Spontaneous necrotizing granuloma of the cerebellum: A case report and review of literature

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Introduction

- Granulomas
  - compact collections of inflammatory cells classically resulting from persistence of a non-degradable product or cell mediated hypersensitivity commonly found in the periphery\(^1,2\)
  - Intracranial granulomatous lesions
    - sparsely reported in the literature\(^-2\)
    - predominantly caused by infection, retained surgical or foreign objects, or granulomatous disorders\(^2\)
    - Patient presentation varies depending on location of lesion

Case Presentation

- A 77-year-old female with history of chronic kidney disease and type II diabetes mellitus presents with sinusitis, fatigue, and 20-pound weight loss.
- MRI performed to evaluate extent of sinusitis revealed posterior fossa lesion
- Patient found to have mild cerebellar signs on neurological exam, remainder non-focal
- Top differentials based on imaging included metastatic disease, abycial meningioma, or glioma
- Consequent negative metastatic disease workup
- Underwent suboccipital craniotomy with removal of lesion
- Lesion identified by pathology as necrotizing granuloma of cerebellum and dura

Further workup
- cANCA and pANCA studies negative
- No acid fast or fungal organisms on acid fast and GMS special stains
- No signs of sarcoidosis or foreign body

Most likely differential: the second reported case of an intracranial spontaneous necrotizing granuloma

Diagnostic Steps

- Figure 1. 3D reconstructed MRI of A, sagittal B, Coronal and C, axial planes of thin thin slice T1 with contrast depicting right paracentral, enhancing mass within the posterior fossa along the inferior border of the right cerebellar hemisphere
- Figure 2. Axial T2 Flair sequence MRI at the level of the 4th ventricle within the posterior fossa demonstrating diffuse right cerebellar hyperintensity (long arrow) and to a lesser extent left cerebellar hemisphere hyperintensity (short arrow) corresponding to the large amount of vasogenic edema produced by the lesion.
- Figure 3. A. Diffusion weighted hyperintense axial MRI B. Hypointense ADC correlative at the level of the 4th ventricle and cerebellar vermis demonstrating restricted diffusion of the vermis thought to be related to mass effect from vasogenic edema causing local tissue injury.

Figure 3. A. Hematoxylin and eosin stained sections of material removed from patient's cerebellum and dura A. 20x magnification of granuloma (circle) in the cerebellum. B. 100x magnification showing epithelioid histiocytes (short arrow) and central necrosis (long arrow) of granuloma from image A. C. 20x magnification of granuloma (circle) in the dura. D. 100x magnification showing epithelioid histiocytes (long arrow) and central necrosis (short arrow) of granuloma in the dura.

Conclusion

- Only one previously reported case of a spontaneous necrotizing granulomatous mass\(^2\)
- Common causes that were excluded\(^2\)
  - Autoimmune granulomatous disorders (including Behcet disease, Churg–Strauss syndrome, and granulomatous polyangitis)
  - Sarcoidosis
  - Foreign bodies
  - Infection
- Treatment: Discharged home on post-op day 5 with a three-day steroid taper, follow up MRI at six weeks showing fluid collection in evacuated space. Another MRI two weeks later found the fluid collection markedly decreased in size. Plan is to follow up in one year. Patient states she is happy with her care despite not knowing the cause of the lesion.

References