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GABAPENTIN INDUCED ACUTE ENCEPHALOPATHY IN A 68 YEAR OLD MALE TREATED FOR POST – HERPETIC NEURALGIA

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Abstract:

Gabapentin is widely used in the management of neuropathic pain and is generally considered safe[1]. However, in rare cases, it may lead to serious neurological adverse effects such as acute encephalopathy, particularly in elderly patients[2]. We present the case of a 68-year-old male who developed sudden confusion, disorientation, and excessive drowsiness shortly after starting gabapentin for post-herpetic neuralgia[3]. Clinical evaluation and laboratory investigations, including renal and liver function tests, were within normal limits[4]. Neuroimaging showed no abnormalities, while electroencephalography revealed diffuse slowing suggestive of encephalopathy[5]. Gabapentin was discontinued, and the patient showed marked improvement within 48 hours, with complete recovery by 72 hours[6]. This case highlights the importance of early recognition of drug-induced neurological adverse effects and the need for cautious use of gabapentin in elderly patients[7].

Keywords : *Gabapentin , Acute encephalopathy , Adverse drug reaction , Neurotoxicity .*

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INTRODUCTION:

Gabapentin is an antiepileptic drug that is commonly used for the treatment of neuropathic pain, post-herpetic neuralgia, and as an add-on therapy in seizure disorders[1]. It is structurally related to gamma-aminobutyric acid (GABA), although it does not directly act on GABA receptors[8]. Instead, it works by binding to the α2δ subunit of voltage-gated calcium channels, thereby reducing the release of excitatory neurotransmitters in the central nervous system[9].

Gabapentin is generally well tolerated, and most of its commonly reported side effects, such as dizziness, fatigue, and mild drowsiness, are usually transient and manageable[4]. Serious neurological adverse effects are rare and often under-recognized[2]. Among these, acute encephalopathy is an uncommon but clinically significant condition that can present with altered mental status, confusion, and excessive sedation[10].

The risk of such adverse effects is higher in elderly patients due to age-related physiological changes, including increased sensitivity to drugs and alterations in the blood–brain barrier[11]. In addition, gabapentin is primarily eliminated through the kidneys, and its accumulation in patients with renal impairment can increase the risk of toxicity[12]. However, cases have also been reported in patients with normal renal function, suggesting that other factors may be involved[3].

Given the widespread use of gabapentin, it is important for clinicians to be aware of such rare but reversible adverse effects[13]. Early identification and prompt discontinuation of the drug can lead to complete recovery and prevent unnecessary investigations[6]. This case report aims to highlight a rare presentation of gabapentin-induced acute encephalopathy in an elderly patient[7].

CASE REPORT:

A 68 year male presented to the emergency department with complains of sudden-onset confusion,disorientation and excessive sleepiness for the past two days. Family members reported a sudden change in behavior, with reduced responsiveness and difficulty recognizing surroundings.There was no associated history of fever, seizures, headache, vomiting, head trauma, alcohol intake or recent infection.

The patient had a recent history of post-herpetic neuralgia, for which he was started on gabapentin 300 mg twice daily five days prior to admission. He was not receiving any other centrally acting medications. There was no past history of epilepsy, renal disease, hepatic illness, diabetes mellitus, or cerebrovascular disease.

On clinical examination, the patient was drowsy but arousable and disoriented to time and place. His vital signs were stable, with normal blood pressure, pulse rate, respiratory rate, and oxygen saturation. Neurological examination revealed no focal deficits. Cranial nerve examination, motor power, sensory system, and deep tendon reflexes were within normal limits. There were no signs of meningeal irritation.

Laboratory investigations including complete blood count, serum electrolytes, renal function tests, liver function tests, and random blood glucose were all within normal limits. Computed tomography (CT) scan of the brain showed no acute intracranial abnormalities. Electroencephalography (EEG) demonstrated diffuse, non-specific slowing suggestive of a metabolic or drug-induced encephalopathy.

Based on the temporal association between gabapentin initiation and symptom onset, along with exclusion of other possible causes, a diagnosis of gabapentin-induced acute encephalopathy was considered. Gabapentin was immediately discontinued, and the patient was managed conservatively with supportive care and close neurological monitoring.

The patient showed significant clinical improvement within 48 hours of drug withdrawal, with complete resolution of symptoms by 72 hours. At discharge, the patient was hemodynamically stable and fully oriented. He was prescribed paracetamol as needed for pain, vitamin B-complex supplementation, and topical analgesic therapy for neuralgia. Gabapentin was permanently avoided, and the patient was counseled regarding drug-related adverse effects.
On follow-up, the patient remained asymptomatic with no recurrence of neurological symptoms.

