

Occam's Razor Versus Hickum's Dictum: Getting the Diagnosis Right

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Abstract

Presentation

We present a patient who attended our Emergency Department with a short history of unilateral weakness and slurred speech.

Diagnosis

The patient had suffered a stroke and investigations pointed to a diagnosis of Giant Cell Arteritis (GCA) as the underlying mechanism.

Treatment

However, with further clinical events occurring the possibility of additional pathology was explored. This led to the diagnosis of paroxysmal atrial fibrillation for which a Direct Oral AntiCoagulant was commenced.

Conclusion

This patient's initial differential diagnosis of GCA was supported by raised inflammatory markers, a vasculitic process evident on Computed Tomography Angiogram, an abnormal Temporal Artery Ultrasound and confirmative histology. Ultimately, the insertion of an implantable loop device led to the detection of paroxysmal atrial fibrillation and the commencement of anticoagulation resulted in no further neurological events facilitating discharge to a rehabilitation facility.

Introduction

At medical school, we are taught the skills to take a history and examine a patient. A differential diagnosis is thus derived leading to further investigations and a clinical plan. We endeavour to find one unifying diagnosis, assuming a single cause for a patient's clinical presentation (Occam's razor). Although, as advised by Hickam's dictum, "a man can have as many diseases as he damn well pleases".¹

Case Report

A 70-year-old right-handed male, presented to the Emergency Department (ED) with a one-day history of slurred speech, right sided weakness and an unsteady gait. On examination, mild dysarthria and right arm ataxia was evident. He was in sinus rhythm and was normotensive. There was no evidence of temporal artery tenderness.

Prior to his presentation, he had a three-week history of muscle aches and pains and received a provisional diagnosis of probable Giant Cell Arteritis (GCA). Steroid therapy had been commenced with plans for a temporal artery biopsy.

Investigations: His blood biochemistry was notable for an elevated C-reactive protein (CRP) 20 mg/L (normal less than 3 mg/L) and Erythrocyte Sedimentation Range (ESR) 65 mm/hr (normal 0-22 mm/hour). A 24-hour electrocardiogram (ECG) monitor showed no atrial fibrillation. His Computed tomography (CT) exam of the brain was normal, however, his Magnetic Resonance Imaging scan of the brain demonstrated acute infarcts in the posterior circulation. In addition, his CT angiogram showed evidence of a large vessel vasculopathy consistent with vasculitis in the setting of biopsy proven giant cell arteritis'. The temporal artery ultrasound scan and temporal artery histology was consistent with the diagnosis of GCA (Figures 1 and 2).

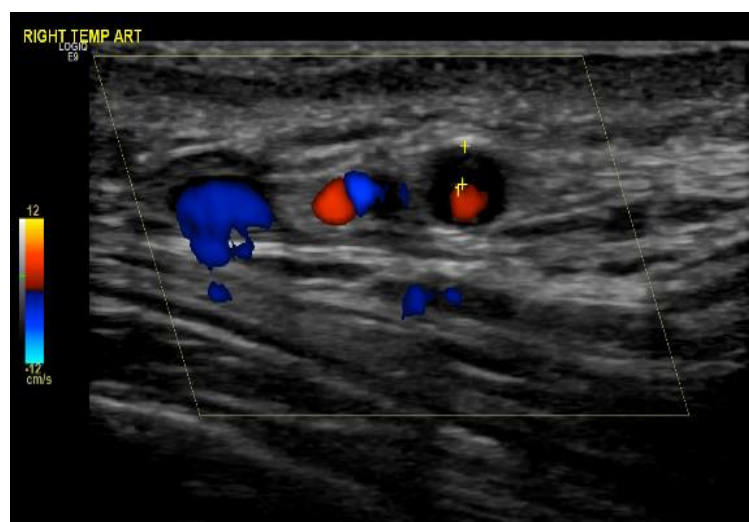


Figure 1: Temporal artery ultrasound - The dark area around the arterial lumen on ultrasonography (yellow crosses) is characteristic of the halo sign.

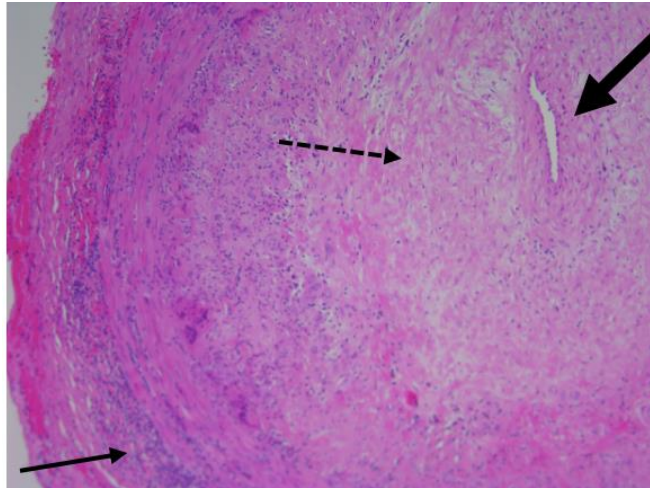


Figure 2: Right temporal artery biopsy specimen under medium power, haematoxylin and eosin stain - Characteristic features of temporal arteritis are seen including a cluster of multinucleated cells (white arrow) and periadventitial lymphocytic inflammatory infiltrate (thin arrow). Age related intimal fibrosis has resulted in a thick lumen (dotted arrow). With continued inflammation, the lumen has become slit-like (thick arrow).

This patient was therefore treated for an ischemic stroke secondary to GCA. His prednisolone therapy was increased to 60 mg per day and aspirin 300 mg per day was concurrently started.

Over the subsequent days, our patient experienced three further transient episodes of speech disturbance. Further brain imaging did not indicate any new ischaemic or haemorrhagic event and our patient was converted to dual antiplatelet therapy (aspirin and clopidogrel). As the patient's CRP and ESR were down trending no changes were made to his steroid therapy and he was referred to cardiology for an implantable loop device which subsequently detected paroxysmal atrial fibrillation (PAF). The patient was anticoagulated thereafter and had experienced no further acute neurological events at the time of his last outpatient review.

Discussion

The nature of the diagnostic process is iterative; by gathering information, the goal is to reduce diagnostic uncertainty, narrow down the diagnostic possibilities and develop a more precise and complete diagnosis.² PAF can be difficult to diagnose if the patient does not have an event whilst being monitored.³ However, the further vascular events on 'optimum treatment', led to a critical reappraisal of our diagnostic thinking.

In conclusion, this patient had two significant diseases – GCA and PAF both of which are common in the older patient. While the patient initially had the clinical and radiological features of GCA, the continuing vascular events led us to reconsider the diagnostic process and arrange further investigations which led to the second diagnosis of PAF and hence Hickam's dictum has been followed.

Declaration of Conflicts of Interest:

The authors declare that there are no conflicts of interest.

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