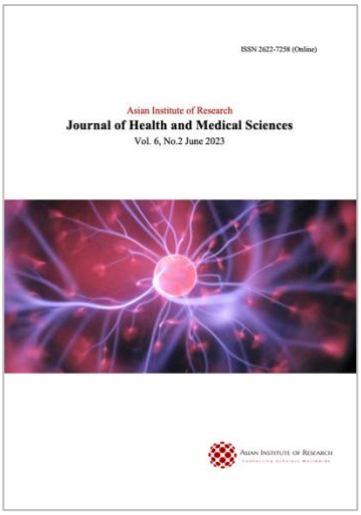

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Bilateral Medial Medullary Stroke: A Single Center Case Series

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Abstract
Introduction. Stroke in the posterior circulation accounts for 20-30% of ischemic strokes (Frid et al., 2019). The medial medullary stroke accounts for less than 1%. It is caused by a lesion in the vertebral artery or its branches, or the anterior spinal artery (Ropper, Samuels, Klein J, & Prasad, 2019). Prognosis depends on the age of the patient and the severity of the motor deficit on admission (Kim & Han, 2019). A bilateral medial medullary infarct is thus very rare. Methodology. We therefore present a case series of three patients in a tertiary hospital in the Philippines and describe their clinicodemographic profile, clinical presentation, imaging and ancillary diagnostic characteristics and outcome. Age range was from 35 to 64 years old. All three presented with dizziness and varying combinations of cranial nerve deficits, motor, sensory ataxia and cerebellar signs. Imaging modalities used are Magnetic Resonance Imaging (MRI) with time of flight (TOF) and computed tomography angiography (CTA). One had a left vertebral artery (VA) occlusion extending into the proximal basilar artery (BA). Another had a non-visualized right VA and bilateral posterior inferior cerebellar artery (PICA). The last case had an unremarkable vessel study. Treatment strategies include dual antiplatelet therapy (DAPT) with Aspirin and Cilostazol (2 of 3), and Enoxaparin plus Aspirin. One received intravenous thrombolysis with Alteplase prior to the DAPT. None were intubated and all were home discharged. Conclusion. We have shown that a bilateral medial medullary stroke can present with minimal disability and a good outcome.

Keywords: Stroke Syndromes, Bilateral Medial Medullary Infarct, Rare, Good Outcome

1. Introduction

Stroke is a devastating illness and the second leading cause of death worldwide. A bilateral medial medullary infarct is a rare occurrence. From case reports, we know that the medial medullary infarct is less than 1% of all stroke syndromes. In the reports made by separate studies of Hu, Nie, Bai, and Liang (2022) and Pongmoragot, Parthasarathy, Selchen, and Saposnik (2013) males are more commonly affected than females (74%) at a mean age of around 60 years old. Based on the structures in the medulla that are affected, a lesion in the bilateral medial area can present as quadriplegia, dysarthria, loss of vibratory and position sense, and tongue paralysis (Ropper et al., 2019). It may not always present with the complete syndrome.

A medial medullary infarct is caused by an occlusion of the intracranial portion of the vertebral artery extending into the orifice of the anterior spinal artery branch. Around 10% of these occurs as a bilateral lesion (Caplan & Van Gijn, 2012). With the use of advanced imaging techniques such as an MRI with a vessel study, appreciation of the lesion location has now become possible although sometimes can still be missed. The common descriptions used for this syndrome are: the heart shape lesion, V shape lesion, and recently, the airpod sign.

Depending on the extent of lesion, patients can have difficulty breathing, swallowing and most likely be intubated or a feeding tube inserted. Case reports and recent studies generally agree that it carries a worse prognosis. The study of Hu et al., (2022) reported a poor prognosis in 93.3% of the 15 cases reviewed. In another study of 38 patients, mortality was at 23.8% (Pongmoragot et al., 2013). We therefore present the following cases seen last year in our institution documented to have the appearance of a bilateral medial medullary infarct on MRI and discuss their clinic-demographic profile, stroke presentation clinically and radiographically, management and outcomes.