Table 1.Daily vitals monitoring chart

Day	Temp	Pulse (bpm)	BP (mmHg)	RR (/min)	SpO_2 (%)	Remarks
Day 1	98.8	88	130/82	18	97	Confusion, somnolence on admission; gabapentin discontinued
Day 2	98.4	84	120/76	18	98	Improved arousal; oriented to person only
Day 3	98.2	80	124/78	17	98	Fully oriented; minimal drowsiness
Day 4	98.2	78	122/80	17	99	Ambulatory; symptom-free
Day 5	98.0	76	120/78	16	99	Discharged in stable condition

Table 2. Laboratory investigation

Parameter	Patient value	Normal range	interpretation
Haemoglobin	13.8g/dL	13.0-17.0g/dL	Normal
Total leukocyte count	7,200cells/mm^3	4,000-11,000	Normal
Platelet count	2.1 lakh/mm^3	1.5-4.0lakh/mm^3	Normal
Serum creatinine	0.9 mg/dL	0.6-1.2mg/dL	Normal
Blood urea Nitrogen	18mg/dL	7-25mg/dL	Normal
eGFR	78 mL/min/1.73m^2	>60	Normal
Serum Sodium	138 mEq/L	136-145mEq/L	Normal
Serum Potassium	4.0 mEq/L	3.5-5.0 mEq/L	Normal
Total Bilirubin	0.8mg/dL	0.2-1.2mg/dL	Normal
AST / ALT	28 / 24 U/L	<40 U/L	Normal
Random blood Glucose	96mg/dL	<200mg/dL	Normal
CT Brain	No acute abnormality	--	Normal
EEG	Diffuse non specific slowing	Normal cortical activity	Encephalopathy

Table 3. Drug Chart

Drug / Dose	Drug Class	Indication	Route	Frequency
Gabapentin 300mg	Antiepileptic/ analgesic	Post-herpetic neuralgia	Oral	BD Stopped
Paracetamol 500mg	Non-opioid analgesic	Pain management	Oral	SOS
Methylcobalamin 500mcg	Vitamin B12 analogue	Neuropathy support	Oral	BD
Vitamin B-complex	Nutritional supplement	Neuro protection	Oral	OD
Topical Lidocaine 5%	Local anaesthetic	Post-herpetic neuralgia	Topical	BD

DISCUSSION :

Gabapentin is widely prescribed for neuropathic pain conditions such as post-herpetic neuralgia and is generally considered a safe and well-tolerated drug[1]. It is often preferred in elderly patients because of its minimal drug interactions and lack of hepatic metabolism[13]. Despite this favorable profile, the present case highlights that gabapentin can, in rare situations, lead to significant central nervous system adverse effects, including acute encephalopathy[2].

In this case, a 68-year-old patient developed sudden confusion, disorientation, and excessive drowsiness within a few days of starting gabapentin therapy[10]. The close temporal relationship between drug initiation and symptom onset, along with the absence of other identifiable causes, strongly supports a drug-induced etiology[14]. The rapid and complete recovery following discontinuation of gabapentin further strengthens this association[6].

When compared with previously reported cases, a similar pattern is consistently observed[3]. Most published reports describe elderly patients presenting with acute onset of altered mental status shortly after starting gabapentin, with symptom resolution typically occurring within 24–72 hours after drug withdrawal[4]. This reversible course is a key feature that distinguishes drug-induced

encephalopathy from other neurological conditions[5]. Our case closely follows this pattern, reinforcing the findings reported in earlier studies.

One of the most commonly cited risk factors for gabapentin-induced neurotoxicity is renal impairment, as the drug is primarily excreted unchanged through the kidneys[12]. Accumulation of the drug in patients with reduced renal function can lead to toxic levels and subsequent neurological symptoms. However, an important aspect of this case is that the patient had normal renal function, which has also been described in a smaller number of reports[12]. This indicates that while renal dysfunction increases risk, it is not a necessary condition for the development of encephalopathy.

Age appears to be a critical independent risk factor. Most cases reported in the literature involve patients above 60–65 years of age[7]. Elderly individuals are more susceptible due to multiple physiological changes associated with aging[11]. There is a gradual decline in neuronal reserve, making the brain less capable of compensating for pharmacological effects. In addition, pharmacodynamic sensitivity to centrally acting drugs is increased, meaning that standard therapeutic doses may produce exaggerated effects[11].

Another important contributing factor is the alteration of the blood–brain barrier (BBB) with advancing age. The BBB normally regulates the entry of substances into the central nervous system, but age-related structural and functional changes can increase its permeability[15]. As a result, drugs like gabapentin may enter the brain in higher concentrations, leading to enhanced central nervous system depression[16]. This mechanism may explain why elderly patients can develop encephalopathy even when standard doses are used and laboratory parameters remain normal.

