CASE SERIES: THE DIVERSITY OF CARDIAC DEATHS IN ISCHEMIC STROKE IN A TERTIARY NEUROLOGY CENTRE

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INTRODUCTION

Cardiac complications in brain injury is a well-documented phenomenon. The spectrum of abnormalities includes hypertension, hypotension, electrocardiogram (ECG) changes, cardiac arrhythmias, release of biomarkers of cardiac injury, and left ventricular (LV) dysfunction.1 This case series is a retrospective review of the confirmed cardiac deaths over a period of twelve months in patients who were admitted to our center for ischemic stroke. Ten patients were identified as confirmed cardiac deaths out of the 76 patients who died whilst being treated for a stroke. All the patients included in this case series had at least two out of three of the following- symptoms suggestive of myocardial injury, new or evolving ECG changes and a rise in cardiac markers from baseline. Nine out of ten were males. Three were known to have a pre-existing heart condition. The management for the stroke syndrome itself is outside the scope of this review.

Keywords: Cardiac complications, ischemic stroke

CASE 1

A 61-year-old Malay woman was brought in after she was found to be not moving the right side of her body for one day. She had end-stage-renal failure (ESRF) on regular dialysis for seven years, diabetes mellitus (DM), hypertension, hepatitis C with liver cirrhosis and bilateral cataracts. She had multiple admissions to the hospital, including a recent one for pneumonia. Her Glasgow Coma Scale (GCS) was E4V1M5. The blood pressure (BP) was low and supported with a low dose of intravenous (IV) noradrenaline while oxygen saturations (SpO2) were 100% on two liters of nasal prong oxygen. She was afebrile. There were bilateral crepitations up to midzones on lung auscultation. Neurological assessment revealed a right eye ptosis with left gaze preference. The gag reflex was absent. Power grading over the right side was 0/5 while on the left side was 3/5 in the upper limbs and 2/5 in the lower limbs. There was generalized areflexia except for the left biceps, which was 2+. The plantars were upgoing on the right side. The National Institutes of Health

Stroke Scale (NIHSS) was 27 on admission, with a clinical impression of a left total anterior circulation infarct (TACI). A plain brain computed tomography (CT) scan showed a recent left parieto-occipital infarct on a background of old lacunar infarct and cerebral atrophy (Figure 1). The patient was started on Aspirin and a statin. Admission ECG showed sinus rhythm, with T inversions in leads V2 to V4. Chest radiograph revealed congestion in the lung fields with patchy opacities suggestive of infection. She was therefore also treated for hospital-acquired pneumonia in view of the recent hospital admission and started on IV Tazosin. During the admission to the ward, her BP continued to be low, requiring double inotropic support. There had been one episode of coffee ground aspirate of 50ml on the third day of admission, however, no malaena was found per-rectally and this resolved spontaneously. OGDS was therefore not performed. The patient's GCS and neurological findings remained the same throughout her admission. An echocardiogram done on the fifth day of admission showed severe tricuspid regurgitation, mild mitral regurgitation and aortic regurgitation, with an EF of 53%. The left atrium was dilated, with no thrombus. During the ninth day of admission, her hemoglobin (Hb) was noted to have dropped from 10.8 to 8.6g/dL. ECG showed deeper T inversions in leads V2 to V4, leading to the clinical impression of anemia-induced acute coronary syndrome. Troponin T levels had doubled from 117 to 542 µg/L. Aspirin was withheld. On the tenth day of admission, during dialysis and blood transfusion, the patient's BP was unrecordable with an absent pulse, and asystole on her cardiac monitor. No cardiopulmonary resuscitation (CPR) was done in view of her do-not-resuscitate (DNR) status.

