

Isolated Leg Monoparesis in a Patient with Atrial Fibrillation and Acute Ischemic Small Vessel Disease

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

ABSTRACT

Monoparesis is most commonly caused by dysfunction of the lower motor neurons (LMNs) which innervates the affected limb. This includes lesion affecting the anterior nuclei, ventral nerve roots, lumbosacral plexus, or peripheral nerve. Nevertheless, it can also be due to upper motor neuron lesion, typically seen in lesions of the spinal cord. In general, many conditions that cause hemiplegia, paraplegia or quadriplegia may begin as monoplegia. We illustrate an elderly patient with hypertension, diabetes mellitus, dyslipidemia and atrial fibrillation, who presented with acute monoparesis and radiological evidence suggestive of recent infarct and small vessel disease.

Keywords: *Acute monoparesis; hypertension; diabetes mellitus; dyslipidemia; recent infarct; atrial fibrillation; small vessel disease.*

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1. INTRODUCTION

Monoplegia refers to weakness of one limb (either arm or leg). The likely cause of leg monoparesis includes frontal lobe hemorrhage, white matter lacunar infarct, brainstem or spinal cord lesion, and spinal or peripheral nerve pathology. Cerebrovascular accident is a common condition with patients usually present acutely with hemiparesis, and rarely with isolated monoparesis [1]. The prevalence of monoparesis in cerebrovascular accident (acute stroke) reported in different studies varies from 2% to 13% [2]. 4.1% of 4802 patients in a 21-year Lausanne Stroke registry presented with monoparesis, of which 63 % was seen in arm, 22% in face, and 15% in leg [2]. In this report, we describe an elderly lady who presented with left lower limb monoparesis, as first clinical manifestation of acute cerebrovascular accident (stroke).

2. CASE HISTORY

A 65-year-old female, non-smoker, with background history of hypertension for 10 years and Diabetes Mellitus for 5 years, presented to the Accident and Emergency Department with first episode of acute left lower limb weakness and numbness lasting for a few hours. This was preceded by 2-days-history of similar but milder symptoms. The strength of her left lower limb partially improved within 12 hours of the onset of the symptoms. There was no associated left upper limb weakness, or right sided body weakness. She had no drooling of saliva, slurring of speech, headache, loss of consciousness, seizure, vertigo, diplopia, dysphagia, dysarthria, backache, urinary or bowel incontinence. She was on Glucophage 500 mg BD (twice a day), Amlodipine 10 mg OD (daily), Metoprolol 200 mg BD, Hydrochlorothiazide 50 mg OD, Simvastatin 10 mg OD, Slow K 600 mg OD, Prazosin 1 mg BD, and Losartan 50 mg OD for the past 5 years.

She was admitted to the hospital on the same day. On physical examination, she was afebrile, alert and conscious. Blood pressure was 161/87mmHg. Her BP remained to be stable, fluctuating between 142-161/70-87 mmHg whilst in the ward. There was atrial fibrillation with apical heart rate of 80beats/minute. Neurological examination revealed a hypotonic left lower limb, with no muscle wasting. The power over the left hip flexor and knee flexor was 3/5. However, there was 0 power on left knee extension, ankle flexion and extension, with complete absence of

movement of the left foot. Deep tendon reflexes of all four limbs were normal. Left plantar response was equivocal, though normal on the right. There was mild reduction to crude touch and pin-prick sensation over the whole left lower limb. Vibration and proprioception sense was intact. Neurological examination of the left upper limb, right upper and lower limbs, cranial nerves, and cerebellum were normal. Speech assessment, visual acuity and visual field assessment were normal. Visuospatial constructional apraxia was noted as the patient was unable to copy the interlocking hexagon, as shown in Fig. 1. There was no tenderness noted over the lumbar spine area. Cardiovascular, respiratory and abdominal examination were otherwise normal.