In comparison to other reported cases, the absence of comorbiditic conditions such as renal disease, hepatic dysfunction, or polypharmacy makes the association more evident[17]. Many cases in the literature involve multiple contributing factors, which can complicate the identification of the exact cause[17]. In contrast, our patient was not taking other centrally acting medications and had no significant underlying illnesses, making gabapentin the most likely causative agent.

The diagnostic approach in this case is also consistent with previously reported cases. Other potential causes of altered mental status, including metabolic disturbances, infections, and structural brain abnormalities, were carefully excluded through laboratory investigations and imaging studies[18]. The EEG findings of diffuse, non-specific slowing are typical of metabolic or drug-induced encephalopathy and have been frequently described in similar cases[19]. This further supports the diagnosis.

From a mechanistic perspective, gabapentin exerts its effect by binding to the α2δ subunit of voltage-gated calcium channels, leading to decreased release of excitatory neurotransmitters[8]. While this action is beneficial for controlling neuropathic pain, excessive inhibition of neuronal activity may result in global cerebral dysfunction. In elderly patients, where drug sensitivity is increased and BBB permeability is altered, this effect can become more pronounced, leading to encephalopathy.

Overall, when compared with similar cases in the literature, this case as several important observations:

- Elderly patients are at higher risk due to age-related CNS changes and BBB alterations[20]
- Encephalopathy can occur even in the absence of renal impairment[20]
- Symptoms typically develop soon after drug initiation [20]
- Rapid recovery follows drug withdrawal, confirming reversibility[20]

Overall, this case reinforces observations from existing literature that gabapentin-induced encephalopathy is more likely to occur in elderly patients, can develop even at therapeutic doses, and is usually reversible with prompt drug discontinuation[20]. Careful monitoring is essential when initiating gabapentin in patients above 60 years of age[21]. Early recognition of symptoms and timely withdrawal of the drug can prevent complications and ensure complete recovery[21].

ROLE OF CLINICAL PHARMACIST

In this case, the clinical pharmacist played an important role in identifying the possible cause of the patient's symptoms. Since the patient developed confusion and excessive drowsiness soon after starting gabapentin, careful review of the medication history helped in suspecting the drug as a likely reason. By correlating the timing of drug initiation with the onset of symptoms, the pharmacist contributed to early recognition of a possible adverse drug reaction.

The clinical pharmacist also assisted in ruling out other medication-related causes, as the patient was not receiving any other centrally acting drugs. This made it easier to focus on gabapentin as the suspected agent. Based on this assessment, the pharmacist recommended immediate

discontinuation of the drug, which played a key role in the patient's recovery.

Another important responsibility was monitoring the patient after stopping the medication. The pharmacist closely observed improvements in mental status and ensured that no further complications occurred. This helped confirm that the reaction was drug-induced and reversible.

The clinical pharmacist also suggested safer alternatives for pain management, such as paracetamol and topical therapies, to avoid recurrence of similar adverse effects. In addition, the patient and family members were counseled about the possible side effects of gabapentin and advised to report any unusual symptoms early in the future.

Overall, this case highlights how the involvement of a clinical pharmacist can improve patient safety by early detection of adverse drug reactions, supporting appropriate treatment decisions, and preventing unnecessary investigations or prolonged hospital stay .

CONCLUSION :

This case highlights that gabapentin, although commonly considered safe and widely used for neuropathic pain, can rarely cause acute encephalopathy, particularly in elderly patients. The sudden onset of confusion, disorientation, and excessive drowsiness shortly after starting the drug, along with the absence of other underlying causes, strongly indicated a drug-induced reaction.

Timely recognition played a key role in the patient's recovery. Immediate withdrawal of gabapentin led to rapid and complete improvement within a few days, confirming the reversible nature of this adverse effect. This emphasizes the importance of early identification and prompt action in preventing complications.

The clinical pharmacist had a significant role in this case by carefully reviewing the medication history, identifying the suspected adverse drug reaction, and supporting the decision to discontinue the drug. Continuous monitoring of the patient's condition and documentation of recovery further helped in confirming the diagnosis. In addition, the clinical pharmacist contributed to patient safety by recommending safer alternative options such as paracetamol and topical therapies for pain management.

From a pharmacovigilance perspective, reporting this case to the Pharmacovigilance Programme of India (PvPI) is important, as it adds to the existing safety data of gabapentin and helps in better understanding rare adverse effects. Proper documentation and reporting can support safer prescribing practices in the future.

Overall, this case underlines the need for cautious use of gabapentin, especially in patients above 60 years of age. Close monitoring after initiation, early detection of unusual neurological symptoms, prompt drug withdrawal, and appropriate alternative therapy are essential steps in ensuring patient safety and improving clinical outcomes .

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