CASE 2

A 55-year-old Indonesian man woke up from sleep with symptoms of dizziness, slurred speech and worsening right-sided weakness. He had a previous stroke with right hemiparesis a year before but was independent and had not been on proper follow up. GCS was full and BP was 164/107mmHg. There were crepitations in his right lung. The neurological examination revealed right eye ptosis with a left upper motor neuron (UMN) facial nerve palsy. His uvula was deviated to the right. The power was 5/5 in all four limbs while the reflexes were 3+ on the right side. Plantars were upgoing on the right side. A plain CT scan of the brain showed old internal capsule and corona radiata infarcts on the left. He was in sinus rhythm, with a heart rate (HR) of 75/min on ECG. The white cell count was 14.8 while the hemoglobin (Hb) was 17.5g/dL with a hematocrit of 50%. The platelet count was normal. The impression on admission was a right lacunar infarct with ataxic hemiparesis. NIHSS was only 2. He was also worked up for secondary polycythemia, although heavy smoking was a likely cause. Antibiotics were started to cover for aspiration pneumonia. Five hours later, he suddenly appeared cyanosed with an SpO2 of 27%. The BP was unrecordable while his pupils were fixed and dilated. He required defibrillation for one episode of VF during resuscitation (Figure 2). Return of spontaneous circulation (ROSC) was achieved after 30 minutes of CPR. Troponin T levels were elevated at 6989 from a baseline of 64. He was treated as non-ST elevation myocardial infarction (NSTEMI) and started on Aspirin. Subsequently, jerky movements of all four limbs were noted and he was started on Valproate. A repeat brain CT showed infarcts in the right middle cerebral artery (MCA) territory, right occipital, bilateral cerebellum and left pontine territories. The patient was ventilated and sedated with inotropic support. He had spiking temperatures, and by day three of admission there were no brainstem reflexes. His electroencephalogram (EEG) showed severe diffuse cortical dysfunction. Another

repeat plain CT of the brain showed unchanged infarct territories with generalised cerebral edema. He went into asystole on Day 7 of admission. No CPR was done as per family's wishes.

CASE 3

A 55-year-old Indian man was brought in for left arm weakness, drowsiness and dyspnea of one-day duration. His co-morbids were Type 2 DM, hypertension, bilateral below knee amputations done 15 years earlier and blindness due to cataracts for 10 years. He was not under any follow-up. His GCS was E4V4M6 with a HR of 90/min and BP of 130/70 mmHg. SpO2 was 95% under room air, with reduced air entry in the left lung. There were bilateral cataracts with no perception to light. Tone was reduced on the left side, with a power of 0/5. Power over the right side was 4/5. His plain brain CT showed multifocal infarcts with recent infarcts in the right MCA territory. ECG showed small voltage complexes, in sinus rhythm while his chest radiograph showed cardiomegaly. The diagnosis on admission was a right lacunar infarct, with an NIHSS of 26. On the third day of admission, he was found to have a small amount of coffee-ground vomitus. This was managed conservatively by administration of proton-pump inhibitors. His vital signs were stable at this point. On the fifth day of admission, he had dyspnea. The GCS dropped to E4V2M6 with an Sp02 of 90% under room air. He did not have overt signs of heart failure, but the lungs had crepitations in bilateral lower zones. At this point his BP and hemoglobin were stable, and there was no more coffee ground vomitus as seen earlier. He was started on regular IV Frusemide as well as IV Augmentin. By day 8 of admission, he deteriorated further, with his ABG showing Type 1 respiratory failure. ECG had deep T inversions in leads V1-V6, I and aVL. His GCS dropped to 3/15. Maximum medical therapy given, including high-flow mask oxygen 15 liters per minute, as the family had requested DNR.