2. Case 1

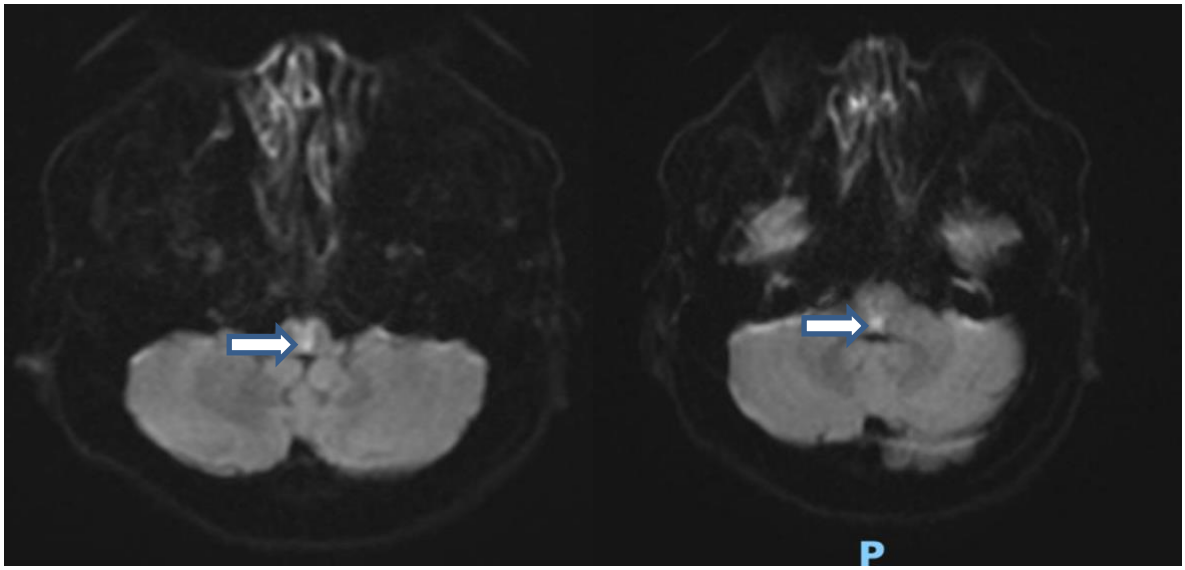

Figure 1.1: A V-shaped hyperintense lesion on diffusion weighted imaging (DWI) in the bilateral medial medulla extending up to the dorsal aspect of the caudal pons

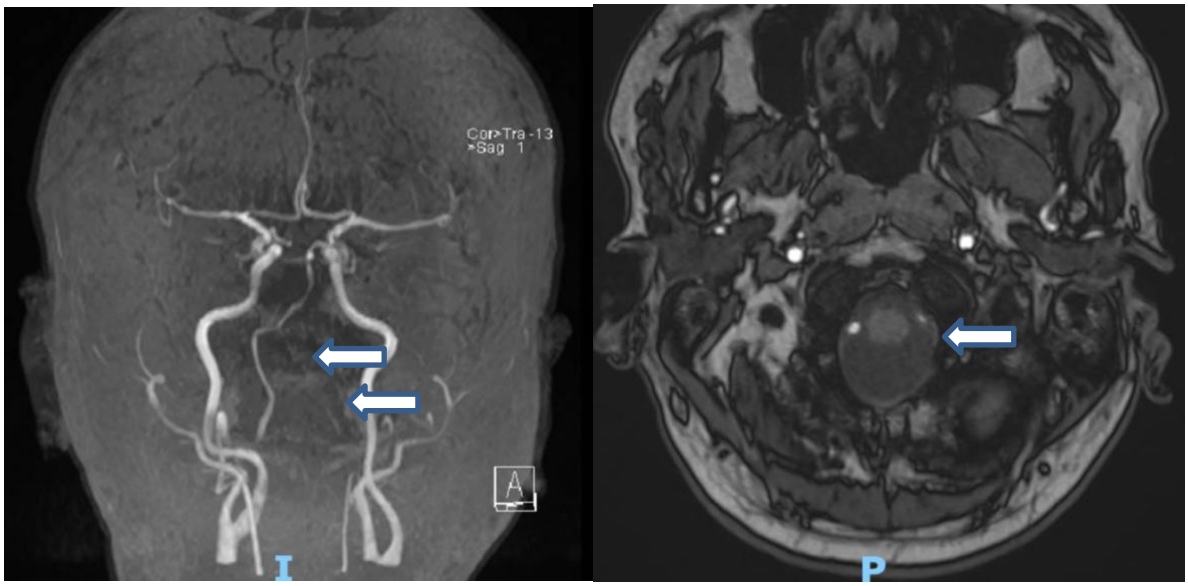

Figure 1.2: A time of flight MRA showing stenosis in the left vertebral artery (white arrows), coronal plane (left), axial plane (right)

