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Case Report

Delayed Inflammatory Reaction after Mechanical Thrombectomy: A Case Report and Literature Review

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Abstract

Mechanical thrombectomy is the standard of care for patients with ischemic stroke due to large vessel occlusion with salvageable penumbra and has resulted in improved outcomes. Mechanical thrombectomy is performed under fluoroscopy and involves the use of microcatheters introduced into the cerebral circulation. A rare side effect of this procedure is granulomatous inflammation due to hydrophilic polymer particles which are part of the composition of the microcatheters and can slough off during their usage resulting in a delayed inflammatory reaction due to the presence of foreign material. We report a case of a 70-year-old woman who presented with a seizure several months after mechanical thrombectomy whose imaging demonstrated vasogenic edema in the territory of her infarct, suggestive of a granulomatous reaction. She was successfully treated with intravenous steroids followed by an oral steroid taper which resulted in clinical improvement and resolution of the lesion. The recognition of this rare condition is important as it can lead to delayed neurological complications, which can be treated with steroids.

Keywords: *Mechanical thrombectomy, Cerebral granulomatous inflammation, Hydrophilic polymer embolism, Ischemic stroke*

Introduction

A standard of care for ischemic stroke caused by large vessel occlusion is mechanical thrombectomy. Cerebral granulomatous inflammation from mechanical thrombectomy is a rare allergic reaction from the hydrophilic polymers coated on endovascular devices. These reactions occur after endovascular devices are used to remove a clot/embolus. There are only a few case reports explaining this phenomenon that are reported in the literature. These allergic reactions can occur days to months after a mechanical thrombectomy and can present with nonspecific findings or renewed neurological deficits in the affected body area post ischemic stroke. Even though the occurrence of an allergic reaction such as cerebral granulomatous inflammation is rare, more research and data should be gathered on this so proper treatment protocols can be established, ensuring efficient patient treatment and care. We add to this body of literature by explaining a case encountered at our institution and a review of the topic. [1-4]

Case Presentation

A 70-year-old female with a history of congestive heart failure was admitted to our hospital with left sided weakness found to have an occlusion of the right internal carotid artery extending into the middle cerebral artery (MCA) for which she underwent mechanical thrombectomy. MRI demonstrated restricted diffusion and FLAIR hyperintensity suggestive of a right middle cerebral artery distribution acute ischemic stroke (Figure 1A). Her transthoracic echocardiogram at that time showed severely reduced ejection fraction (35%) and mural thrombus. She started anticoagulation with warfarin. She improved post-procedure, though on discharge she had persistent left hemiparesis and was discharged to a subacute rehabilitation facility.

Three months later, while still residing at the subacute rehab facility, she was noted to have twitching of her eyes, right gaze preference, dysarthria, and confusion consistent with a focal-onset seizure that resolved by the time she arrived in the emergency room. She had a second seizure in the emergency department which lasted approximately 30 seconds upon which she was given 1000 milligrams of intravenous levetiracetam. After stabilization, her computed tomography (CT) head showed vasogenic edema in the right cerebral hemisphere.

She was readmitted for further workup and MRI of her brain revealed vasogenic edema in the right cerebral hemisphere (Figure 1B). Additionally, there were numerous associated small enhancing parenchymal nodules within the region of the vasogenic edema. She had no infectious symptoms and the lesions appeared atypical for a neoplastic process. Imaging findings were most consistent with cerebral granulomatous inflammation from mechanical thrombectomy.

Subsequently, 1000 mg of intravenous methylprednisolone was administered daily for three days followed by an oral steroid taper down to 20 mg of prednisone daily. She was continued on antiepileptic medication with levetiracetam 500 mg twice daily, as well as warfarin dosed to an INR goal of 2-3.

At 3-month follow-up, she had had no further seizures and had a repeat MRI brain which demonstrated resolution of the vasogenic edema with persistent encephalomalacia relating to her prior stroke (Figure 1C) and was gradually tapered off the prednisone with no further clinical sequelae.