CASE 4

A 67-year-old Malay man presented with sudden-onset left sided body weakness and slurring of speech about 4.5 hours after his symptoms started. His co-morbids were Type 2 DM, hypertension, and a right foot ulcer one year earlier for which a Ray amputation had been done. His GCS was E4V5M5 with a BP of 164/107mmHg and HR of 72/min. SpO2 was 100% under room air. As for the muscle power, the right side was 3/5 while the left was 0/5. Reflexes were 2+ and the plantars were upgoing bilaterally. His plain CT Brain showed right Sylvian fissure effacement, with no other features of an infarct (Figure 3). The clinical impression was an acute right partial anterior circulation infarct (PACI), NIHSS 16, and he was admitted to the ward. Aspirin had to be withheld the next day, however, as he was noted to have coffee-ground vomitus. By the third day his GCS was E3V5M6, and he was febrile and developed crepitations in the right lung. IV Ceftriaxone 2g od was started. There was no drop in Hb. Aspirin was therefore restarted on the fourth day of admission. The following day, his GCS was E4V2M6 while the BP became high at 174/114mmHg. At this point he was tachycardic, and his Troponin T had increased from 70 to 486. He was started on acute coronary syndrome treatment (Fondaparinux and Clopidogrel) as well as Frusemide. A plan was made for referral to a cardiology center for intervention, while completing 5 days of Fondaparinux. The antibiotic was also switched to IV Tazosin 4.5g QID. Unfortunately, the following day, he developed fast atrial fibrillation (AF) without any symptoms of chest pain or dizziness. A 12-lead ECG showed atrial flutter with ischemia in the lateral leads. This responded to IV Digoxin and he was planned for transfer to a coronary care unit (CCU). He then developed an acute kidney

injury and hypernatremia over the following week, which improved with hydration. An ultrasound of the renal system did not show any obstructive uropathy. Throughout this time his general condition remained the same (GCS E4V4M6) and he remained afebrile. On the seventeenth day of admission, his GCS dropped to 3/15 at midnight, with pupils that were bilaterally sluggish. His BP dropped to 85/65mmHg, requiring IV Noradrenaline. The clinical situation was complicated by a difficult intubation, however, good oxygenation was seen on the blood gases pre-intubation. The ECG did not show new ischemic changes. A repeat CT brain showed a right MCA territory infarct. Aspirin was restarted and the antibiotics were escalated to Meropenem in view of spiking temperatures. His sodium levels improved from 170 to 165 mmol/L. On the twentieth day of admission, he was found to be unresponsive. Pulseless electrical activity (PEA) was initially seen on the cardiac monitor when CPR was commenced, but he developed ventricular fibrillation (VF) 15 minutes later, defibrillated twice. Unfortunately, he was unable to be revived despite 30 minutes of resuscitation.

CASE 5

A 68-year-old Chinese man complained of sudden-onset dizziness and vomiting upon waking up, associated with sweating.
He had a history of hypertension and ischemic stroke five years earlier from which he fully recovered. He was not compliant to Aspirin. He did not have any chest pain or dyspnea. On examination, his GCS was full , with a HR of 66/min and BP of 170/83 mmHg. The SpO2 was 99% and he was afebrile. There were no murmurs, and the lungs were clear. He had a lateral gaze palsy on looking to the right, with horizontal nystagmus. The pupils were 3 mm bilaterally reactive. He was found to have a left upper motor neuron facial nerve palsy. The gag reflex was weak. Power over the right side was 5/5 both upper and lower limbs while on the left side was 4/5 in the upper limb and 5/5 in the lower limb. Reflexes were 2+ and sensation was intact. There was past-pointing and dysdiadochokinesia over the left side, with rebound phenomenon. The clinical impression was a posterior circulation stroke. NIHSS was scored at 10. A plain CT Brain showed a multifocal right frontal and cerebellar infarcts; therefore, the patient was planned for an MRI Brain. ECG showed sinus rhythm with T inversions in leads V4 to V6, and left axis deviation. A repeat ECG showed no evolving changes. Cardiomegaly was seen on the chest Xray. A full blood count showed a white cell count (WCC) of 10, Hb of 17 and platelet (Plt) of 175. He was found unresponsive around midnight on the third day, and was resuscitated unsuccessfully for 40 minutes. Troponin T levels taken before resuscitation 612 µg/L.

