Paclitaxel-Induced Transient Encephalopathy in a Patient with Metastatic Breast Carcinoma: A Rare Adverse Event

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Introduction

Paclitaxel, a microtubule-stabilizing taxane, is widely used for solid tumors. Its main side effects are neuropathy and myelosuppression. CNS toxicity is uncommon due to limited penetration across the blood-brain barrier (BBB). Proposed mechanisms include BBB disruption by the Cremophor EL solvent, cytokine-mediated inflammation, and direct neurotoxicity. We present a rare case of paclitaxel-induced transient encephalopathy that resolved after drug withdrawal.

Case Presentation

A 50-year-old female with Stage IIA breast carcinoma declined neoadjuvant chemotherqpy ,underwent right mastectomy, received chest wall radiotherapy, and was maintained on letrozole. Six months later, she developed disease recurrence with mediastinal lymphadenopathy, lung nodules, and pleural effusion. Weekly paclitaxel (80 mg/m^2) was started. After the eighth dose, she presented two days later with confusion, disorganized speech, and mild headache. No fever, seizures, or focal neurological deficits were present. Examination revealed stable vitals and no signs of meningitis or focal deficit.
Investigations:

- Serum electrolytes, renal and liver function: Normal.
- CSF: Normal protein and glucose, no cells, PCR Meningitis pannel negative.
- MRI brain: Normal, no metastasis or ischemia.
- Other causes (infection, metabolic or hepatic encephalopathy) were excluded.

Symptoms resolved completely within 4 days without any specific treatment. Paclitaxel was discontinued.

Discussion

CNS toxicity due to paclitaxel is rare but has been reported in literature. Symptoms typically occur within hours or days after infusion and improve once the drug is stopped. Proposed causes include:

- Blood-brain barrier disruption by Cremophor EL.
- Cytokine-induced neuroinflammation (IL-1β, TNF-α, IL-6).
- Direct neurotoxicity in cases with altered BBB.

This case supports previous reports showing reversible encephalopathy without brain lesions. The strong temporal relation with paclitaxel infusion and spontaneous recovery suggest a direct link.

Management is supportive, focusing on observation and discontinuation of the suspected agent. Re-challenge with paclitaxel is generally avoided, though substitution with nab-paclitaxel (albumin-bound formulation, devoid of Cremophor EL) may be considered in selected cases.

Conclusion

Paclitaxel-induced transient encephalopathy is rare but reversible. It should be suspected in patients who develop acute confusion after paclitaxel infusion even without CNS disease. Early recognition prevents unnecessary tests and enables timely management.

References

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