A 48-year-old male, known hypertensive, non-smoker with occasional alcohol intake presented 12 hours prior to admission with dizziness and dysphagia. On examination, he had torsional nystagmus, contralateral central facial paralysis, weak gag reflex ipsilaterally, tongue deviation ipsilaterally, motor strength of 4/5 in the left extremities, tremors, ipsilateral dysmetria; and impaired joint and position sense bilaterally. MRI revealed the characteristic heart-shaped sign of hyperintensity on DWI and a corresponding drop on apparent diffusion coefficient (ADC) indicating an acute stroke. Time of flight images revealed stenosis of the left vertebral artery. He was managed with Enoxaparin 1mg/kg subcutaneously every 12 hours for 7 days and Cilostazol 100mg tablet twice a day. On the second hospital day, the patient became dysarthric and noted to have bilateral lateral rectus palsy thus Aspirin 80mg tablet once daily was added to the regimen. He was eventually discharged after 14 days of hospitalization with residual deficits of resolving central facial paralysis, dysarthria, weak gag and tongue, motor strength of 4/5 in all extremities, and impaired vibratory and position sense. A nasogastric tube was kept in place.

3. Case 2

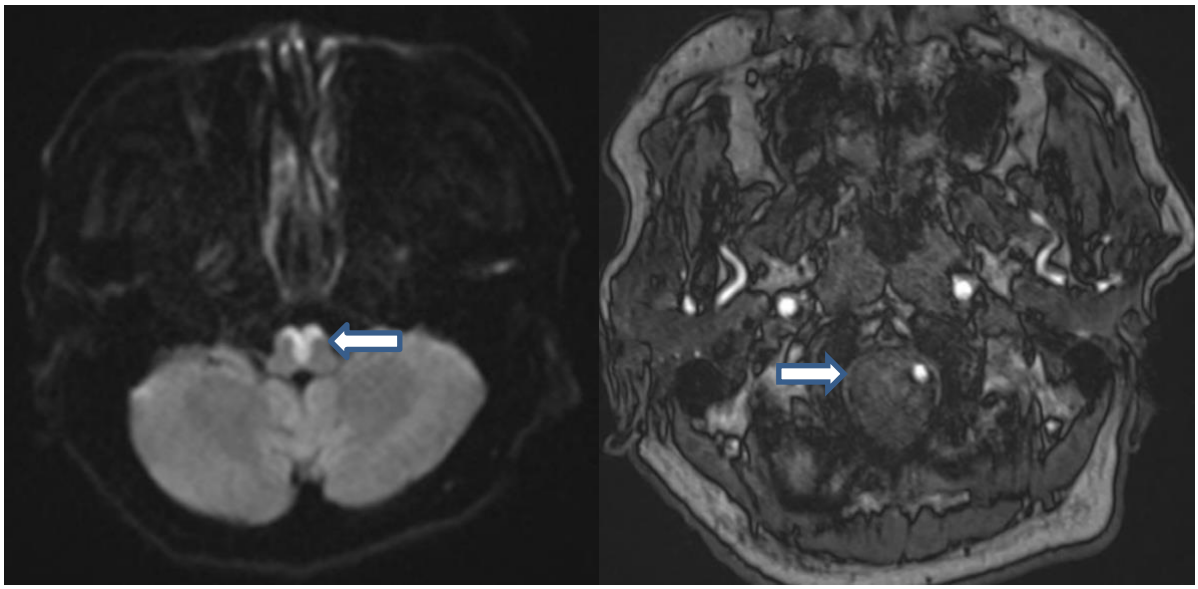

Figure 2.1: A heart-shaped hyperintense lesion on DWI affecting the bilateral medial medulla (white arrow). The time of flight image on the right shows a non visualized right vertebral artery (white arrow).

