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NEUROLOGICAL BULLETIN
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Watershed Infarcts in Acute Hypereosinophilia

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A 79-year-old woman presented with generalized weakness, encephalopathy, and non-ST elevation myocardial infarction. Complete blood cell count showed elevated white blood count of 23.7/mm^3 with 67% eosinophils and absolute eosinophil count of 15.9/mm^3. Initial MRI revealed punctate scattered infarcts throughout cerebral and cerebellar hemispheres and the brainstem within watershed territories. There were no episodes of hypotension. Her neurological exam worsened the next day with coma and extensor posturing. Repeat MRI within 24 hours showed worsening with innumerable infarcts.

While neurological involvement is common, it is under-recognized in hypereosinophilic syndrome.[1] Acute watershed infarction is either due to in-situ thrombus formation from hyperviscosity or thromboembolism from a distant source.[2]

References

1. Moore PM, Harley JB, Fauci AS. Neurologic dysfunction in the idiopathic hypereosinophilic syndrome. Ann Intern Med 1985;102:109-114.
2. McMillan HJ, Johnston DL, Doja A. Watershed infarction due to acute hypereosinophilia. Neurology 2008;70:80-2.

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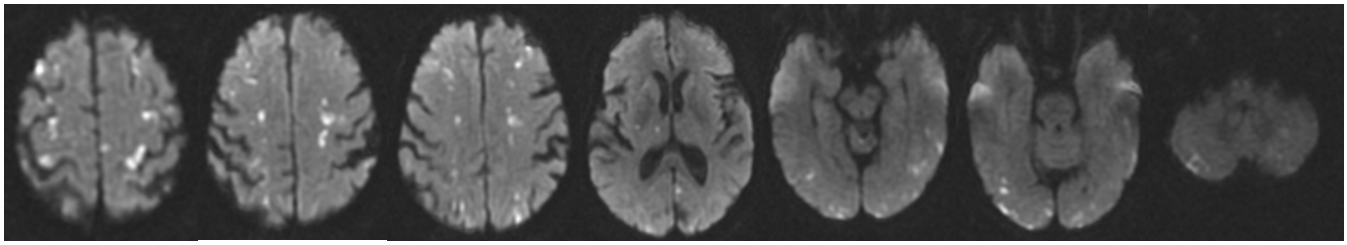

Figure 1: MRI DWI sequences showing acute punctate infarcts in watershed distribution.

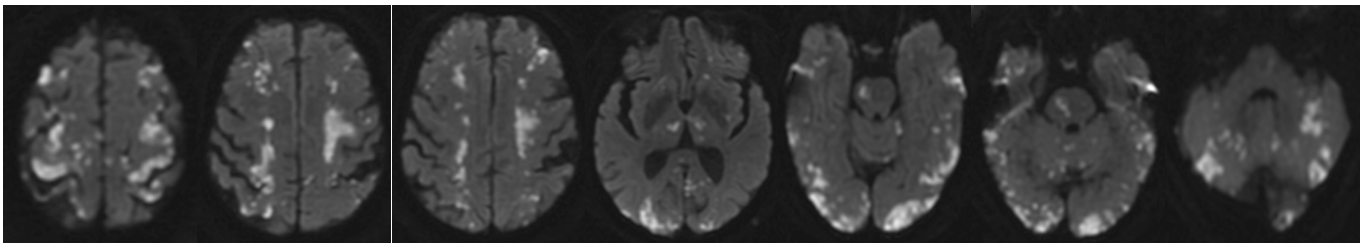

Figure 2: Repeat MRI in 24 hours, after worsening neurological exam.