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Long-Term Follow-Up of Actilyse-Thrombolysis for a Disabling Acute Ischemic Pontine Stroke

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Abstract

Background: The current thrombolysis guidelines allow only a small number of acute ischemic stroke (AIS) patients to be treated with Recombinant Tissue Plasminogen Activator (rtPA) known as Alteplase under the kind of conditions prevailing in clinical trials (imaging, timing, experience and hesitancy). The Case: A 58-year-old male doctor, with mild diabetes mellitus, presented with sudden left-sided hemiplegia as well as right trigeminal, abducens, facial and vestibular palsy indicating an acute pontine infarct at 12 based on the National Institute of Health Stroke Scale (NIHSS) and +4 by modified Rankin score (mRs). Since he presented within 3 hours of the onset of symptoms and the initial brain CT did not show an intracranial bleed, he was treated with rtPA followed by unfractionated heparin for 24 hours then Acetylsalicylic acid and Clopidogrel for 1 year then Acetylsalicylic acid alone. By 12 hours, he had resumed his motor power fully (mRs +2) and by 72 hours; he had only left-sided numbness (mRs +1). At that time, CT confirmed right posterolateral non-hemorrhagic pontine infarct. MRI angiogram by day 7; showed 0.6 × 1 cm subacute non-hemorrhagic pontine infarct and normal extra- and intracranial vascular disease. Moreover, tests for hypercoagulable and autoimmune disorders were negative. The patient was able to resume his active work, 1-week post-AIS, and remained stable for 13 years later. On follow up MRI, the infarct size regressed to 0.5 × 0.4 × 0.4 cm, by 3 months, and remained quiescent at 1 and 13 years later. **Conclusion:** In selected patients, with AIS, thrombolysis improves morbidity and quality of life.

Keywords

Alteplase, Acetylsalicylic Acid, Clopidogrel, Ischemic Stroke, Pontine Stroke, Thrombolysis, National Institute of Health Stroke Scale, Modified Ranke

Score, Recombinant Tissue Plasminogen Activator, rtPA

1. Introduction

Acute ischemic stroke (AIS) is the second leading cause of death and a major cause of disability in the world with an incidence of 92/100K population. Its incidence tends to be higher in men than in women and is more prevalent in black and Hispanic adults compared to their white counterparts, although this varies by geographic region and prevalence of co-morbid conditions [1]. Pontine ischemic stroke (PIS) is a common subtype of stroke occurring in the posterior part of the brain and accounts for approximately 7% of all AISs and may affect both sides of the brain in about 10% - 33% of cases [2]. Studies focusing specifically on PS are limited [3]. However, it is associated with high morbidity and mortality with a 2018 review report showing a 48.1% mortality rate within 30 days [2]. Additionally, PS may recur 4.1% at 1 month, 6.3% at 6 months, and 7.8% at 8 months [4]. Previous management of AIS was limited to stabilization of its co-morbidities and antiplatelet agents [5]. In 1981, a key publication changed the entire approach to stroke management by documenting the Penumbra, which is nonfunctional yet salvageable brain ischemia in animals [6]. In 1985, Dr Justin Zivin published a seminal article in Science showing a functional benefit of thrombolytic therapy after embolic stroke using tissue plasminogen activator in rabbits [7]. In 1995, the NINDs trial for the use of Recombinant Tissue Plasminogen Activator (rtPA), known as Alteplase for AIS, in humans, was published [8]. Despite its approval in 1996, by the Food and Drug Administration for AIS, thrombolytic therapy, by rtPA, did not fully enter mainstream medical use for fear of intracranial bleed [8]. Multiple guidelines and contraindications for its use were issued. The absolute contraindications were: allergy, bleeding disorders, liver disease, and recent bleeding lesions/surgery. The relative ones include: 1) arrival timing (before needling) > 4.5 hours, 2) non-disabling AIS, and 3) large ones [9]. Moreover, a wave of large, randomized controlled trials and meta-analyses hampered the enthusiasm for thrombolysis and even warned against the use of dual antiplatelet therapy adding more to the hesitancy and inexperience [10] [11]. In this case report, we present the details of presentation and management of a patient with significant disability following PS in whom vigilant rtPA-therapy improved his morbidity and quality of life.

2. The Case

A 58-year-old man presented with sudden weakness of the left upper and lower limbs. He reached the hospital 3 hours after its onset. Previously, he only had mild type II diabetes mellitus that was adequately controlled with Diamicron MR 120 mg daily with Glucophage XR 500 mg twice/daily. He did not consume alcohol or cigarettes before. On his initial physical examination, the patient was conscious,

