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Neuroleptic malignant syndrome in a traumatic brain injury using quetiapine in ICU: A case report

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Introduction

Neuroleptic malignant syndrome (NMS) is not a commonly occurring complication, but when it does occur, it is a neurological emergency that can lead to death. NMS often presents with nonspecific symptoms such as hyperthermia, dystonia, diaphoresis and tachycardia, making it difficult to diagnose. In particular, because patients with traumatic brain injury (TBI) often experience complications such as posttraumatic agitation and paroxysmal sympathetic hyperactivity (PSH), early diagnosis can be challenging. This case report aims to present a successful diagnosis and management of NMS caused by quetiapine in a patient with PSH.

Case presentation

A 54-year-old male, after a 3-meter fall, arrived at the emergency room with an altered mental state. A CT scan showed a subdural hematoma from the right frontal to the occipital lobe, along with subarachnoid hemorrhage. The patient underwent decompressive craniectomy and evacuation of the hematoma with duroplasty. The neurosurgeon started quetiapine early, known to control posttraumatic agitation and offer neuroprotective effects. During the process of weaning off sedatives, the patient began to exhibit symptoms suggestive of PSH, including tachycardia, hypertension, tachypnea, dystonia and diaphoresis. The patient was evaluated with a clinical feature scale (CFS) of 15 and a diagnosis likelihood tool (DLT) score of 11, indicating severe and probable PSH. Increased doses of baclofen, propranolol, gabapentin, codeine, and the fentanyl patch were implemented to manage PSH symptoms, resulting in a decrease in the frequency of episodes. Due to pneumonia deterioration, quetiapine and PSH medication were discontinued, and sedatives were reintroduced. Thereafter previous medications resumed at prior doses. Upon reintroduction, PSH episodes surged to 26 times in 10 days (Figure 1). Despite increased PSH medication dosage and intermittent administration of morphine sulfate, controlling symptoms remained challenging compared to prior efforts. The neurosurgeon conducted a differential evaluation of infection including CSF tapping. Due to suspicion of NMS after high-dose quetiapine reintroduction, it was stopped. Dantrolene via NG tube was initiated, along with propofol and dexmedetomidine infusion, increasing hydration. Following these interventions, symptoms improved rapidly (Figure 2). In this case, no complications occurred when neuroleptics were initially used, but NMS appeared upon reintroducing high doses of neuroleptics following weaning off sedatives. Specific symptoms differentiating NMS from PSH were absent. However, discontinuation of neuroleptics and NMS specific treatment yielded remarkable improvement, reinforcing NMS suspicion.

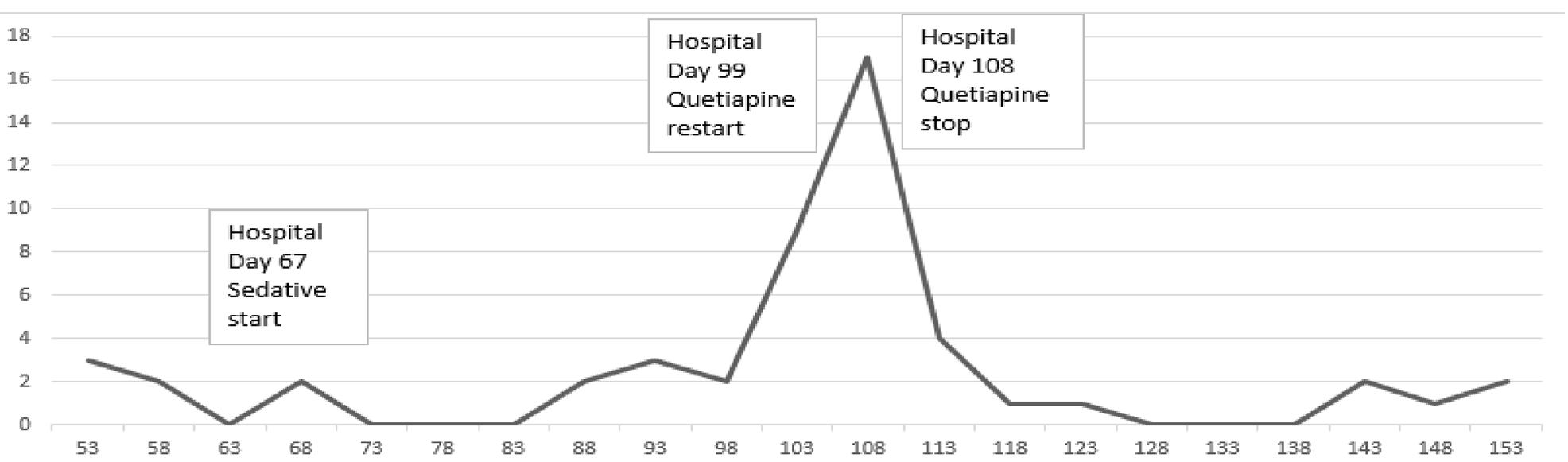

Figure 1. The frequency of suspected neuroleptic malignant syndrome symptoms occurrence measured every five hospital days in ICU

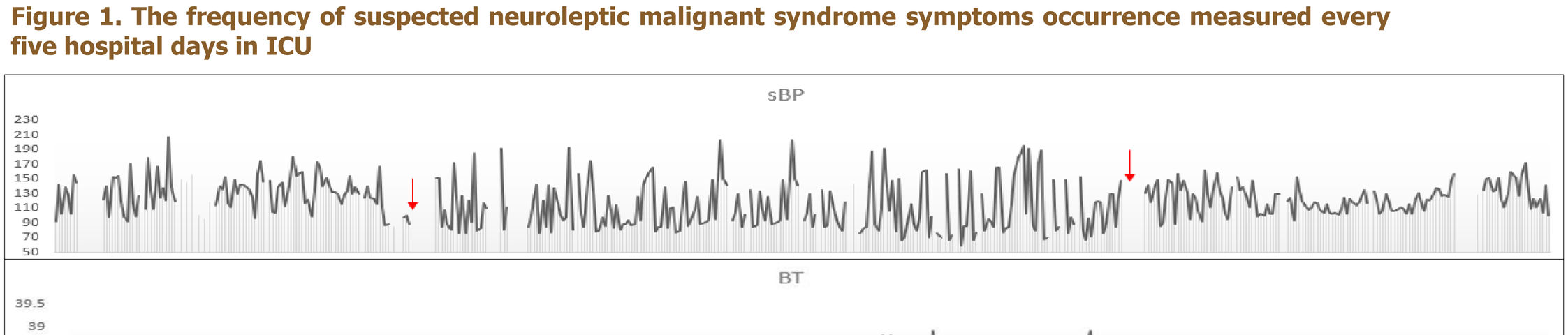

Figure 2. Vital signs monitored from hospital day 94 to 114 of intensive care unit admission. The red arrows indicate the points at which quetiapine was restarted and subsequently discontinued. sBP, systolic blood pressure; BT, body temperature; RR, respiratory rate; HR, heart rate

Conclusion

When using neuroleptics, clinicians should cautiously adjust doses, starting low and closely monitoring for adverse effects. Any change in the patient's stable condition should raise suspicion for NMS, prompting early diagnosis efforts.