When Sepsis Isn't the Whole Story: Invasive GAS and Concurrent Pulmonary Embolism
A Case Report

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CASE TIMELINE

34F P2 presented six days post NVD with suspected invasive GAS sepsis.

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Despite antibiotics, persistent symptoms prompted escalation and further imaging

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CTPA revealed left lower lobe pulmonary embolism.

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She received IV ceftriaxone, clindamycin and therapeutic anticoagulation completing treatment with full recovery

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Discharged on oral cephalexin and therapeutic clexane for three months.

INTERPRETATION AND SYSTEM LEARNING

- Postpartum women are hypercoagulable, immunologically altered, endothelial activated and at peak inflammatory state
- Invasive GAS can cause rapid toxin-mediated deterioration and may mask other pathology due to systemic inflammatory response
- Pulmonary embolism risk is 20–60x higher in first 6 weeks postpartum
- RANZCOG guidance on postpartum VTE stresses low threshold for imaging in symptomatic women and highlighting that infection itself is a VTE risk factor

PRACTICE-CHANGING POINTS

- Postpartum deterioration may represent **more than one pathology**.
- Identification of sepsis does not exclude concurrent venous thromboembolism.
- Persistent tachycardia or hypoxia despite antimicrobial therapy warrants early imaging.
- Infection is itself a risk factor for VTE; reassess thrombosis risk dynamically.
- Early multidisciplinary coordination improves maternal outcomes.

OPPORTUNITIES FOR SAFER PRACTICE

Structured postpartum VTE reassessment in women with severe infection.
Avoid diagnostic closure in complex postpartum presentations.
Clear escalation triggers for tertiary transfer.
Early obstetric medicine involvement in dual pathology cases.