CASE 6

A 74-year-old Chinese man with underlying hypertension was brought in for sudden-onset left lower limb weakness, which caused him to fall thrice. There was no slurred speech or facial asymmetry. His GCS was full and vital signs normal. There were no murmurs and the lungs were clear. As a result of the fall, he had bruising over the right supra-orbital region and a wound over his nasal bridge with no active bleed. There was a right racoon eye, swollen upper lip, bruises over the left knee and small wounds over his toes. There was no mastoid hematoma. The visual fields were normal and there was no cranial nerve palsy. Power over the left upper limb was 4/5 except for finger grip which was 5/5. The left lower limb was 3/5. Power over the right side was full, however, there was tenderness over the right shoulder joint. Reflexes were 2+ bilaterally, except for the ankle jerk which was 1+ bilaterally. Plantar responses were flexor and there were no cerebellar signs. A plain CT Brain showed a hypodensity at the

right basal ganglia. ECG showed right bundle branch block while the chest radiograph did not show any cardiomegaly, but a left 3rd rib fracture. A shoulder radiograph revealed a fracture at the neck of the right humerus. He was admitted to the ward with a diagnosis of right lacunar infarct, NIHSS 6. However, on the second day of admission Aspirin was withheld due to bleeding from his external urethral meatus. The bleeding resolved and his GCS and vital signs remained the same. On the seventh day of admission, he suddenly became aphasic (GCS E3V1M6) and had desaturated to an Sp02 of 84%. The BP remained around 130/70mmHg. There was a tachycardia with a HR of 120/min and temperature of 38.3ºC. Lung auscultation revealed reduced breath sounds at the right base with crepitations. ECG showed sinus arrythymia with deep T inversions in leads V1-V3. Troponin T levels were 483µg/L.He was started on IV Tazosin 4.5g QID and intubated. However, he went into asystole three hours later and was unable to be revived despite active resuscitation.

CASE 7

A 46-year-old Chinese man presented with sudden-onset unsteadiness upon waking up. His co-morbids were congestive heart failure (EF33% and a normal angiogram 2 years earlier) and hypertension. He had been on Aspirin and diuretics. He was alert with a BP of 172/119 mmHg and HR of 112/min, in AF. Extraocular movements were full, with horizontal nystagmus on left lateral gaze. There was head titubation, right upper motor neuron (UMN) 7th nerve palsy, with an absent gag reflex, dysarthria, and reeling to the left side. Tone was normal while the power was full bilaterally. Reflexes were not brisk and sensation was intact. Plain CT was normal. ECG showed AF while chest radiograph showed cardiomegaly. He was admitted with a diagnosis of left posterior circulation stroke, NIHSS 10. On the third day of admission he developed a fever, and went into fast AF, which was controlled with Digoxin. The patient remained alert and orientated, but the BP was persistently high at around 201/126mmHg by day 4. The HR was in the range of 140-150 per minute while the Troponin T levels rose from 27 to 81. A repeat plain brain CT did not show any infarct and he was planned for an MRI. By the fifth day, he was febrile, with a HR of 144/min. Continuous cardiac monitoring showed AF while his ECG showed deepening ST depressions from V2 to V6. He was given IV Digoxin and then IV Amiodarone. A few hours later, the HR went up to 220/min while the GCS dropped to 12/15 (E4V3M5). After a second dose of IV Digoxin 0.5mg, he was given synchronized cardioversion. His GCS then dropped to 6/15 (E4V1M1). He was intubated and cardioversion with 200J was given unsuccessfully thrice. The BP then dropped further, requiring inotropes, with a persistently fast HR (around 170/min). Shortly after that, he went into asystole and CPR was commenced, but turned out futile.