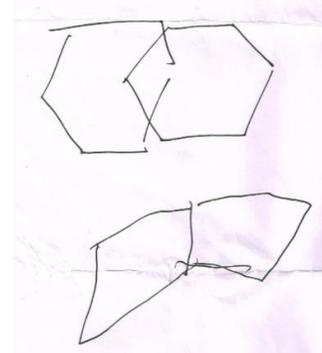


Fig. 1. Drawing showing patient was unable to copy interlocking hexagon

Electrocardiography (ECG) result showed that the patient had atrial fibrillation (Fig. 2), with no acute ischemic changes. Computed Tomography (CT) of the brain revealed a well-defined hypodense lesion in the right occipital region in keeping with old infarct (Fig. 3a). There was cerebral atrophy, with multifocal old (anterior limb of right internal capsule) and recent (right thalamus and right lentiform nucleus) infarcts, and microvascular ischemia of the deeper white matter (Fig. 3b). Prothrombin Time (PT) was 10.8 s, International Normalised Ratio (INR) was 0.97. Other results were Hemoglobin (Hb) 15.1 g / dl, white blood cell count (WBC) $8.36 \times 10^9 / l$, platelets $351 \times 10^9 / l$, packed cell volume (PCV) 43.8%, urea 4.1 mmol / l, sodium 134 mmol / l, potassium 3.9 mmol / l, creatinine 51 $\mu\text{mol} / l$, and random blood glucose 9.4 mmol / l.

She was treated for acute right cerebrovascular accident and subsequently referred for physiotherapy as well as occupational therapy. She was discharged three days after admission

with Rivaroxaban 20 mg ON (every night), Atorvastatin 40 mg ON, metoprolol 50 mg BD, Cardiprin 100 mg OD (once daily), Glucophage 500 mg BD (twice per day), Slow K 600 mg OD,

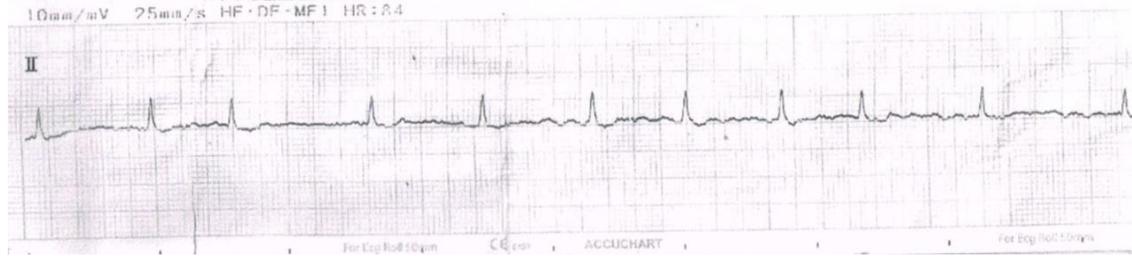


Fig. 2. ECG showing atrial fibrillation



Fig. 3(a). CT brain of an old infarct in right occipital lobe (white arrow)



Fig. 3(b). CT brain of recent infarcts in right lentiform nucleus (black arrow) and right thalamus (white arrowhead), with generalized cerebral atrophy and periventricular lucency

3. DISCUSSION

Isolated monoparesis is rarely seen in patients with stroke [1,2], in which only 2.5% of stroke patients were found to present with such symptoms. Study has shown that majority of monoparetic patients has brachial monoparesis, whilst crural (leg) monoparesis is present in 33% [1]. This patient has a sudden onset of weakness, affecting the distal limb more severely. This presentation is atypical for a patient with upper motor neuron lesion with CT evidence of recent cerebral infarct, as it was generally perceived that a more severe distal weakness is suggestive of a peripheral nerve lesion (a lower motor neuron lesion) [3]. Studies did not show consistent pattern of muscle weakness, as regards to proximal-to-distal gradient, or greater extensor weakness [4]. Thus, a more distal limb weakness might not necessarily mandate a diagnosis of lower motor neuron lesion. Additionally, there was no typical sensory deficit, other lower limb or cranial nerve abnormalities, urinary or bowel incontinence to suggest brainstem, spinal cord, or peripheral nerve pathology.

