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CASE REPORT

A Rare Case of Cerebral Infarct Leading to Acute Heart Failure With a 4-Year Follow-Up

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Abstract

It is not uncommon for ischaemic heart conditions to cause cerebral infarcts. However, what is rarely seen is a cerebral infarct leading to heart failure. This case report describes a 42-year-old male admitted with acute onset symptoms in keeping with a posterior circulation infarct, who later deteriorated during admission as a result of left ventricular heart failure. At the 18-month follow-up, the patient's overall condition had improved due to management for the cerebral infarct and the heart failure which restored the patient's heart function. The patient remained symptom-free throughout the follow-ups across 4 years. Due to the lack of pre-existing heart conditions in this patient, the most plausible cause of their heart failure was as a result of the reduced blood supply to the lateral medulla. This phenomenon has been rarely commented on thus far and warrants further discussion around its pathophysiological mechanisms and management.

Key words: case report; acute heart failure; cerebral infarct; posterior reversible encephalopathy syndrome

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Introduction

The term cerebral infarction should be used when there is evidence of brain or retinal cell death due to cerebral ischaemia (Saver, 2008). 20% of cerebral infarcts occur in the posterior circulation, with lateral medullary syndrome being the most prevalent posterior ischaemic stroke syndrome (Forshing et al, 2019). This was originally defined as a clinical syndrome caused by acute ischaemia or infarction of the lateral medulla oblongata. A lack of blood supply is consequential to an occlusion of the intracranial portion of the vertebral artery, the posterior inferior communicating artery or its branches. This patient suffered from vertebral artery dissection, commonly seen in young patients, inducing cardiac dysfunction. It is important to note there have been links of an infarct to the right insula resulting in sympatho-vagal imbalance therefore raising catecholamine levels, aggravating cardioelectrical instability and raising blood pressure, worsening ventricular strain (Oppenheimer and Cechetto, 2016). Recent study has also suggested in severe cases, a possible link between reduced blood supply to the hypothalamic-pituitary-adrenal axis leading to an exaggerated cardiovascular stress response resulting in

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heart failure (Doehner et al, 2023). However, the notion of decompensation of heart failure through the route of reduced blood supply to the medulla is an unfamiliar concept hence its novelty promotes further investigation.

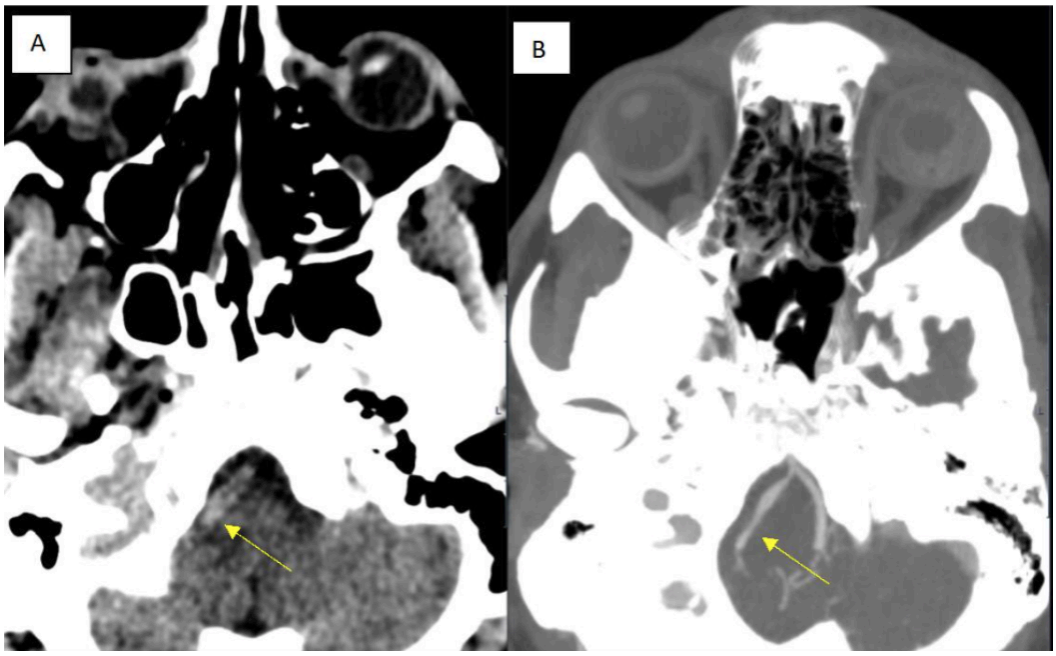

Fig. 1. Unenhanced computed tomography (CT) head and CT angiogram (CTA) imaging at initial presentation. (A) CT head showing focal hyperdensity in the V4 segment of the right vertebral artery (yellow arrow). (B) CTA showing corresponding irregularity (yellow arrow) suggestive of dissection.