CASE 8

A 56-year-old Indian man, a cook, with underlying diabetes was brought in by friends for syncope while cooking at a restaurant. He was reported to have sudden-onset right –sided body weakness. His GCS was E4V1M5 on assessment, with stable vital signs. There were burns over the dorsum of both hands. There was a left gaze preference with a right UMN 7th nerve palsy. The pupils were 3 mm bilaterally reactive and gag reflex was present. Power over the right side was 0/5 over both upper and lower limbs, while on the left the power was 3/5. The reflexes were generally 2+ except for ankle jerks (1+). His CT Brain showed a dense cord sign over the left MCA (Figure 4). NIHSS score was 21 and he was therefore given thrombolysis with IV Actilyse 3hours from onset of symptoms. NIHSS improved to 15 an hour post-thrombolysis. By the second day, the NIHSS had improved to 12, and he was

alert but aphasic. Vital signs were normal. His CTA Brain showed an acute left MCA territory infarct with no evidence of left MCA stenosis. On the fourth day, he suddenly complained of chest pain during morning rounds and was found to be tachypneic and sweaty. His ECG showed ST elevation (Figure 5) in the anteroseptal leads (V1-V4). He was intubated in view of impending cardiorespiratory collapse as his BP started to drop and oxygen saturations reduced to 80-84%. He was referred to the cardiologist for urgent PCI for the anteroseptal MI. However, he went on to develop cardiogenic shock and was unable to maintain his BP despite 3 inotropes. He was in SVT for which synchronized cardioversion was done, and then defibrillated for pulseless VT. He eventually became asytolic but was unable to be revived despite 30 minutes of CPR.

CASE 9

An 80-year-old Indian man came to hospital for slurring of speech for one day and giddiness for two days. He had DM, hypertension and ischemic heart disease for which a coronary artery bypass graft (CABG) was done 22 years earlier. A year before, he underwent stenting to one vessel. His GCS was full, with normal vital signs. He was ataxic and dysarthric, with a right UMN 7th nerve palsy. The gag reflex was present. Power was 4/5 throughout while reflexes were 2+. Sensation was intact and plantars were downgoing bilaterally. A plain CT Brain showed only old cerebellar infarcts. He was admitted for posterior circulation stroke, with an NIHSS of 16. His Hb was 15.3g/dL while his platelets were 156. ECG showed ST depressions in leads V4 to V6, and repeat ECG showed further ST depressions in leads I, aVL, V4 to V6 (Figure 6). On the second day of admission, he complained of sudden onset dyspnoea and chest heaviness. ECG showed left bundle branch block with T inversions in leads V4 to V6. Troponin T had risen from 35 to 809. He was treated as NSTEMI and started on LMW heparin and dual antiplatelet. Four hours later the chest pain had resolved. However, the next day his BP began to drop and he was electively intubated in view of impending collapse. The ECG did not show any evolving changes at this point, but he was persistently tachycardic. Synchronized cardioversion was carried out, bringing down the HR from 177 beats per minute to 156/min. Nevertheless, he succumbed and passed away shortly after.

CASE 10

An 89-year-old Chinese man was brought in by his nephew after he was found to be less responsive at home. He was not moving the right side of his body. He had been living alone and had a background history of ischemic heart disease. He was also on a pacemaker but not on proper follow up. He had DM, hypertension and dyslipidemia. His GCS on arrival was E1V2M5, with a HR of 83/min and BP of 168/56mmHg. Pupils were not equal. Oxygen saturations were normal and his lungs were clear. There was a pansystolic murmur over the left apex. He had a ptosis over the left eye. The tone was increased over the right side. Power over the right upper limb was 0/5 while over the lower limb it was 2/5. power was full on the left side. The reflexes were brisk, and plantars were upgoing on the right side. An urgent plain CT Brain showed hypodensity at the left cerebellum and pontomedullary regions. He was admitted with a clinical diagnosis of Weber's syndrome. ECG showed left bundle branch block with poor R wave progression; pacemaker spikes were seen. By the sixth day of admission, his GCS was E3V1M5. He had been started on IV augmentin in view of cough. He was planned to be discharged once family learnt nursing care and after completing antibiotics. He was on DVT prophylaxis throughout admission. Apart from one episode of fresh blood seen from his mouth, the admission was uneventful until the 22nd

day, when he started to become tachypneic. He had been in the ward awaiting placement by social welfare as his family members could not care for him. His ECG at this point showed new T inversions in leads II,III, avF, and V3 to V6. Troponin T levels were elevated at 721µg/L He was managed conservatively as discussed with his family and no CPR was done when he went into asystole.

