Journal of Clinical Cases and Reports

Case Report | Vol 2 Iss 2

Neurological Decompensation of an Old Ischaemic Stroke following Infection

Muhammad Imran Ahmad Qureshi*

*Consultant Acute Physician, Worcestershire Royal Hospital, UK

***Corresponding author:** Muhammad Imran Ahmad Qureshi, Consultant Acute Physician, Worcestershire Royal Hospital MBBS, FRCP(Glasgow), MRCP(UK), MRCP(London), MRCPE(UK), MRCGP(UK), MACP, PGC-MedEd, UK, Tel: 07917330003; E-mail: mia.qureshi@nhs.net

Abstract

Early neurological deterioration is a well-established complication of an acute ischaemic stroke, however late deterioration is an uncommon finding and could be very challenging to diagnose and treat in timely manner.

I report a case of an 82-year-old male who had suffered a stroke 28 years earlier and was functionally performing very well afterwards and living independently.

This patient was presented to Worcestershire Hospital with signs and symptoms consistent with Urinary Tract Infection diagnosis. He was treated with Trimethoprim and his clinical condition improved significantly.

While awaiting for his social rehab he suddenly developed dysphonia and dysphagia, along with mixed upper and lower motor neuron lesion signs consistent with a posterior circulation stroke.

He was investigated extensively and MRI spine ruled out any spinal cord compression however MRI head revealed old infarcts in left parietal lobe, cerebellar region and thalamic area (no acute changes). Surprisingly MRI head also shown global atrophic change with significant atrophy of midbrain in comparison to the pons (Hummingbird sign) which rose suspicion of progressive supranuclear palsy.

Initially patient was treated for acute stroke as per stroke team advice however patient was later reviewed by neurologist who explained that symptoms were attributed due to neurological decompensation of chronic infarcts (left parietal, thalamic, cerebellar) exacerbated by acute infection. Progressive Supranuclear Palsy was also excluded based on sudden onset of symptoms as compared to gradual onset of actual disease.

His neurological symptoms improved slowly, but the dysphagia and dysphonia persisted. The risk of aspiration pre-empted the insertion of a percutaneous endoscopic gastrostomy (PEG) for feeding before he was discharged for rehabilitation.

Keywords: ***Neurological deterioration; Ischaemic stroke; Stroke progression; Stroke Recrudescence***

Received Date: December 11, 2018; **Accepted Date:** January 02, 2019; **Published Date:** January 09, 2019

Citation: Muhammad Imran Ahmad Qureshi, Neurological Decompensation of an Old Ischaemic Stroke following Infection. J Clin Cases Rep 2(2): 28-34. DOI: https://doi.org/10.46619/joccr.2019.2-1034

Background

Early Neurological deterioration following an acute ischaemic stroke is a well-described entity, occurring commonly in almost half of cases within one day of admission [1]. Also known as "stroke progression", several causes have been described, including extension of the infarct, acute recurrence, and systemic causes such as an inflammatory response to the infection [2].

A late deterioration, or neurological decompensation/stroke recrudescence of a pre-existing i.e. chronic infarct, is however an uncommon and unrecognized feature. Infection or metabolic disorders could potentially cause this, thereby unmasking neurological deficits which have otherwise clinically resolved [3].

I report a case of an 82-year-old man with a background history of an old ischaemic stroke who developed new symptoms 28 years later following an episode of infection.

Case Presentation

An 82-year-old Caucasian Gentleman was admitted to the emergency department following a fall. He described that it happened following his legs 'feeling weak'. He had a previous fall 2 years ago and felodipine was stopped. He denied any warning symptoms before the fall.

His past medical history included a stroke in 1988, radical nephrectomy for a right renal tumour, and bilateral cataracts. He didn't have any known cognitive disorders or diagnosed dementia.

Systemic examination revealed past pointing and intention tremors, with an otherwise unremarkable examination. He had an Abbreviated Mental Score of 6/10. Raised white blood cell count (12.4 × 109/L) along with an episode of hematuria following admission and a urine dip which was positive for nitrites (++), leucocytes (+), and blood (++) prompted treatment with trimethoprim for a urinary tract infection.

His medications on admission included Indapamide (1.5 mg once daily), Simvastatin (40 mg at night), and Aspirin (75 mg once daily). He consumed 5 units of alcohol/week which was recently reduced from 30 units/day and had a BMI of 23.2. He denied smoking. He lived in his house and independently managed daily activities of life, mobilizing with the aid of a stick.