Case Report

Day 1–10

A 42-year-old male with no past medical history was admitted with a 3-day history of occipital headaches, right-sided ataxia, diplopia on lateral gaze, vertical nystagmus, vertigo and vomiting. He was a non-smoker. The patient also reported severe hiccups which, compounded by the aforementioned symptoms, were indicative of a posterior circulation infarct. The patient had an initial National Institutes of Health Stroke Scale score of 5 (Odderson, 1999), dropping to 3 after 24 hours. Computed tomography (CT) showed no acute intracranial findings but revealed a small hyperdensity of the right vertebral artery (Fig. 1A). Cerebral infarct is a clinical diagnosis and in an acute presentation a CT scan can be normal. Hyperdensity of the right vertebral artery with the patient's clinical presentation suggested a possible thrombus in the right vertebral artery. This prompted us to do a CT angiogram of the carotid and vertebral arteries. CT angiogram showed a small, filling defect and irregularity of the right vertebral artery (Fig. 1B), suggestive of a small thrombus or possible dissection. Chest X-Ray (CXR) was normal. However, magnetic resonance imaging (MRI) showed an acute infarct within the right lateral medulla (Fig. 2A,B). Diffusion weighted imaging (DWI) sequence of the MRI brain has very

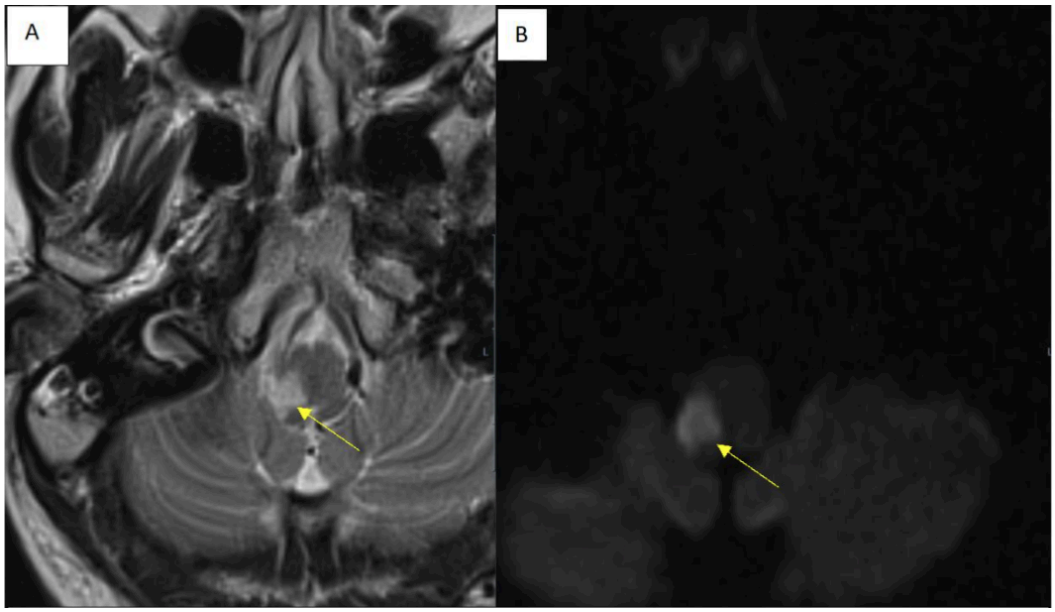

Fig. 2. Magnetic resonance imaging (MRI) brain imaging axial T2 weighted imaging (T2WI) and diffusion weighted imaging (DWI) sequence at presentation. (A) Acute right lateral medullary infarct (yellow arrow) in the axial T2WI. (B) Acute right lateral medullary infarct (yellow arrow) in the corresponding DWI.