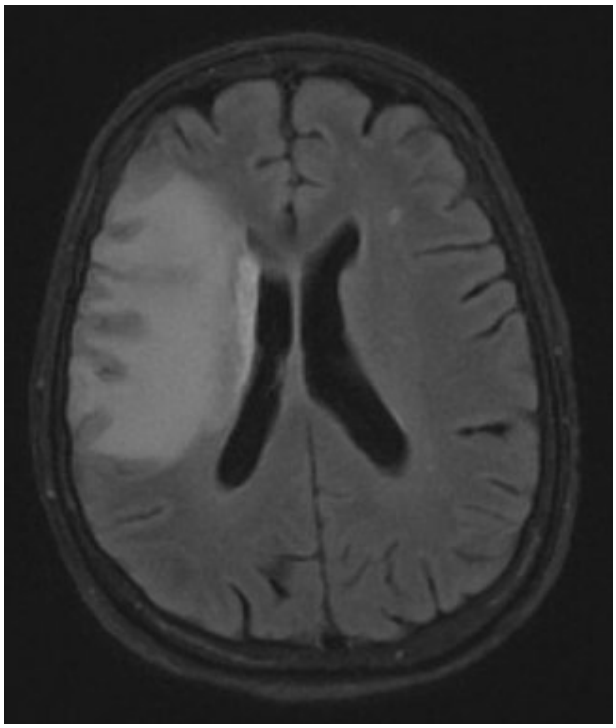

Figure 1A. MRI brain during hospitalization for seizure demonstrating interval progression of vasogenic edema and new enhancing nodules suggestive of granulomatous inflammation.

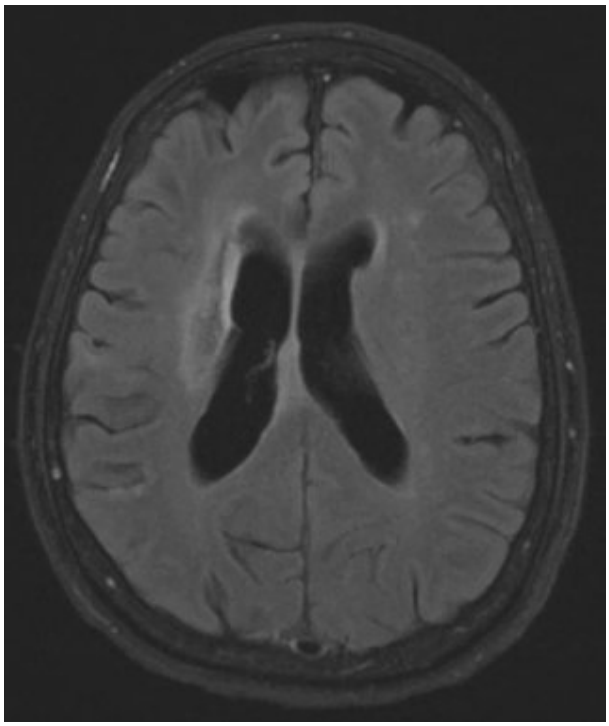

Figure 1B. Follow up MRI brain after treatment with steroids demonstrating resolution of the vasogenic edema and inflammation, with persistent encephalomalacia from infarcted tissue.

Table 1. Review of previously published cases in the literature.

Patient Age & Gender	Patient Presentation	Imaging Findings	Treatment
69-year-old Female	Presented multiple times with episodes of transient confusion, disorientation, headache, and nausea	MRI findings after thrombectomy revealing punctate T2 FLAIR hyperintensities in the right parietal lobe with associated enhancement and vasogenic edema.	Management consisted of high dose dexamethasone and a taper, followed by serial imaging for continued monitoring, demonstrating reduction in edema and nodule size, and improvement of her symptoms. However, the inflammation recurred each time steroids were tapered. Lesions were presumed to be granulomatous in foreign bodies, based on appearance. Biopsy results of the right parietal dura and lesion demonstrated mild leptomeningeal mononuclear cell inflammation; however, this was influenced by steroid use
59-year-old Female	Presented with a complete occlusion of her right proximal middle cerebral artery (MCA) and imaging showed a large area of penumbra. Cerebral angiogram and mechanical thrombectomy were successfully performed with reversal of clinical symptoms. Eight months following her stroke, she developed progressive recurrence of left-sided neurological deficits with new right gaze preference	Repeat MRI imaging showed a lesion with progressive confluent T2 hyperintensity with greater than 30 admixed punctate areas of enhancement, involving cortex and white matter of the right parietal, temporal, and occipital lobes on T1. Diagnostic brain biopsy of the lesion was identified as necrotizing granulomatous inflammation secondary to hydrophilic polymer.	She was treated with pulse dose methylprednisolone for five days followed by a slow taper of 60 mg prednisone daily dropping by 10 mg every three days to 20 mg daily. At the time of discharge, the patient had near complete recovery of her left lower extremity strength, partial recovery of left upper extremity strength, with recovery of headaches near baseline. Follow-up imaging six weeks after steroid therapy showed significant improvement in her inflammatory response with post-inflammatory evacuation.
73-year-old Female	Presented with complete occlusion of the right middle cerebral artery. Cerebral angiography and MT were successfully performed with improvement in clinical symptoms. Left hemiparesis and a disturbance in attention appeared after discharge and progressed slowly. She was re-admitted to our hospital 120 days after cerebral infarction	Foreign body granulomas diagnosed on biopsy	Successfully treated with steroid therapy
78-year-old Male	Presented with occlusion of the left cervical internal carotid artery and the left middle cerebral artery. Cerebral angiography, percutaneous transluminal angioplasty, and MT were successfully performed. On the 34th day, he experienced progressive consciousness disorder	On the 34 th day, his consciousness worsened; the previously detected high-intensity area on FLAIR progressively enlarged and more than 50 contrast-enhanced lesions were in the same area. The CSF showed a cell count of <1/μL, a protein level of 109 mg/dL, and a glucose level of 103 mg/dL.	Administered methylprednisolone at a dose of 1000 mg for three consecutive days per week for 6 weeks. After 1 week of steroid treatment, his consciousness disorder improved. On the 61 st day, the multiple enhanced lesions on MRI had disappeared and edema on FLAIR imaging had reduced.