oriented ×3 and without distress of shortness of breath or pain. He was afebrile and with normal blood pressure. His body weight was 90 kg. He did not have lymphadenopathy, goiter, jugular venous distension or oedema. Systemic examination did not show abnormality except for left-sided grade 1/5 motor power left-sided hemiplegia with loss of sensation for pain and temperature in the affected areas as well as right trigeminal, abducens, facial and vestibular cranial nerve palsy yet without ataxia and conjugate gaze palsy indicating an acute PS at 12 based on the National Institute of Health Stroke Scale (NIHSS) and +4 by modified Rankin score (mRs). Laboratory tests showed normal peripheral leucocytic and platelet counts. Hemoglobin was 13 g/L with normal transferrin saturation% and vitamin B12 level. Prothrombin and activated partial thromboplastin times were normal. Serum urea, creatinine, electrolytes, CO_2-content, liver function tests and TSH were normal. Serum glucose was 12 mmol/L and hemoglobin A1c was 9%. LDL-cholesterol was 4 mmol/L yet triglycerides were normal. Urine routine and microscopy did not show abnormality except for glucosuria without ketonuria. Chest x-ray and ECG were normal. Abdominal and pelvic ultrasound did not show any abnormalities. Carotid doppler, transthoracic echocardiography and 24-hour Holter-monitoring did not show abnormalities. Urgent computerized tomographic scan (CTS) of the brain did not show abnormality (Figure 1(a)). Hence, the patient was transferred to the intensive care unit and was started on rtPA for thrombolysis of his AIS. The starting dose was 8 mg as an intravenous bolus over 1 minute followed by 73 mg diluted in normal saline and infused at constant rate over 1 hour *i.e.* total dose of 0.9 mg/kg (81 mg). After reconfirming normal activated partial thromboplastin time following rtPA, unfractionated-heparin was started for 24 hours, at a dose of 10,00 IU/hour. Following that, Aspirin was started at a dose of 100 mg daily. After confirming absence of intracranial bleed, 3 days later, Clopidogrel 75 mg daily was added to Aspirin. Prior to starting rtPA, the patient had received IV Omeprazole 40 gm daily that was changed to 20 mg daily after the 3rd day to protect against stomach stress ulceration and subsequent bleeding. Moreover, Atorvastatin 40 mg was added, on a daily basis, to control his hypercholesterolemia. Further investigations emerged a few days later confirming that his initial tests for hypercoagulable disorders viz. Factor V Leiden and the prothrombin G20210A mutation, protein C, protein S, antithrombin III, antiphospholipid antibodies and homocysteine were normal. Moreover, serum complements, protein electrophoresis, antinuclear antibodies, anti-cytoplasmic antibodies, cryoglobulins, hepatitis B surface antigen and hepatitis C antibodies were normal. With such management, the patient resumed his motor power and mRs decreased to +2 by 12 hours. By 72 hours, he improved further with only numbness on the left side and mRs decreased to +1. CTS at that time confirmed a right posterolateral non-hemorrhagic pontine infarct (Figure 1(b)). Magnetic resonance imaging (MRI) angiography by the 7th day showed normal extra- and intracranial vascular disease (Figure 2) with 6 × 10 mm subacute non-hemorrhagic pontine infarct (Figure 3(a)). The patient was able to resume his active work, 1-

week post-AIS, and remained stable for 13 years later. Moreover, follow up MRI, 3 months later, showed regression of the infarct size to 4 × 5 mm (Figure 3(b)) that remained quiescent at 1 and 13 years later (Figure 4).

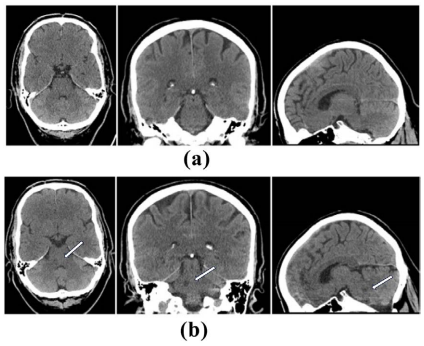

Figure 1. Axial, coronal and sagittal images of a plain CT scan of the pons showing (a) no lesion on admission and (b) a posterolateral hypodense lesion (Arrows) indicating an acute ischemic infarct 3 days later.

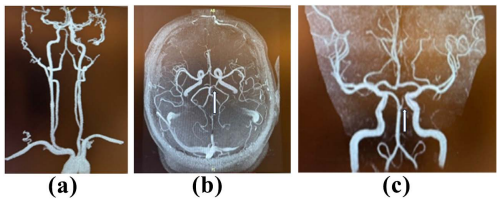

Figure 2. Magnetic resonance angiogram of the neck and brain,1-week post-stroke, showing normal extracranial and intracranial vasculature (a) and circle of Willis except for fetal pattern of the left posterior cerebellar artery which is a normal variant (Arrows) in ((b) & (c)).