DISCUSSION

The relationship between the heart and the brain is divided into the heart's effects on the brain (for example embolic stroke), the brain's effects on the heart (neurogenic heart disease), and neuro-cardiac syndromes such as Friedreich disease.[2] Clinicians are often faced with the conundrum of deciding which is the cause and which is the effect in situations where both neurological and cardiac symptoms are present. Furthermore, many of these patients already have vasculopathy related to diabetes which predisposes them to both cardiac and neurogenic events. The brain and heart crosstalk is being increasingly recognized as a rationale for cardiac complications after ischemic stroke. Neurocardiology is the specialty that focuses on this brain- heart connections.

Proposed mechanisms and pathways for cardiac complications in stroke include the hypothalamic–pituitary–adrenal axis, the immune and inflammatory responses, gut dysbiosis and traditional risk factors primarily involved in the pathogenesis of ischemic stroke.[3] The neurogenic stunned myocardium, although more commonly implicated in subarachnoid hemorrhage than in ischemic stroke, is thought to be due to an increase in sympathetic stimulation of the myocardium after brain injury. This is evidenced by circulating catecholamines that can remain elevated for as long as 10 days after an insult to the brain.[4] The site of ischemic injury also plays a significant role in the type of cardiovascular compromise occurring. BP and HR control are altered by stimulation of the orbital surface of the frontal lobe and cingulate gyrus, whereas ischemic lesions of the insular cortex affect BP control and trigger arrhythmias and autonomic dysfunction.[5] Right-sided stroke is usually associated with more cardiac complications than left-sided stroke. Ischemia of the right insular cortex is associated with higher arterial pressure and norepinephrine levels,[6] while left insular cortex injury can be associated with cardiac dysfunction and myocardial wall motion impairment.[7] The damage to the insular cortex produces the highest incidence of cardiac death when compared to other brain regions.[8]

In a population-based US study that included 980 patients who died after their first ischemic stroke, a cardiac cause could be implicated in 19% of the patients.[9] In another retrospective study using the Ontario Stroke Registry, it was found that between 2.5 and 4 out of 100 patients with an acute ischemic stroke developed cardiac arrest in the hospital. Pre-existing cardiovascular disease, older age, and greater stroke severity are the most common predisposing factors.[10] Prosser et al reports that the cardiac death rate is higher in the first four weeks after stroke (46%), then gradually declines. About 18% of cardiac deaths in stroke patients were preceded by at least one non-fatal serious cardiac adverse event, i.e. ventricular tachycardia, VF, acute myocardial infarction, pulmonary edema and moderate-to-severe cardiac failure. These events peaked between day 2 and day 3 and was a poor overall prognostic indicator. Nearly 80% of these adverse events occurred in patients with a history of ischemic heart disease and/or congestive heart failure.[11]

Beta-blockers, prolonged cardiac monitoring and anticoagulation have been suggested as a means to reduce the cardiac mortality after ischemic stroke, but more research is needed to determine the best way to manage cardiac complications in ischemic stroke. The importance of multidisciplinary management among

neurology, cardiology and intensive care teams is indubitable.

CONCLUSION

Cardiac dysfunction is an important cause of morbidity and mortality among stroke patients. A wide array of cardiac abnormalities may follow the different type of stroke syndromes. Identifying patients at risk and closer cardiac monitoring are vital to reduce the number of cardiac-related deaths. In patients who survive these cardiac complications, early cardiac referral is warranted for secondary prevention of stroke.

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