Pathologically, studies have shown that 76.5% of monoparesis was due to ischaemic strokes, and 25% was as a consequence of small cerebral haemorrhages. In cases with ischemic stroke, small artery disease was the cause of the presentation in 39.2%, cardio-embolism in 15.7%, and atherosclerosis in 9.8% [1]. Previous reports have suggested that the lesions most likely to produce pure motor monoparesis (PMM) are those in the cortical or near-cortical area. This is because somatotopic motor representation is most widely separated at this region. However, recent reports show that only 48% of patients with monoparesis, had a cortical lesion. Meanwhile, 31% of them had lesions in the subcortical area and 8% were noted to have a brainstem lesion [5]. Other study has shown that 25% of patients with leg monoparesis have an anterior cerebral artery occlusion, causing the infarct of the medial aspect of the precentral gyrus [1,6]. The CT scan of the brain in our patient shows no acute lesions in the anterior cerebral artery (ACA) territory. Nevertheless, it is to be noted that CT brain is normal in about 40% of the patients with monoparesis following stroke. This is especially true in the case of pure motor monoparesis confined to lower limb because the lesion may be located in the top of the frontal lobe cortex, an area that can be easily missed by routine scans [5]. More often, patients with leg

monoparesis have lesions in the contralateral corona radiata or internal capsule, anterior choroidal artery or perforators (30%), or in the brainstem (25%) [5]. Leg monoparesis caused by cerebral infarction could be associated with temporary sensory deficits limited to one extremity [7], as noted in this patient. It may occur among 14-42% of patients with PMM (Pure Motor Paresis) due to extension of the cerebral oedema along the central sulcus to postcentral gyrus [5]. However, this patient exhibited no changes in personality, judgment, urinary incontinence, and primitive reflexes, to suggest a pre-frontal lesion, as typically observed in ACA(Anterior Cerebral Artery) infarct [8].

Lacunar infarcts are small infarcts (2-20 mm in diameter) commonly affect the deep cerebral white matter, basal ganglia, or pons, due to occlusion of a single small perforating artery which supplies the subcortical areas of the brain. Lacunar stroke is responsible for a quarter of all ischemic strokes (25%) [9]. This patient's CT brain suggests a recent lacunar infarct, affecting the right lentiform (basal ganglia) nucleus and thalamic nucleus. She also had multiple old and recent infarcts. She had no known clinical stroke syndrome in the past. This is typically seen in lacunas. It is frequently observed coincidentally on imaging in older people, often not clearly associated with discrete neurological symptoms [10]. Silent, unrecognised lacunar infarcts are at least 5 times more common than symptomatic infarcts. Both types are part of a broader spectrum of cerebral small-vessel disease which also includes vascular white matter disease [11]. Lacunes located more posteriorly in the internal capsule are more likely to produce a greater deficit in the leg than in the arm. This includes motor deficit, or sensory deficit [12]. The internal capsule area is supplied by lenticulostriate arteries, which are terminal arterioles, commonly occluded by cardiac thromboemboli from atrial fibrillation [10,12]. This patient had atrial fibrillation, and there might be thromboemboli to the internal capsule area to explain her sudden leg monoparesis. Nevertheless, the patient's CT brain shows no obvious acute infarct detected over the posterior limb of the right internal capsule to explain the monoparesis in this patient. On the other hand, this patient's initial symptoms started 2 days prior to the acute presentation of the monoparesis, This is more consistent with a thrombotic stroke [13], occurring in the small vessel of the deeper brain. In contrast, the clinical features suggestive of cardio-embolic stroke includes sudden onset to

maximal neurological deficit and decreased level of consciousness at onset. Cardio-embolic stroke is less commonly seen in lacunar infarct, especially when it is multiple [14]. In one recent studies, only 25% of patients with clinical radiologically defined lacunes had a potential cardiac embolic cause for their strokes [15]. Furthermore, lacunar infarcts due to emboli or middle cerebral artery stenosis are recognised by being larger than non-embolic / stenotic lacunes [9]. As the recent infarct seen in the CT brain was of rather minute size, thus, it is more consistent with a non-embolic nature of small vessel disease of the brain.