He was admitted to a geriatric ward to address the issue of his falls where his Indapamide was stopped. His Abbreviated Mental Score was 9/10 after completion of the course of antibiotics.

Before commencing his discharge planning (day 8 of admission), he developed new onset acute expressive dysphasia, dysphagia, postural instability, and urinary incontinence.

Neurological exam showed mixed upper/lower motor neuron signs, with hypertonia in all four limbs. Power was normal in upper limbs but 2/5 in lower limbs. Reflexes were normal in all limbs with downward facing plantars. Past-pointing and intention tremors were also noticed similar to admission. Sensations were intact.

He was investigated extensively and has had a CT head which showed no acute changes, an MRI of the spine which ruled out spinal cord compression, and an MRI of the head which showed old infarcts in left parietal lobe, cerebellar region and thalamic

area (no acute changes). However global atrophic change with significant atrophy of midbrain in comparison to the pons rose suspicion of progressive supranuclear palsy.

He was initially seen by Stroke team who advised starting acute stroke management in view of his symptoms. He was then reviewed by a Neurologist after 7 days from the start of symptoms and noticed improvement in neurological symptoms. (Power was (4/5) over the right lower limb and (5/5) over the left lower limb. Reflexes and tone were normal in both lower limbs, Sensations were normal and cranial nerve examination was normal. Based on history and examination, progressive supranuclear palsy was ruled out, attributing symptoms to cerebrovascular decompensation due to the urinary tract infection.

He was reviewed by Speech and language therapists on multiple occasions because of poor swallowing where they advised for enteral feeding through a nasogastric tube due to risk of aspiration. Following regular review, he had a flexible endoscopic assessment which raised concerns over silent aspiration. Due to failure of improvement of swallow, a PEG (percutaneous endoscopic gastrostomy) was inserted. He was discharged following PEG insertion for further rehabilitation in a community hospital by stroke physiotherapists in view of his unsteady mobility.

Investigations

On admission, lab investigations showed leucocytosis (12.4 × 109 /L) with neutrophilia (10.5 × 109 /L) due to infection which returned to normal range following antibiotic therapy. All his haematological and biochemical investigations were within normal range. His B12, Folate, Thyroid-stimulating hormone, Calcium, Magnesium, and Liver function tests were within normal range. A clotting screen also showed no abnormality.

ECG showed sinus rhythm with T wave inversion in V1 and V2 which were comparable to an ECG taken in 2014.

A CT scan done after development of the new symptoms showed generalized volume loss without a specific gradient, as well as small vessel ischemic changes. Mature left occipital lobe cortical infarct and Bilateral periventricular lacunar infarcts were demonstrated. There was no intra or extra axial bleed, proximal intravascular thrombus, sulcal effacement, brain shift or hydrochephalus.

MRI Spine demonstrated chronic degenerative changes most obvious at L4/L5 level where there is stenosis of the spinal canal and distortion of thecal sac but no compression of the cauda equina.

MRI Head showed periventricular white matter changes keeping with small vessel disease. There was mature chronic infarct of left parietal lobe with hemosiderin deposition due to old bleed. There were also dilated perivascular spaces at the level of the basal ganglia with chronic lacunar infarcts at the thalami. Finally, there were small chronic focal peripheral infarcts in cerebellar hemispheres.

Differential Diagnosis

Based on the symptoms, a wide array of different pathologies needs to be excluded. Although imaging can reliably exclude many diagnoses, clinical examination remains an important criterion for several neurological diseases that can't be confirmed by imaging alone.

Although cauda equine syndrome typically presents with lower motor neuron features, involvement of the conus medullaris can present with a mixed upper/lower motor neuron symptom [4]. This diagnosis was excluded by the MRI of the spine.

Radiological imaging of the head would be important to ensure no acute cerebrovascular events are causing the symptoms, and both CT as well as MRI head revealed the previous mature infarcts, but no evidence of acute changes. Metabolic and electrolyte disturbances that could potentially induce neurological deficits have been excluded by lab investigations.

The other main diagnoses to exclude are disorders that present with parkinsonian features as progressive supranuclear palsy (PSP).