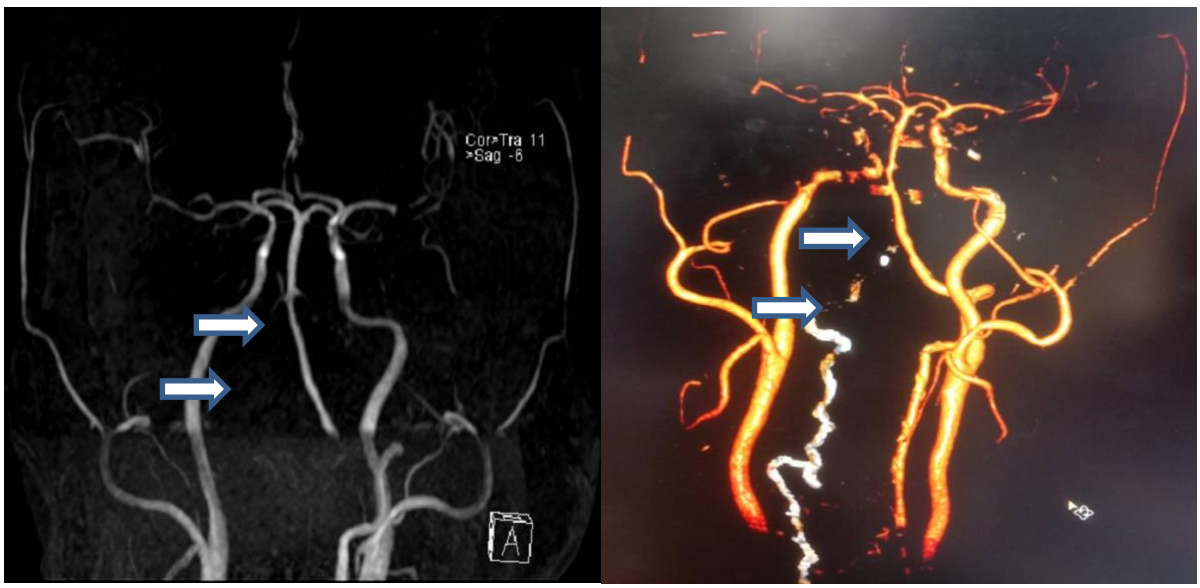

Figure 2.2: The non-visualized right vertebral artery on the right is more evident here in this reconstructed time of flight image on the left (white arrows). On the right is the CT angiogram study of the head and neck of the

same patient. There was non-opacification of the right vertebral artery after it takes off from the right subclavian artery up to the intracranial segment just before it joins the left vertebral artery (white arrows).

A 64-year-old male, hypertensive, 30-pack year smoker who binge drank just prior to admission came in with a chief complaint of right-sided weakness 4 hours prior. He was complaining of a bifrontal headache, moderate with episodes of vomiting prior to the weakness and dizziness. On assessment he only had motor symptoms such as central facial palsy on the right, dysarthria, motor strength of 2/5 in the right upper extremity and 4/5 in the lower extremity, hyperreflexia on the right and Babinski, right. He was within the window for thrombolysis thus a cranial CT scan was done which did not show any acute infarct or hemorrhage. He was given 0.6mg/kg of Alteplase. Less than 24 hours post thrombolysis, there was noted worsening of the right leg weakness to 1/5 and a new leg weakness on the left, 3/5 with hyperreflexia and bilateral Babinski signs. There was new onset sensory deficit to pain and temperature on the right extremities. MRI cranial stroke protocol done showed a bilateral medial medullary infarct with flow void in the right vertebral artery probably secondary to severe stenosis. Aspirin and Cilostazol were started. CTA done showed long stenosis of the right vertebral artery from the aorta. He was eventually discharged home after 12 days with residual deficits of resolving central facial paralysis, motor strength of 0/5 in right extremities, 4/5 in the left upper extremity, 3/5 in the left lower extremity, impaired vibratory and position sense, hyperreflexia and bilateral Babinski. He was discharged without needing a feeding tube.