Her other multiple subcortical lacunes and white matter lesions (WMLs) is possibly the result of cerebral small-vessel disease (SVDs), arise from progressive ischemia secondary to stenosed deep perforating arterioles. SVDs is largely a condition with diffuse endothelial failure with thrombotic tendency [9]. It is commonly seen in hypertensive patient. It causes cognitive impairment and increases future stroke risk [10], as is seen in this patient who was hypertensive. Lacunar ischemic stroke also appears to be more closely associated with white matter lesions than cortical ischaemic stroke [9]. White matter lesions are abnormal areas of hypodensity (on CT scans) in the deep hemispheric periventricular white matter, as observed in this patient. They are in turn associated with cognitive decline, and increased risk of future stroke, particularly lacunar type. White matter lesions also progress rapidly after lacunar stroke, in which new silent lacunar infarcts" occur on follow-up imaging. . This is clearly exhibited in this patient who had multiple old and recent lacunar infarcts, with white matter disease and cerebral atrophy. This patient has SVDs, as shown radiologically, with periventricular hypodensity (Fig. 3b), and has features suggestive of constructional apraxia (Fig. 1), to suggest cognitive impairment. Vascular cognitive impairment (VCI) comprises of all states of cognitive dysfunction syndrome related to cerebrovascular disease [16], in which SVDs is the leading cause [16]. As compared to Alzheimer's Disease with more pronounced episodic memory deficits, VCI related to SVDs usually presents more insidiously, with early executive dysfunction (i.e.in performing daily activities), psychomotor slowness, impaired capacity to use complex information, to exercise emotional and behavioural self-control, as well as depression [17]. Construction apraxia includes clock-drawing test, copy simple diagrams, or

construct simple figures. It is reflective of visuospatial, constructional, and executive dysfunction, which is an early feature of VCI [18]. Constructional apraxia, as seen in this patient, predicts decline in patient's Mini Mental State Examination, in 4 years [19]. Apart from the constructional apraxia, consistent with a diagnosis of VCI, this patient exhibits no memory deficit and is able to recall the events leading to the admission.

Evidence shows that there is no association between atrial fibrillation and white matter disease [17]. Thus, anticoagulating a patient with atrial fibrillation, i.e. using Novel Oral Anticoagulant (NOAC) Rivaroxaban as in this case, may not prevent the recurrence of lacunar stroke, though it might reduce the risk of thromboemboli to larger artery which causes larger infarct, i.e cortical infarct specifically. A systematic review of risk factor opined that atrial fibrillation was more associated with non-lacunar infarction [9]. Only 10% of patients with lacunar stroke have atrial fibrillation [20]. No Echocardiography was done however in this patient. The absence of cardiac source of thrombo-emboli was not verified in this patient, at time of discharge. On the other hand, antiplatelet monotherapy, is beneficial for secondary stroke prevention in patients with lacunar stroke or small vessel disease. There is 22% relative risk reduction in recurrent stroke by single antiplatelet therapy [21] in such patients as compared to placebo. There is no clear benefit with dual antiplatelet therapy. This patient is started on aspirin, upon diagnosis of the cerebrovascular event, and thus would reduce stroke risk due to thrombosis of larger atherosclerotic vessels, and small vessel disease. The management of vascular cognitive impairment (VCI) should also include life-style modification and satisfactory control of hypertension, diabetes mellitus, and dyslipidemia [17]. Nevertheless, the combined treatment of single antiplatelet and anticoagulant increases the risk of clinically relevant bleeding by 50% according to some studies [22]. Thus, the risk-benefits of such combination therapy need to be reassessed. In the advent of continuous use, the patient needs to be vigilantly monitored for bleeding. Other treatment options for small vessel disease includes phosphodiesterase inhibitors, i.e. cilostazol, which needs further studies to ascertain its clinical efficacy [21].

