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Bilateral Occipital Ischaemic Stroke Due to Sepsis

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Abstract

Presentation

A 48-year-old female presented with LIF pain. CT Abdomen/Pelvis revealed sigmoid diverticulitis with pericolonic abscess (Hinchey 1B).

Diagnosis

X became peritonitic while awaiting percutaneous abscess drainage. An emergency Hartmann's procedure was therefore performed. She bilateral visual loss post-extubation which was attributed to bilateral occipital infarcts seen on MRI Brain. TOE, telemetry, CT Angiogram Intracranial/Carotids, and a Haematology blood panel revealed no aetiological source. The cause of X's stroke was concluded to be a combination of sepsis-related cerebral hypoperfusion and hypercoagulability.

Treatment

She was given Aspirin 300mg daily for two weeks and discharged on Aspirin 75mg od for life.

Discussion

This case underscores the association between infection and ischaemic stroke, even without an underlying cardiac, vascular, or haematological cause. It emphasises the importance of rapid and effective source control in patients with infection to prevent sepsis and associated sequelae. This includes stroke, which can precipitate significant and permanent functional deficits in otherwise young and healthy patients.

Introduction

Infection has been implicated as a risk factor for ischaemic stroke, with multiple mechanisms proposed to underlie this relationship.[1-3] The case below demonstrates the association between sepsis and stroke; a 48-year-old female patient who developed bilateral occipital infarcts due to perforated diverticulitis.

Case

A 48-year-old female (X) presented to hospital with LIF pain and per rectal bleeding. Her medical background and family history were non-contributory, and she took no regular medication. CT Abdomen/Pelvis revealed sigmoid diverticulitis with a pericolonic abscess (Hinchey 1B). She was kept NPO, prescribed TPN, and initially improved under conservative management with IV antibiotics (Cefuroxime and Metronidazole).

X underwent repeat CT Abdomen/Pelvis on day six post-admission, which showed that her abdominal abscess had reduced in size. However, a new left renal infarct was also visible. The subsequent plan was to treat with therapeutic low-molecular weight heparin and pursue percutaneous abscess drainage under CT guidance. However, X became hypotensive, tachycardic, and peritonitic while being transferred to Interventional Radiology. A repeat CT Abdomen/Pelvis showed intra-abdominal free fluid and pneumoperitoneum consistent with bowel perforation and abscess rupture. She then underwent an emergency Hartmann's procedure.

X was gradually weaned off sedation, ionotropes, and antibiotics in the ICU. She was extubated on day fifteen post-admission and complained of bilateral blurred vision on waking. Neurological examination suggested global visual field deficits. A non-contrast CT Brain suggested bilateral occipital infarcts, which was confirmed on MRI Brain (see Image 1).

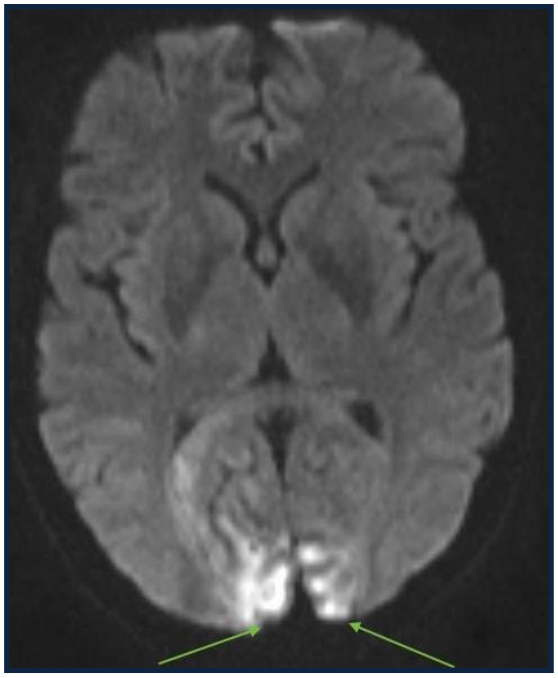

Image 1: Axial DWI MRI Brain showing bilateral occipital infarcts.

X next underwent aetiological investigation. CT Carotid/Intracranial Angiogram showed basilar artery thrombus but outruled carotid disease (see Image 2). A TOE did not find any evidence of LV/LAA/peri-valvular thrombus, infective vegetations, or an atrial septal defect, and telemetry did not detect any arrhythmia. A normal platelet count and fibrinogen level excluded DIC and TTP. Haematology concluded that X's renal and occipital infarcts were precipitated by a combination of non-DIC hypercoagulability and sepsis-induced hypoperfusion. She received 2 weeks of Aspirin 300mg po daily and was prescribed Aspirin 75mg po od for life thereafter.

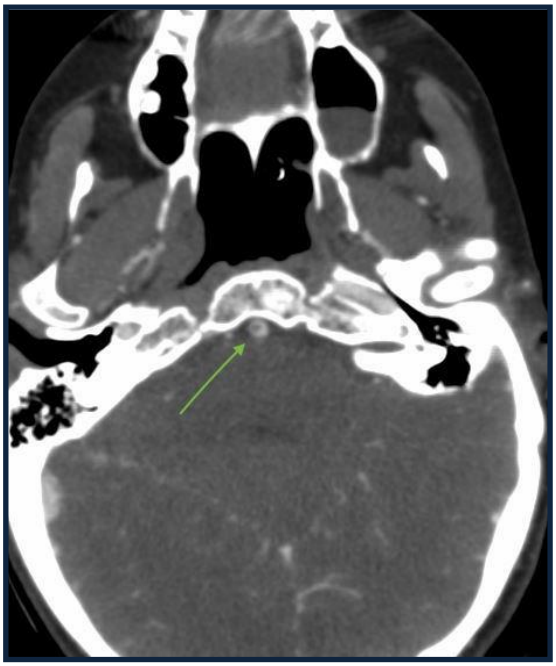

Image 2: Axial MIP CT Angiogram showing basilar artery thrombus.

Discussion

Ischaemic stroke is becoming more common amongst young individuals and can occur due to embolism, thrombosis, and systemic hypoperfusion.[4-5] Importantly, infection can be found in up to 13% of young stroke patients belonging to any of the aforementioned aetiological subcategories.[6] For example, sepsis has been associated with cardio-embolic stroke through the development of new-onset Atrial Fibrillation.[7] Thrombosis can occur in infected patients due to co-existing coagulopathy, which has been found in up to 80% of septic patients.[3,8] Cerebral infarction can also occur if infection precipitates haemodynamic instability and systemic hypoperfusion including the brain.[3]

X's case adds credence to the link between infection and cerebral thrombosis. This can be clinically challenging however, as sepsis-related hypercoagulability can range from severe prothrombotic states like DIC to milder forms such as immunothrombosis (fibrin activation to control local infection).[9-10]

Indeed, X's basilar clots and renal infarct occurred in the absence of any detectable haematological derangements. This underscores the need for a high clinical index of suspicion in infected patients for thrombosis including stroke. Lastly, X's deterioration into septic shock is a classic example of the haemodynamic compromise that can occur during infection, with end-organ damage through cerebral hypoperfusion. Aggressive source control must therefore be pursued to prevent sepsis and associated sequelae such as stroke, which can severely reduce function and quality-of-life in young and previously healthy patients like X.

Declaration of Conflicts of Interest:

There are no financial interests or connections, direct or indirect, or other situations that might raise the question of bias in the work reported or the conclusions, implications or opinions stated – including pertinent commercial or other sources of funding for the individual authors or for the associated departments or organizations, personal relationships, or direct academic competition.

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