4. Case 3

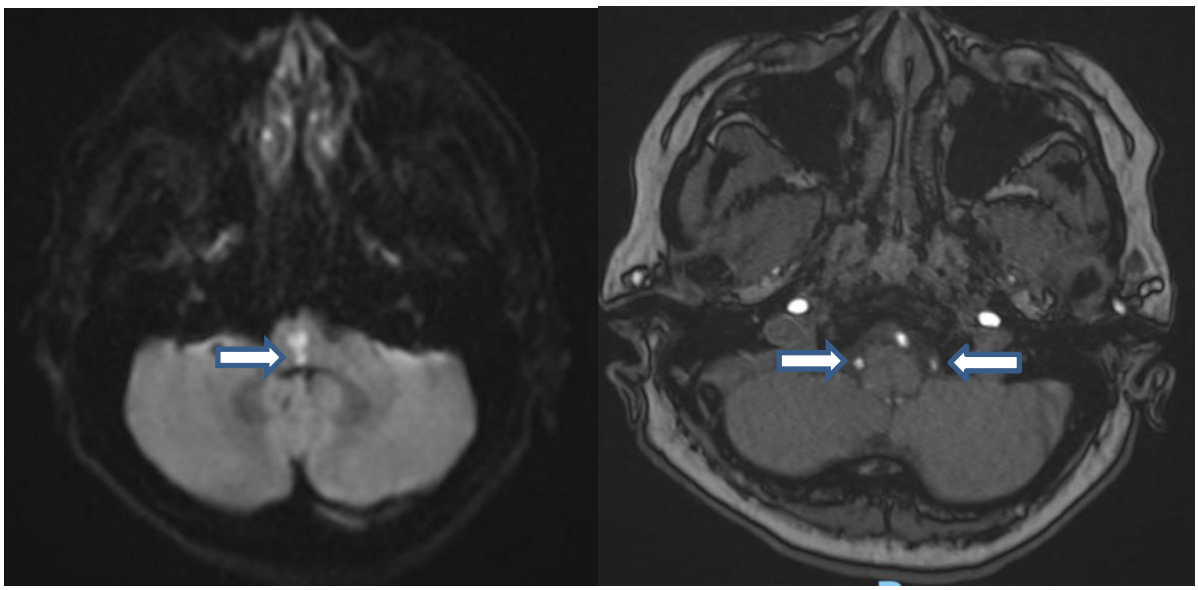

Figure 3.1: A key-like hyperintense lesion in the medial bilateral medulla (white arrow) can be seen on this DWI sequence on the left. On the right, both the vertebral arteries are visible and almost the same in caliber in this cut.

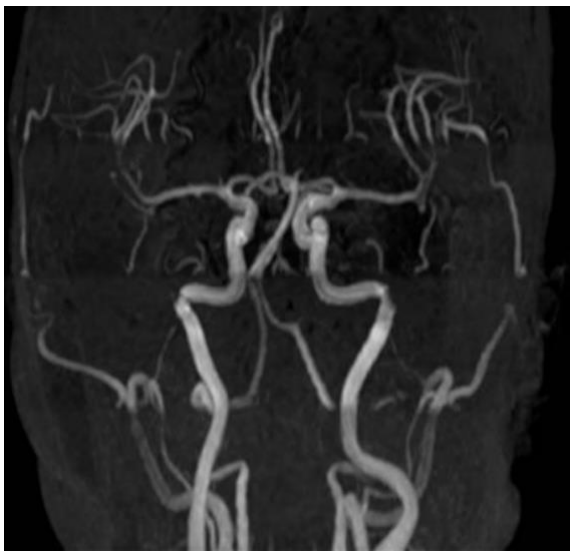

Figure 3.2: The time of flight image did not show any focal narrowing or flow void.

A 35-year-old female, hypertensive, with chronic kidney disease on maintenance hemodialysis, non-smoker, non-alcoholic came in with dizziness and a sense of imbalance when ambulating one day prior to admission. It was associated with binocular blurring of vision. On the 7th day of hospital stay she was referred due to persistence of dizziness, vertiginous in character, had bidirectional nystagmus with horizontal and vertical component, truncal ataxia veering to the right and dysmetria on the right. MRI showed an isolated lesion in the bilateral medial medulla and an unremarkable time of flight vessel study. She was started on Aspirin and Cilostazol and was discharged home on the 9th day without progression of deficits.

5. Discussion

As mentioned earlier this rare presentation has an average target population of 60 years of age and usually affects males which is also the demographic profile of stroke cases in general. In our series, the two patients belong in the younger population with one patient considered a stroke in the young. Two out of three were males. The common risk factor among these three was hypertension. Two others were newly diagnosed with uncontrolled diabetes mellitus during the workup. A summary of the clinicodemographic profile of these three cases is appended in table format below.

As to the presentation, dizziness was the first symptom to appear in all cases followed most commonly by motor symptoms/signs (2 of 3) such as central facial paralysis, dysarthria, hemiparesis; and cerebellar symptoms (2 of 3) such as nystagmus, dysmetria; and impaired joint position sense (2 out of 3). Only one patient presented with dysphagia and had to be fed via a nasogastric tube. The neurologic deficits matched the extent of infarction as seen on imaging judging by the size and shape of the lesions. Although we note here that stroke lesions on imaging appeared almost symmetrical despite findings of asymmetric neurologic signs on neurologic examination. In the first case, we see a heart-shaped lesion spanning the area of the dorsal to ventral bilateral medial medulla and reflected on the presenting symptom and the progression of deficits. The admitting complaint of dizziness with examination findings of ipsilateral tongue deviation, nystagmus, and limb ataxia pointed to involvement of the dorsal medullary structures starting from the CN XII nuclei and medial longitudinal fasciculus to the impaired joint position sense as the lesion spreads to include the medial lemniscus and moving forward to the ventral aspect to the medullary pyramids which caused the hemiparesis. The lesion then moves superiorly to the caudal pons and thus the patient presented with a bilateral lateral rectus palsy at the wards. The second case showed the same heart-shaped lesion advancing more ventrally into the medullary pyramids thus we see here a more severe motor deficit syndrome. The last case was a key-like lesion dorsal than ventral in location thus the presentation was more of dizziness, sense of imbalance and limb ataxia. This lesion location and corresponding deficit confirms the findings of Kim and Han (2009) in which they mapped the medulla and found that motor dysfunction was more common