high sensitivity and specificity for acute cerebral infarcts if done within 2 weeks of the symptoms. The common symptoms of lateral medullary infarction include vertigo, nystagmus, nausea and vomiting, ataxia, hiccups and dysphagia. Our patient had most of these symptoms. His initial electrocardiogram (ECG) showed multiple ventricular ectopic beats (VEBs), albeit sinus rhythm, and an echocardiogram estimated a left ventricular ejection fraction (LVEF) of 64%. His young stroke work up (erythrocyte sedimentation rate (ESR), lupus, anti-cardiolipin antibodies, antinuclear antibodies (ANA), human immunodeficiency virus (HIV), syphilis) was normal. Dual antiplatelet therapy (Aspirin 75 mg omne die (o.d.) and Clopidogrel 75 mg o.d.) and Atorvastatin 40 mg o.d. (for secondary prevention of cerebral infarct) were commenced; chlorpromazine 25 mg ter in die (t.i.d.) for the hiccups; and paracetamol 1 g quater in die (q.i.d.) for the headaches were given for 3 weeks. The subsequent 24-hour ECG revealed VEBs of 42%.

Day 10–20

On day 10 of the patient's admission, after 'straining' on the toilet, he reported worsening headaches (pain severity of 9/10) and sudden-onset right homonymous hemianopia with blurring of vision; staff reported new-onset generalised tonic-clonic seizures. His oxygen levels desaturated, mandating oxygen supplementation and chest examination revealed bilateral course crackles in his lungs up to the middle zones. CXR confirmed pulmonary oedema (Fig. 3). Repeat CT angiogram revealed mild aneurysmal dilation of the distal vertebral artery (Fig. 4A). This usually implies the complication and progression of an underlying dissection. A CT head (Fig. 4B) showed bilateral hypo-attenuation of the occipital lobes. MRI/Magnetic resonance angiogram (MRA) confirmed acute right vertebral artery dissection, pro-

gressing from baseline, as well as cortical/subcortical peri-occipital abnormalities suggestive of posterior reversible encephalopathy syndrome (PRES) (Fig. 5A–D). Most of the patient's neurological symptoms could be attributed to the progression of the vertebral dissection causing PRES. Blood tests showed troponin increase (26 ng/mL to 255 ng/mL) and elevated brain natriuretic peptide (BNP) (30,815 pg/mL). He was admitted to the High Dependency Unit and was treated with furosemide infusion and high flow oxygen. His seizures were treated with levetiracetam and the dual antiplatelets were switched to treatment dose of dalteparin (1.5 mg/Kg). The decision for anticoagulation was taken due to the progression of his vertebral dissection to prevent further cerebral infarcts. A repeat echocardiogram showed reduced LVEF (35–40%). His heart failure management continued in the cardiology ward. CT coronary angiogram done here was normal. He started improving and was transferred back to the stroke unit.

