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Irreversible Brainstem Atrophy Following Fibrin-Sealant–Induced Delayed Eosinophilic Meningoencephalitis After Microvascular Decompression: A Case Report

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Objective

To describe a rare and severe case of delayed eosinophilic meningoencephalitis and irreversible brainstem atrophy following fibrin-sealant application during microvascular decompression (MVD), and to highlight the underlying immunopathologic mechanism and clinical implications.

Case Description

A male patient with a 10-year history of trigeminal neuralgia underwent MVD with insertion of a Teflon pledget secured using Beriplast® fibrin sealant. Immediately after surgery (POD 0), he developed profound quadriplegia (MRC grade 1). MRI revealed ventral medullary and pontine T2/FLAIR hyperintensity without diffusion restriction, suggestive of acute vasogenic edema. Despite complete removal of the surgical materials on POD 1, neurological deficits persisted.
Subsequent imaging demonstrated:
POD 8: New diffusion-restricted lesions in anterior medulla and lower pons (cytotoxic injury)
POD 36–62: Progressive eosinophilia peaking at 21.0% with diffuse rash
POD 65: Bilateral anterior medullary inflammatory changes
POD 268: Medullary thinning and expansion of pre-medullary CSF space consistent with brainstem atrophy
Extensive infectious, autoimmune, vascular, and drug-related evaluations were negative.
Electrodiagnostic studies showed markedly prolonged SEP/MEP latencies, indicating irreversible long-tract damage

Results

This case demonstrated a multi-phasic immune-mediated injury process. 1. Early phase (POD 0): Acute innate immune–mediated vasogenic edema without infarction. 2. Subacute phase (POD 8): Progression to cytotoxic edema and secondary ischemic injury. 3. Delayed phase (POD 36–62): Marked eosinophilia and systemic rash consistent with Type IVb delayed hypersensitivity reaction. 4. Chronic phase (POD 268): Irreversible medullary atrophy and persistent neurological deficit. Despite intensive rehabilitation, no meaningful functional recovery occurred.

Conclusion

This case illustrates a rare but devastating fibrin-sealant–associated delayed hypersensitivity reaction leading to: Eosinophilic meningoencephalitis, Progressive cytotoxic brainstem injury, Irreversible medullary atrophy. Early recognition of postoperative inflammatory changes and avoidance of bovine-derived or aprotinin-containing fibrin sealants are critical to prevent catastrophic neurological outcomes

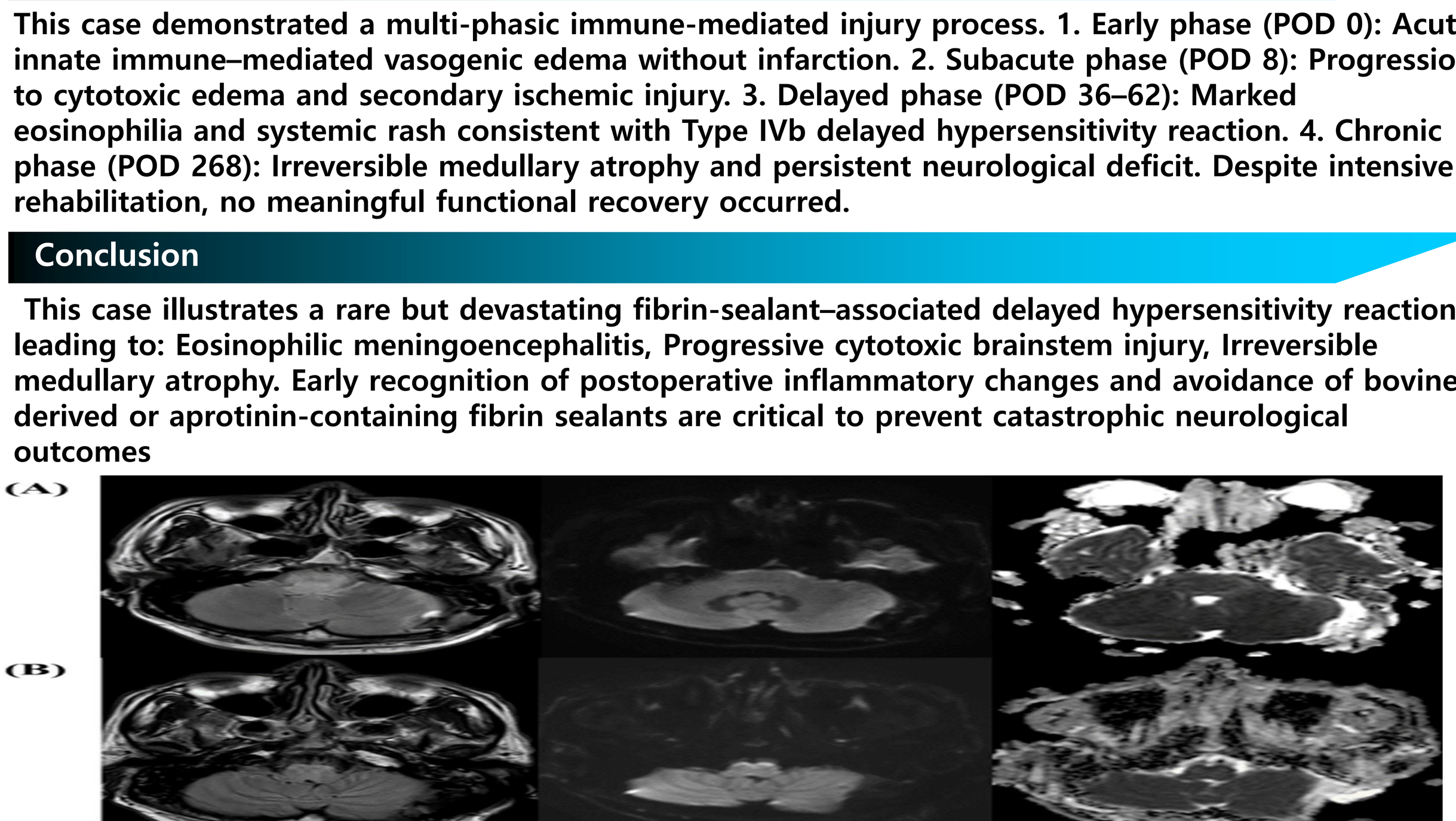

Figure 1. Evolution of brainstem injury following microvascular decompression. Axial panels (A–D): T2-FLAIR, DWI, ADC sequence. Panel (E): midline T1-weighted sagittal images. (A) POD 0: Ventral medullary/pontine T2-FLAIR hyperintensity without diffusion restriction (vasogenic edema). (B) POD 8: New diffusion-restricted lesions in anterior medulla and lower pons with ADC hypointensity (cytotoxic injury). (C) POD 65: Bilateral anterior medullary T2-FLAIR hyperintensity (subacute inflammation). (D) POD 268: Improvement of FLAIR hyperintense changes. (E) Preoperative vs POD 268: Pronounced medullary atrophy with thinning and expansion of pre-medullary CSF space.