in ventral lesions, sensory in the middle and vertigo and dizziness in the dorsal parts. The structures that control respiration are located more laterally hence none of our cases required airway support in any form.

The stroke mechanisms in our series showed severe stenosis of either vertebral artery with the occlusion extending into the proximal basilar artery and a probable small vessel etiology in the last case. Larger studies also showed large vessel atherosclerosis as the most common mechanism and that an atheromatous branch occlusion is more common in a bilateral lesion (Kim & Han, 2019; Hu et al., 2022; Pongmoragot et al., 2013).

Despite one case being given a low molecular weight heparin initially and another receiving reperfusion therapy with Alteplase, all three cases received a dual antiplatelet combination of Cilostazol and Aspirin. Other supportive therapies used were statin, permissive hypertension during the acute stroke phase and intravenous hydration with normal saline. Although available case reports vary in their secondary stroke prevention strategies depending on the possible stroke mechanism, using a dual antiplatelet regimen in this specific stroke was not commonly described.

As to the outcome, these patients were home discharged without further neurologic deterioration after staying 9 to 14 days in the hospital. This is in contrast to the case reports and larger reviews on similar cases which reported a poor prognosis requiring intubation and subsequent mechanical ventilatory support in some. Acute respiratory failure was also not a complication in this series. It is also worth noting that despite a very elevated glycosylated hemoglobin values in two of our patients, they did not suffer any more complications than the third patient who had no diabetes. This was also shown in the study by Fri et al. (2019) wherein diabetes is strongly associated with a posterior circulation stroke than anterior.

6. Conclusions

Our study has shown that a bilateral medial medullary infarct as a rare stroke syndrome can affect any age including the younger population. It can present with a combination of cranial nerve deficits, motor, sensory and cerebellar symptoms and may lead to difficulty swallowing depending on infarct size and extension into surrounding structures. An MRI and a vessel study like CTA and magnetic resonance angiography (MRA) help in confirming the lesion location and identify the site of possible occlusion. A combination of dual antiplatelet therapy seemed to confer a good secondary stroke prevention strategy in a bilateral medial medulla infarction. Despite its known severity in literature, our data showed that some people could have minimal disability and can be discharged with good outcome.

Table 1: Clinicodemographic profile

	CASE 1	**CASE 2**	**CASE 3**
Age (years)	48	64	35
Sex	M	M	F
Smoking history	No	Yes	No
Alcoholic beverage drinking history	Yes	Yes	No
COMORBIDITIES			
Hypertension	Yes	Yes	Yes
Diabetes	No	No	No
Cardiac pathology	No	No	No
Chronic Kidney Disease	No	No	Yes
COVID vaccination status	Fully vaccinated	Vaccinated x 1 dose	Unvaccinated
LENGTH OF HOSPITAL STAY (days)	14	12	9

Table 2: Ancillary Tests

DIAGNOSTICS	**CASE 1**	**CASE 2**	**CASE 3**
ECG 12L	Sinus tachycardia	Sinus rhythm Inferolateral wall ischemia	Sinus rhythm
CHEST XRAY	Unremarkable	Pneumonia, bilateral Atherosclerotic aorta	Cardiomegaly
COVID RT-PCR ASSAY	Negative	Negative	Negative
HbA1c (%)	13	13.6	-
FBS (mg/dL)	309.19	212.07	86.31
LDL (mg/dL)	298.84	125.48	155.21
TRIGLYCERIDES (mg/dL)	175.22	114.16	225.66
TOTAL CHOLESTEROL (mg/dL)	303.47	169.11	223. 55
SERUM CREATININE (mg/dL)	1.20	0.83	9.05
ALT (U/L)	19.23	30.61	9.45
AST (U/L)	18.7	23.58	20.96

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