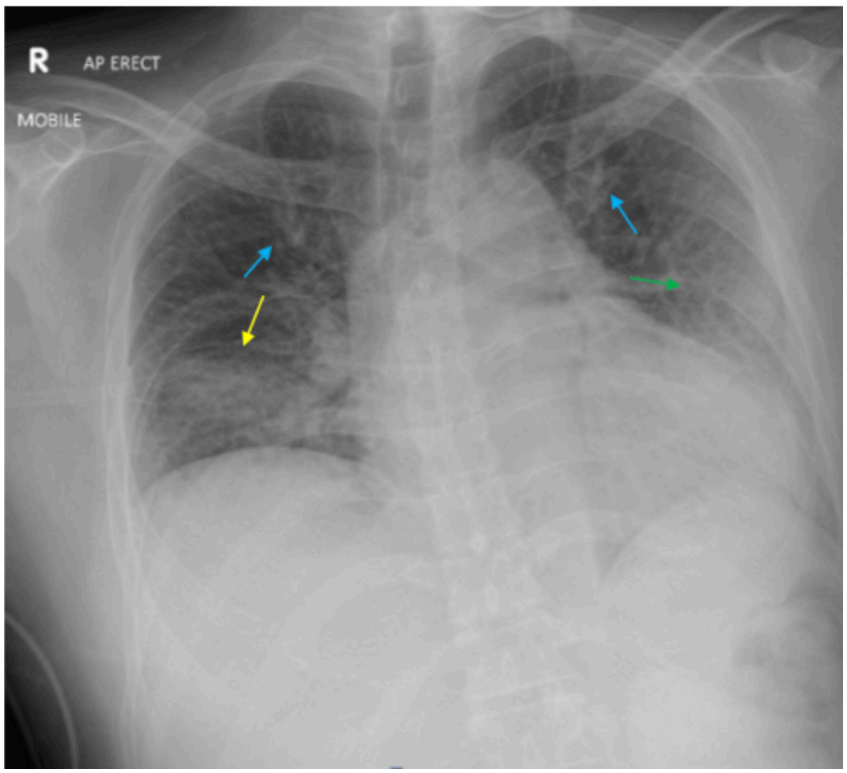

Fig. 3. Chest X-Ray (CXR) showing bilateral pulmonary parenchymal opacification in the right lower zone (yellow arrow) and the left mid zone (green arrow) with upper lobe pulmonary venous diversion (blue arrows) consistent with pulmonary oedema. R, right.

Day 21–30

The patient started to recover, and his cerebral infarct and heart failure symptoms continued to improve. He was started with warfarin and the target international normalized ratio (INR) was reached with close monitoring. Another repeat echocardiogram showed an estimated LVEF of 55% MRI brain at 1 month showed almost complete resolution of parenchymal changes with no residual gliosis (Fig. 6A).

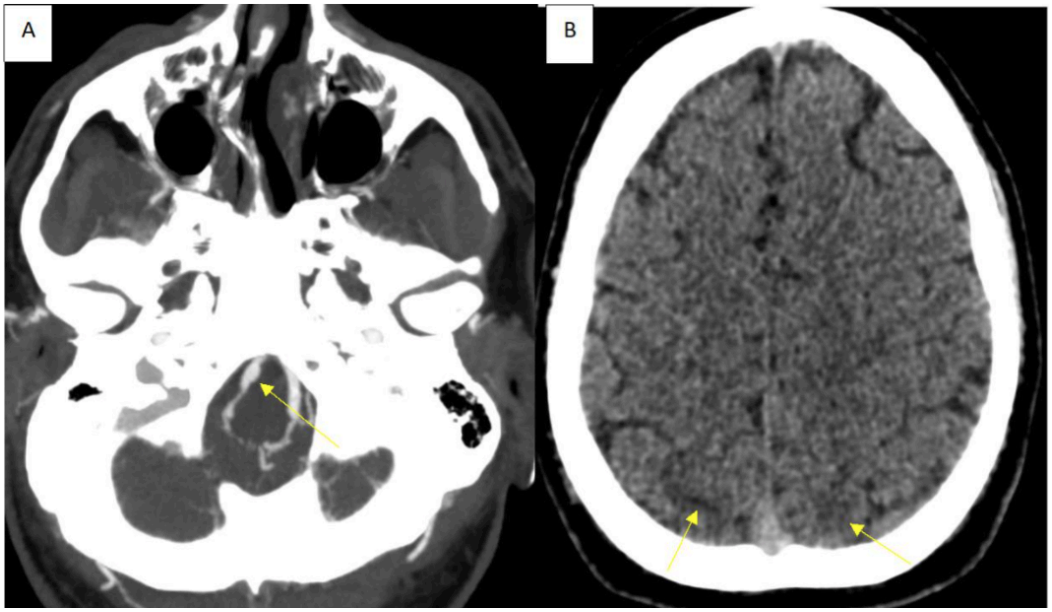

Fig. 4. Computed tomography angiogram (CTA) and unenhanced computed tomography (CT) head imaging on day 10 during his deterioration. (A) CTA showing persistent irregularity of the right vertebral artery with new pseudoaneurysm formation (yellow arrow). (B) CT head showing symmetrical oedema in the posterior parietal lobes (yellow arrows).

Cardiac MRI at 1 month showed a nondilated left ventricle with low normal systolic contractility (LVEF 58%) and no convincing myocardial oedema, fibrosis, infiltrate or infarction.

