absorption is compromised.
- * The reversibility of gastroparesis alongside neurological recovery suggests that restoring systemic dopaminergic transmission could improve enteric motility and **disrupt the vicious cycle of dysautonomia** in PD patients.



SCAN ME

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