Discussion

Cerebral granulomatous inflammation from mechanical thrombectomy is incredibly rare, which means patients with this condition are less likely to be correctly assessed and treated. Since this is still a very rare event in medicine, it is important these cases are formally documented so treatment protocols can be standardized and performed. Following endovascular procedures such as embolectomy or thrombectomy, more physicians should be aware of this reaction in patients. With early recognition and medical intervention, clinical providers can improve patient outcomes and decrease possible complications.

As noted in previous cases, weeks, or months after an embolectomy or thrombectomy, patients should be monitored for physical symptoms such as transient confusion, disorientation, paresis or hemiparesis, headache or nausea. If MRI imaging is obtained and physicians are aware of the previous endovascular procedure within the past few months, MRI findings such as punctate T2 flair hyper densities, penumbras, contrast-enhancing lesions in the same area where the endovascular procedure was performed can suggest the possibility of cerebral granulomatous inflammation. A cerebral granuloma is a type IV hypersensitivity reaction, for which corticosteroids are considered first-line treatment. As seen in previous cases, management with corticosteroid therapy such as methylprednisolone or dexamethasone tapers have shown remarkable improvement in patients' symptoms. Patients are followed with serial imaging for continued monitoring around 4 to 6 weeks after stopping anti-inflammatory treatment. Many patients had total or near-total resolve of their symptoms associated with granulomatous inflammation.

However, the optimal dosage, duration, and tapering regimen of corticosteroid therapy for cerebral granulomatous reactions is undefined and unstandardized. As additional clinical cases are documented, systematic research aimed at establishing evidence-based corticosteroid protocols should be prioritized. Standardization would provide clear guidance on corticosteroid initiation, dose adjustments, and tapering schedules across the patient’s multiple weeks of treatment, reducing the risks of both under-treatment and corticosteroid-related adverse effects. Furthermore, defining appropriate follow-up care — whether under the supervision of a neurologist or in collaboration with primary care physicians—will be critical to ensuring long-term monitoring and comprehensive patient management.

To note, granulomatous reactions following mechanical thrombectomy (MT) can also be obscured by the residual sequelae of the initial cerebral infarction, which can also lead to underdiagnosis. Further research into the pathophysiology and clinical presentation of cerebral granulomatous reactions are essential to increase recognition of this disorder among health care providers. Increased knowledge and awareness will enable physicians to differentiate post-stroke sequelae more effectively from symptoms of cerebral granulomatous inflammation. Diagnostic distinction is vital to decreasing the likelihood of underdiagnosis, implementing the appropriate therapeutic treatment, and ultimately improving long term patient outcomes. Finally, developers of devices used in a mechanical thrombectomy, or other endovascular procedures should become aware of the risk of a patient developing a granulomatous reaction from their product so alterations can be made to prevent these reactions in the future. Recognition of this complication could inform future device design and material selection, thereby reducing the risk of a granulomatous reaction. Also, awareness of this issue highlights the critical need for collaboration between physicians, researchers, and device developers so adverse medical events or immune reactions to medical devices can be prevented and improve patient outcomes. [1-11]

Conclusion

We reported on a case of a cerebral granulomatous inflammation reaction which occurred three months after our patient underwent a mechanical thrombectomy following an occlusion in the right internal carotid artery that extended into the middle cerebral artery (MCA). These adverse immune reactions are rare and as such more data needs to be collected so efficient treatment guidelines can be established. Ultimately, optimal dosing, duration, and tapering regimen of corticosteroid therapy for cerebral granulomatous reactions should be researched and standardized to improve patient outcomes and decrease possible complications.