He was prescribed medications targeting heart failure (bisoprolol 5 mg b.i.d., ramipril 1.25 mg od, eplerenone 12.5 mg o.d.), anti-epileptics (levetiracetam 500 mg b.i.d.), atorvastatin 40 mg o.d., chlorpromazine 25 mg o.d. and warfarin (target INR 2.0–3.0) for his vertebral dissection and cerebral infarct and discharged from cardiology and stroke with initial 6-monthly follow-ups with both teams.

1 Month–6 Months

The patient received community therapy and showed clinical improvement. MRI brain at 3 months (Fig. 6B) showed complete resolution of the parenchymal changes with no residual gliosis, which had further improved from his 1-month MRI brain scan (Fig. 6A). These findings of the reversibility of parenchymal changes from abnormal to normal combined with improved clinical symptoms of the patient further supported our diagnosis of PRES.

6 Months–4 Years

His serial 24-hour ECGs all showed subsequent reductions of VEBs (42% to 38% in 6 months, 11% in 1 year, 3%, and <1% at 3 years). A 2-year follow-up echocardiogram revealed resolved heart failure, with an ejection fraction (EF) of 66%.

His warfarin was switched to apixaban after a year, later at 18 months this was switched to clopidogrel as a secondary prevention to his vertebral dissection and cerebral infarction.