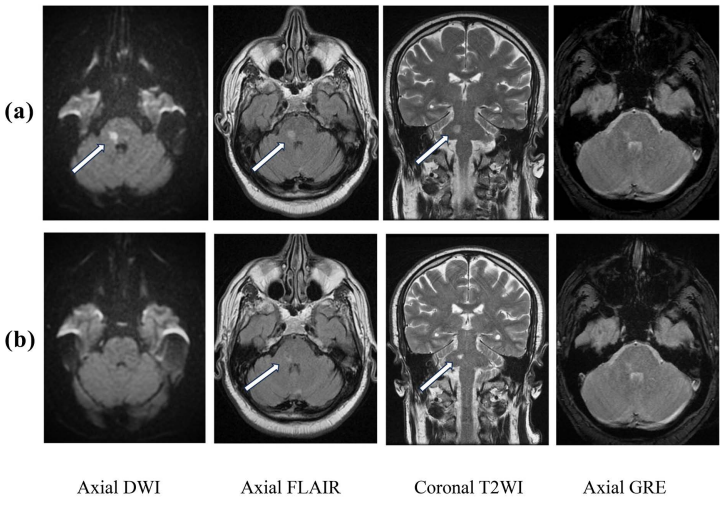

Figure 3. MRI of the brain at; (a) 1-week post-CVA showing 6 × 10 mm early subacute pontine infarct (Arrows) as shown by marked restricted diffusion in DWI images with slightly high in FLAIR and T2WI, and (b) 3-months later showing reduction of its size to 4 × 5 mm and encephalomalacia with peripheral gliosis (negative lesion on DWI and depicted focal change in FLAIR & CORONAL T2WI). Axial GRE is negative for bleed in both MRI.

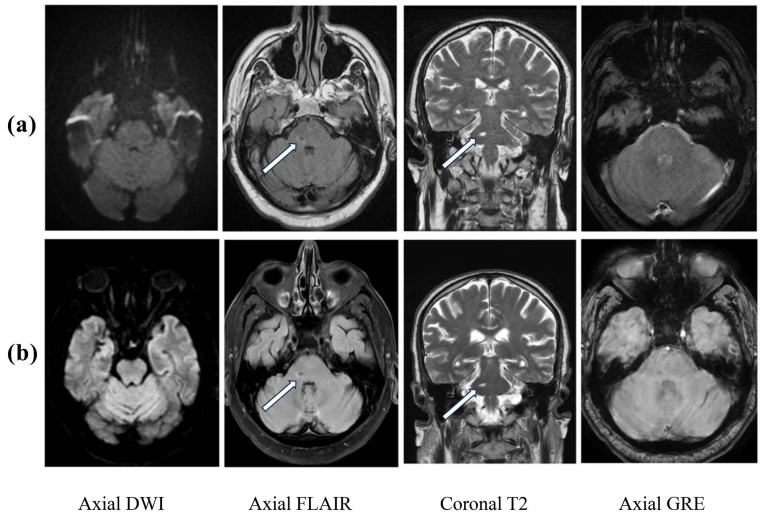

Figure 4. Follow up MRI of the brain at 1-year (a) and 13-years later showing stable post-ischemic stroke changes and encephalomalacia (Arrows) since depicted by axial FLAIR and coronal T2WI and negative DWI. Axial GRE is negative for bleed in both MRI.

3. Discussion

Patients with PIS typically present with crossed hemiplegia contrary *i.e.* ipsilateral cranial nerve defects associated with contralateral pyramidal tract disease, contrary to contralateral facial and pyramidal disease in classic brain strokes. In the latter, predisposing factors are atherosclerosis via diabetes mellitus, hypertension, dyslipidemia, smoking, as well as cardiogenic emboli. Moreover, the disease is in the anterior circulation. In PIS, diseased basilar artery and cerebellar arteries are the culprits. Moreover, small vessel diseases viz. cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), are more common in PS compared to atherosclerosis and emboli in the anterior circulation. The prognosis of pontine stroke can vary widely and depends on its etiology, size and location [12]. Patients with unilateral and mid-pons PS may experience a favorable recovery contrary to those with bilateral or caudal ones that often face a poorer prognosis [2]. Hence, it is essential to recognize that prognosis can differ significantly based on individual factors and the specifics of each stroke case. Vigilance is essential to detect those with established major stroke and those with rapidly progressing ones. Hence, patients with AIS should be managed by a specialized multidisciplinary team to avoid time loss with inexperience and hesitancy. Patients, with AIS, should receive; 1) conservative therapy for their co-morbid conditions, 2) early start of anticoagulants for embolic strokes, rtPA in major distal ones that lacks guideline contraindications, and endovascular thrombectomy with thrombolysis in proximal major AIS, and 3) early start of single or dual antiplatelets therapy if minor strokes without established and or potential bleed [13]. In our patient; we used the same thrombolysis protocol of acute myocardial infarction and pulmonary embolism with early start of unfractionated heparin following rtPA since rtPA is rapidly cleared from the plasma, with an initial half-life of less than 5 minutes [14].

As in our patient, initial CTS may not show an AIS in the first 3 days, yet it is essential to assess for intracranial bleed that restricts the use of antiplatelets and thrombolysis. Moreover, it improved, rapidly, his morbidity and quality of life that may have been limited by the conventional conservative therapy and antiplatelet agents.

4. Conclusion

Emergency reperfusion therapy is unquestionably indicated for patients with disabling AIS without contraindications for treatment.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare that they have no potential conflict of interest related to the contents of this article.

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