Conflict of Interest

The authors declare that they have no conflict of interest.

References

1. Mehta, R. I., Rai, A. T., Vos, J. A., Solis, O. E., & Mehta, R. I. (2019). Intrathrombus polymer coating deposition: A pilot study of 91 patients undergoing endovascular therapy for acute large vessel stroke. Part I: Histologic frequency. Journal of NeuroInterventional Surgery, 11(12), 1191–1196. https://doi.org/10.1136/neurintsurg-2018-014684

2. Meiers, C., Abebe, Y., Alberto, N., Riedinger, J., Breker, D., Manchak, M., Drofa, A., & Teigen, C. (2017). Cerebral granulomatous inflammation secondary to hydrophilic polymer embolization following thrombectomy. American Journal of Case Reports, 18, 507–511. https://doi.org/10.12659/AJCR.902598

3. Qureshi, M., Quinlan, M., George, R., Heller, R., Gerber, A., & Radmard, S. (2024, April 9). Cerebral granulomatous inflammation as a complication of mechanical thrombectomy (P8-5.003). Neurology, 102(7_Supplement_1). https://doi.org/10.1212/WNL.0000000000205392

4. Shun Ishikawa, Yosuke Kudo, Shigeta Miyake, Taisuke Akimoto, Sawako Chiba, Wakiko Saruta, Takahiro Mochizuki, Satoru Shimizu, Yu Amano, Ryoo Yamamoto, Kazumitsu Amari, Shigeru Koyano, Ken Johkura, Tetsuya Yamamoto, Yasunobu Nakai. Cerebral foreign body granulomas after mechanical thrombectomy: Two case reports and a review of the literature. Journal of Stroke and Cerebrovascular Diseases, Volume 32, Issue 3, 2023, 106990, ISSN 1052-3057. https://doi.org/10.1016/j.jstrokecerebrovasdis.2023.106990

5. Barnwell, S. L., D'Agostino, A. N., Shapiro, S. L., Nesbit, G. M., & Kellogg, J. X. (1997). Foreign bodies in small arteries after use of an infusion microcatheter. AJNR American Journal of Neuroradiology, 18(10), 1886–1889. https://www.ajnr.org/content/18/10/1886

6. Chopra, A. M., Mehta, M., Bismuth, J., Shapiro, M., Fishbein, M. C., Bridges, A. G., & Vinters, H. V. (2017). Polymer coating embolism from intravascular medical devices—a clinical literature review. Cardiovascular Pathology, 30, 45–54. https://doi.org/10.1016/j.carpath.2017.06.004

7. Kitamura, T., Oishi, H., Fujii, T., Teranishi, K., Yatomi, K., Yamamoto, M., & Arai, H. (2020). Delayed complications due to polymer coating embolism after endovascular treatment. NMC Case Report Journal, 7(1), 5–10. https://doi.org/10.2176/nmccrj.cr.2018-0319

8. Lorentzen, A. O., Nome, T., Bakke, S. J., Scheie, D., Stenset, V., & Aamodt, A. H. (2016). Cerebral foreign body reaction after carotid aneurysm stenting. Interventional Neuroradiology, 22(1), 53–57. https://doi.org/10.1177/1591019915609171

9. Mehta, R. I., Mehta, R. I., Solis, O. E., Jahan, R., Salamon, N., Tobis, J. M., Yong, W. H., Vinters, H. V., & Fishbein, M. C. (2010). Hydrophilic polymer emboli: An under-recognized iatrogenic cause of ischemia and infarct. Modern Pathology, 23(7), 921–930. https://doi.org/10.1038/modpathol.2010.74

10. Shapiro, M., Ollenschleger, M. D., Baccin, C., Becske, T., Spiegel, G. R., Wang, Y., Song, X., Raz, E., Zumofen, D., Potts, M. B., & Nelson, P. K. (2015). Foreign body emboli following cerebrovascular interventions: Clinical, radiographic, and histopathologic features. AJNR American Journal of Neuroradiology, 36(11), 2121–2126. https://doi.org/10.3174/ajnr.A4415

11. Ishikawa, S., Kudo, Y., Miyake, S., Johkura, K., Yamamoto, T., & Nakai, Y. (2023). Cerebral foreign body granulomas after mechanical thrombectomy: Two case reports and a review of the literature. Journal of Stroke and Cerebrovascular Diseases, 32(3), 106990. https://doi.org/10.1016/j.jstrokecerebrovasdis.2023.106990