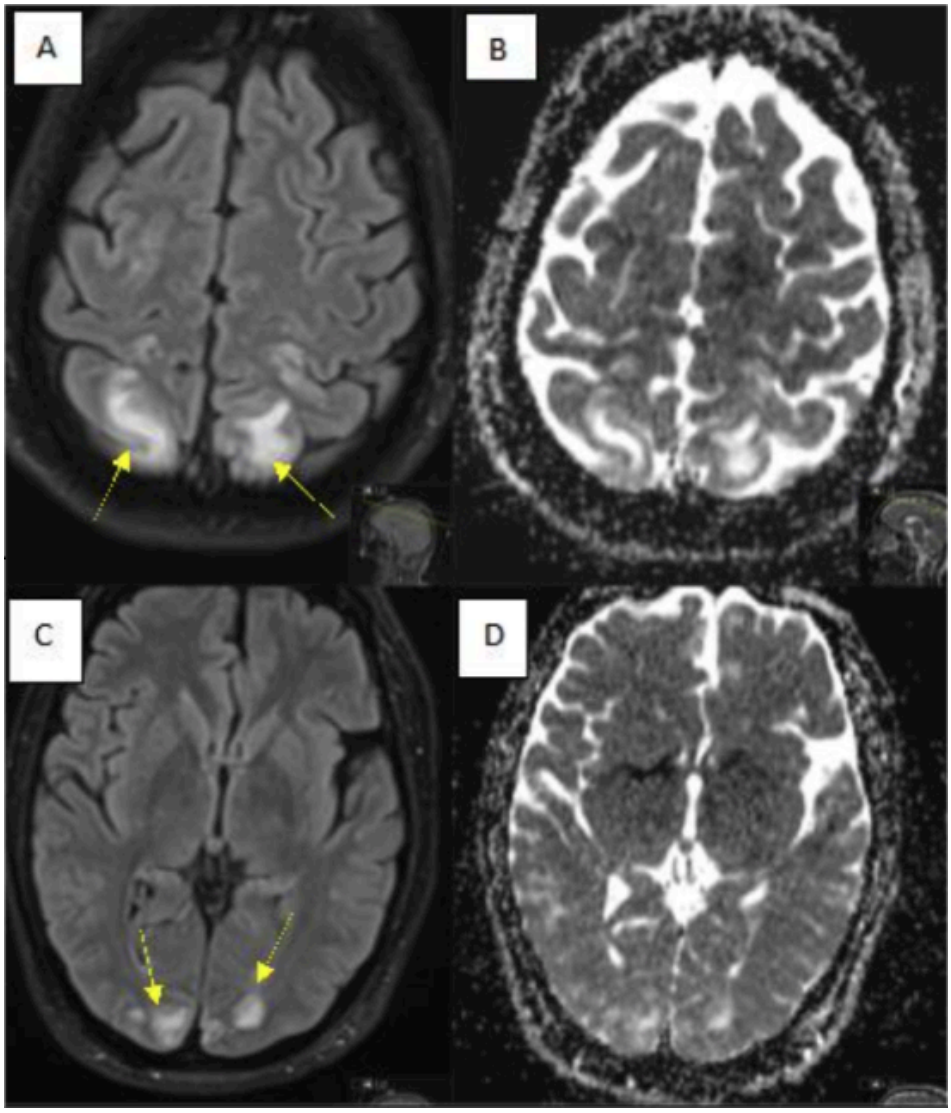

Fig. 5. Magnetic resonance imaging (MRI) fluid-attenuated inversion recovery (FLAIR) imaging and apparent diffusion coefficient (ADC) map on day 10 during the patient's deterioration. (A) MRI FLAIR imaging showing bilateral symmetrical areas of vasogenic oedema (yellow arrows) in the parietal lobes. (B) MRI ADC map showing no associated restricted diffusion in the parietal lobes, findings consistent with posterior reversible encephalopathy syndrome (PRES). (C) MRI FLAIR imaging showing bilateral symmetrical areas of vasogenic oedema (yellow arrows) in the occipital lobes. (D) MRI ADC map showing no associated restricted diffusion in the occipital lobes, findings consistent with PRES.

At 2-year review, the patient could mobilise without a frame for some time, had improved concentration and no episodes of double vision. There were also no complaints of chest pain, palpitations, or breathlessness.

At 4-year review, he had started working and remained cardiovascularly and neurologically stable. He is currently taking bisoprolol b.i.d. (5 mg AM; 2.5 mg PM), atorvastatin 40 mg o.d., levetiracetam 500 mg b.i.d., clopidogrel 75 mg o.d. The Care Checklist has been attached as **Supplementary material** associated with this article.

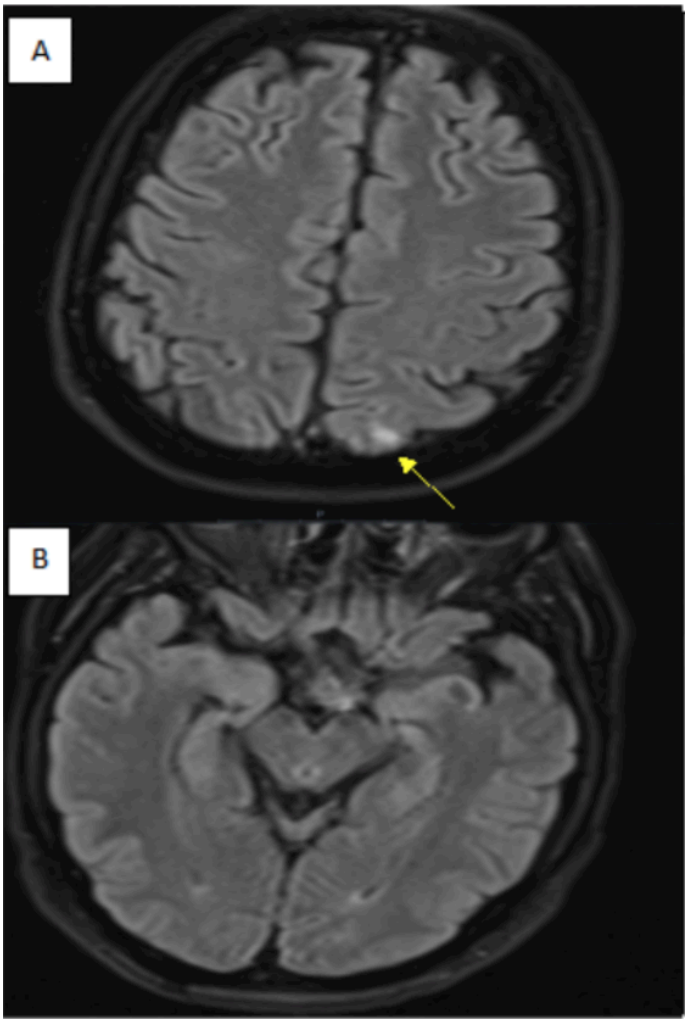

Fig. 6. Follow-up magnetic resonance imaging (MRI) at 1 and 3 months. (A) MRI brain fluid-attenuated inversion recovery (FLAIR) imaging at 1 month showing almost complete resolution (yellow arrow) of parenchymal changes with no residual gliosis. (B) MRI brain FLAIR imaging at 3 months showing complete resolution of parenchymal changes.