Occipital stroke is a consequence of posterior circulation stroke. This is seen in about 20% of

patients with cerebral ischemia [23]. In typical occipital involvement affecting the primary or secondary visual cortex, patients might have occipital blindness, with macular sparing, impaired visual interpretation, or hemianopia. This may cause difficulty to perform daily routines, such as cooking or getting dressed. In general, occipital stroke does not cause other motor, sensory, or coordination (non-visual) disabilities, unless there is associated infarct in other area of the brain. The neurological deficits may be absent or subtle, leading to difficulty in diagnosing posterior circulation strokes [24]. Old right occipital infarct was noted in this patient. Patient was functioning normally in the community, prior to this presentation of monoparesis. Therefore, this patient is most likely to have an old silent occipital infarct, without clinical syndrome of stroke. A systematic review showed that patients with atrial fibrillation who present with symptomatic cerebral ischemia have had previous asymptomatic cerebral infarction, significantly more frequently than persons in sinus rhythm, as observed in this patient. A preliminary report from Petersen et al. [25] examining asymptomatic patients with atrial fibrillation, records a striking incidence of silent cerebral infarction. The silent occipital infarct possibly originates from cardiac thromboembolism, or it might be due to large vessel atherosclerosis, which is more prevalent in patients with atrial fibrillation. Large-artery atherosclerosis is twice as common in patients with atrial fibrillation than those without [20].

In cases with pure monoparesis, only 17% of the patients had more than 1 cerebrovascular risk factor, as compared with 26% of those with hemiparesis [2]. Hypertension is the only frequent risk factor for monoparesis (53%) [2]. This patient's acute neurological presentation is associated with a rather elevated BP of 161/87 mmHg. However, the acute cerebrovascular event was not associated with hypertensive emergency, which is a condition with patient presenting with BP more than 180/120 mmHg, associated with end-organ-damage, primarily affecting the brains, hearts, and kidneys [8].

Hypotonia tone, as seen in this patient, is a rather common feature noted in acute stroke. Upper motor neuron dysfunction can decrease tone and reflexes if motor paralysis is sudden and severe, or if the lesion damages the motor cortex of the precentral gyrus and not nearby motor association areas [8]. There is no set amount of time that the flaccid stage remains.

For some it can resolve in days, others weeks or months [26]. Spastic leg weakness may occur after several weeks.

The plantar response in the affected limb, was equivocal in this patient. She had normal down going plantar response on the right. Extensor plantar response (EPR), or Babinski signs, is a clinical feature in which there is upward movement of the great toe with fanning of the lateral four toes upon firm stimulation of the lateral border of the sole. It indicates upper motor neuron dysfunction [27]. It is termed "equivocal" when the response to plantar stimulation is weak or absent, as shown in this patient [28]. The EPR had low sensitivity (50.8%), but high specificity (99%) in identifying pyramidal tract disease [29]. Hence, an equivocal EPR does not necessary rule out an upper motor-neuron corticospinal tract lesion. A study has shown as high as 18.6% of normal controls had EPR. However, the physiological extensor response is characteristically different from pathological response, whereby the pathological EPR is more reproducible, sensitive to stimulation (with response induced with early stimulation at the lateral sole) and sustainable (with extension of big toe throughout stimulation), as compared to physiological EPR [30].

The functional outcome of monoparesis was generally promising, compare to more extensive motor deficit, with a spontaneous favorable evolution. This allows the patient to return to former activities (41%) or to live in a slight handicap requiring some assistance (41%) [2].

4. CONCLUSION

Monoparesis of acute onset should always include the differential diagnosis of cerebrovascular accident, especially when the presentation is of acute onset. Though these are rare and easily misdiagnosed, a high index of suspicion and assessment of risk factors is required to diagnose the stroke presentation. In the vast majority of cases (>95%), monoparesis corresponded to ischemic stroke with a favorable outcome. More initiative and studies should be done to identify the risk factors to small vessel disease, and thus serve to prevent its occurrence. Anticoagulating patients with atrial fibrillation might not prevent further deterioration of small vessel disease. Yet, it signifies more extensive atherosclerosis in larger vessels, and increased risk of larger cortical infarct, and thus needs to be managed optimally.

CONSENT AND ETHICAL APPROVAL

As per international standard or university standard guideline participant consent and ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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