Discussion

The patient's deterioration began whilst 'straining' on the toilet, potentially stimulating an increased intracranial pressure thus, propagating a vertebral artery dissection and dampening parasympathetic activity. This would have potentiated PRES and the florid pulmonary oedema that was seen.

The vertebral artery supplies both the medulla oblongata and occipital lobes. Therefore, a vertebral artery dissection would present with occipital headaches alongside PRES, whereby cerebral blood flow is disturbed. Physiologically, heart rate and contractility are controlled by the vasomotor centre in the reticular substance of the medulla. More specifically, parasympathetic control of the heart to reduce heart rate occurs via the parasympathetic efferent pre-ganglionic neurons within the nucleus ambiguous of the medulla oblongata (Gronda et al, 2022). The signals from the nucleus ambiguus get carried by the efferent branches of cranial nerve X to reduce heart rate (Petko and Tadi, 2020). A lack of blood supply to the

lateral medulla, would thus decrease parasympathetic activity and proportionally overload sympathetic activity, which could have led to the patient's unanticipated report of hiccups. This would also explain the heart failure with reduced EF, since unopposed sympathetic activity would stimulate the splanchnic nerves that synapse on chromaffin cells within the adrenal gland to release acetylcholine resulting in the release of adrenaline. Adrenaline acts on the beta-1 receptors to raise heart rate and myocardial contractility (Dalal and Grujic, 2023), leaving less time for the ventricles to fill before contracting—reducing EF. We hypothesise this as catecholamine release has led to similar findings in studies showing hypoperfusion to the right insula can lead to the progression of pre-existing heart failure due to raised catecholamine levels (Doehner et al, 2023). This is also similar to the ventricular-atrial strain caused by vasoconstriction and high blood pressure variability due to reduced blood supply to the hypothalamic-pituitary axis; however instead of cortisol being the driving factor for cardiac dysfunction, in this case, we consider that hypoperfusion to the lateral medulla stimulates adrenaline release potentially resulting in the downstream effects causing heart failure. The generalised tonic-clonic seizure was likely secondary to the cerebral infarct and/or PRES as this occurs in 60–75% of cases of patients with PRES (Lu et al, 2021).

Another hypothesis is that we may have seen the examination findings of pulmonary oedema in our patient's chest directing us to a diagnosis of heart failure (HF) due to the seizure the patient experienced. Seizures have been known to increase systemic vascular resistance and thus lead to left ventricular failure and neurogenic pulmonary oedema (NPO) as a result (Brambrink and Dick, 1997). Similar cases have described patients with NPO secondary to vertebral artery dissection (Raja et al, 2018; Aljishi and Jayathissa, 2018). Whilst the pulmonary oedema is more likely to be cardiogenic in nature due to the reduced EF, the mechanism causing NPO is not fully understood, we know it is likely related to excessive sympathetic activation through medulla oblongata and hypothalamus injury, further potentiating the causative mechanism to be due to the infarct to the lateral medulla (Davison et al, 2012). Therefore, this case offers the novel consequence of heart failure due to the vertebral artery dissection allowing us to broaden our perspectives on the neurocardiac interactions during cerebral infarctions.

VEBs are premature heartbeats originating from the ventricles which reduce ventricular filling through ventricular contraction before atrial, explaining the reduced EF. If more than 10% of the heartbeats are VEBs, as seen in this patient (42%), dyssynchrony can occur around the left ventricle with the rest of the heart (Ng, 2006). The presence of VEBs >10% on the 24-hour tape prior to the patient's deterioration may suggest an underlying heart condition was present; however, there was no further evidence for this. This was supported by the reversibility of the LVEF on echocardiogram and normal cardiac MRI and CT coronary angiogram. This high ectopic burden was likely secondary to his initial infarct.

It is also important to consider the possibility of this improvement in the patient's EF and VEBs being due to successful HF management rather than the resolution of medullary dysfunction, however, a very few percentage of cases of heart failure with reduced EF are reversed with minimal medical management, making the

improvement more likely to be due to the combination of resolution of medullary dysfunction and treatment of HF.

It should also be noted that the heart failure may have been multifactorial as PRES has been seen to cause widespread vasogenic oedema and cardiac stress in a similar case (Perwez et al, 2021). The authors noted that both PRES and cardiac stress disproportionately occur in postmenopausal females linking shared aetiological grounds. Their literature search showed 12 females and one 69-year-old man reported to have both conditions. Our patient is a man in his early 40s. Furthermore, our patient had 40% VEBs at presentation and during his first 9 days prior to developing seizures and PRES on day 10, making both of these conditions unlikely to be the leading differential cause of HF.

Takotsubo cardiomyopathy is another possible pathology to be considered as it is a temporary heart condition occurring after extreme physical or emotional stress. Since our patient's trigger was straining on the toilet—we could relate the left ventricular (LV) dysfunction on imaging to Takotsubo cardiomyopathy. This disease is also as a result of large-scale production of catecholamines leading to myocardial hypokinesia via direct cardiomyocyte toxicity and induction of coronary microvascular dysfunction (Komamura, 2014). Although this is less likely to be the cause of HF in our patient as their VEBs only came down to the clinically significant $<$10% after 1 year, whereas Takotsubo cardiomyopathy usually resolves within days to weeks. In addition, our patient had global LV impairment when he deteriorated and not the focal apical hypokinesia or ballooning that is characteristic of Takotsubo cardiomyopathy, making this less likely to be the causative mechanism.

Limitations

The nature of this being a single case report provides valuable insights into the unique clinical presentation following an infarct to the lateral medulla, however, its findings are inherently limited by the lack of generalisability and statistical power. The conclusions drawn are speculative and may not reflect broader population trends; thus, this report should be viewed as hypothesis-generating rather than definitive, serving as a foundation for future studies to validate or refute the proposed mechanism through larger controlled investigations to determine the clinical significance of the observations.

Conclusion

Whilst the case of heart conditions that consequently lead to reduced blood supply to a region within the brain, by clot formation is not unheard of, the opposite has not been reported often. However, there is increasing evidence that cerebral infarcts can affect the heart function via mechanisms such as reduced blood supply to the hypothalamic-pituitary-adrenal axis, and the right insula, which leads to ventricular and atrial strain. This case demonstrated that when an infarct within the region of the brain controlling heart rate occurs such as the nucleus ambiguus within the medulla oblongata, heart failure is seen as a consequence. Our hypothesis is that the adrenaline release is triggered due to overloaded sympathetic activity which would increase heart rate and myocardial contractility to reduce the ejection

fraction. This case report emphasizes the significance of understanding the link between the region of the brain that the cerebral infarct occurs within, and complications attributed to decreased brain control. It is important to monitor these patients closely, anticipate these complications and manage them swiftly to get the best outcome of these serious conditions.

Learning Points

- The unique aspect of the case is that the patient had no risk factors for either a cerebral infarct or heart disease. Yet the patient suffered from the vertebral dissection that led to the cerebral infarct and subsequent deterioration.
- Cerebral infarcts can cause heart dysfunction due to various mechanisms which include the reduced blood supply to the right insula, the hypothalamic-pituitary-adrenal axis and the lateral medulla.
- Cervical dissection (carotid and vertebral) can be treated with either antiplatelet therapy or anticoagulation. Anticoagulants are considered when there is a progression of a dissection, or a floating thrombus detected in the arteries.
- PRES can occur in the context of hypertension, renal failure, immunosuppressive or chemotherapy drugs and with autoimmune disorders.
- Frequent VEBs $>10\%$ are a marker of cardiac dysfunction often resulting in reduced ejection fraction.
- It is important to follow acute heart failure and chronic heart failure management algorithms to obtain the optimal health outcomes.

Availability of Data and Materials

All the data of this study are included in this article.

Author Contributions

AS identified the case and was the principal clinician for the patient. AS collected the data for the case. AS annotated the images. AS and SR analysed and interpreted the data. SR wrote the first draft of the case report. Both authors made principal editorial changes to the manuscript. Both authors read and approved the final manuscript. Both authors participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Written informed consent was obtained from the patient for publication. This research was completed in accordance with the Declaration of Helsinki.

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Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://www.magonlinelibrary.com/doi/suppl/10.12968/hmed.2024.0752.

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