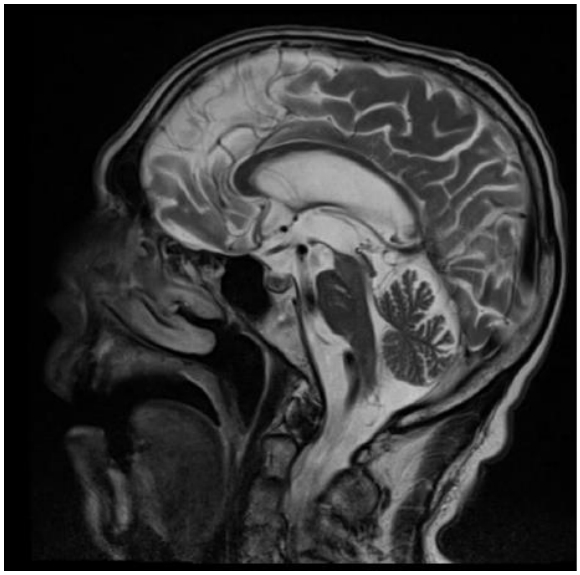

Figure 1: MRI Head showing atrophy of the midbrain compared to the pons (Hummingbird sign).

The MRI atrophy of the midbrain compared to the pons (Hummingbird sign) (Figure 1) along with dysphonia, dysphagia, and gait instability could all support supranuclear palsy. Although these MRI changes are a good indicator for PSP [5], diagnostic clinical criteria are essential to confirm diagnosis as it largely remains a clinical diagnosis [6]. The acute onset of symptoms and absence of supranuclear palsy on cranial nerve examination prompted the exclusion of PSP by the neurologist. PSP is classically identified with a slow progressive onset along with oculomotor findings on examination of cranial nerves III, IV, and VI [7].

Treatment

The patient was on 75 mg of clopidogrel and Simvastatin 20 mg when he developed neurological symptoms. He was loaded with 300mg of Aspirin for 4 days and discharged on 75 mg of aspirin, 75 mg of clopidogrel, and 40 mg of simvastatin.

A percutaneous endoscopic gastrostomy was inserted pre-discharge due to risk of aspiration following assessment by speech and language therapists.

Outcome and Follow-up

Improvement of neurological symptoms occurred during admission, with persistence of dysphagia and instability which led to insertion of a percutaneous endoscopic gastrostomy (PEG) line and further rehabilitation for mobility.

Epidemiology

Globally, the incidence of stroke due to ischemia is 68 percent, while the incidence of hemorrhagic stroke (intracerebral hemorrhage and subarachnoid hemorrhage combined) is 32 percent, reflecting a higher incidence of hemorrhagic stroke in low- and middle-income countries [8]. In the United States, the proportion of all strokes due to ischemia, intracerebral hemorrhage, and subarachnoid hemorrhage is 87, 10, and 3 percent, respectively [9].

Worldwide, stroke is the second most common cause of mortality and the third most common cause of disability [10].

In the United States, the annual incidence of new or recurrent stroke is about 795,000, of which about 610,000 are first-ever strokes, and 185,000 are recurrent strokes [9].

Men have a higher incidence of stroke than women at younger but not older ages, with the incidence reversed and higher for women by age 75 years and older [9].

There is very limited data available for Stroke Recrudescence. In 2017 a large study was conducted in the Massachusetts General Hospital to identify patients for the period January 1, 2000, to November 30, 2015, who had a primary or secondary diagnosis of cerebrovascular disease, who underwent magnetic resonance imaging of the brain at least once, and whose inpatient or outpatient clinician note or discharge summary stated the term recrudescence [11].

Of the 153 patients, 145 had prior infarct, 8 had hypertensive brain haemorrhage, and 164 admissions for post stroke recrudescence (PSR) were identified. The patients' mean (SD) age was 67 (16) years, and 92 (60%) were women. Recrudescence occurred a mean (SD) of 3.9 (0.6) years after the stroke, lasted 18.4 (20.4) hours, and was resolved on day 1 for 91 of the 131 episodes with documented resolution time (69%) [11].

Compared with the control group (patients who did not experience recrudescence), the PSR group (patients who were hospitalized for recrudescence) had more women, African American individuals, and those who self-identified as being from "other" race [11].

Discussion

Transient worsening of post stroke neurologic deficits or reemergence of previous stroke-related deficits (or poststroke recrudescence [PSR]) in the setting of toxic metabolic factors is a frequently encountered phenomenon that has not been adequately characterized [11].

In 2017, a crossover and cohort study of 153 patients confirmed that post stroke recrudescence happen almost 4 years after the initial event and more common with ischemic stroke than hemorrhagic stroke [11].

It was also mentioned that main triggers are the infection, hypotension, hypothermia, insomnia or benzodiazepine use [11]. Previous studies have shown that larger or more severe strokes confer greater susceptibility to infections [12,13]. Given the diverse and systemic nature of these triggers and the fact that PSR involves a subset of prior stroke symptoms without new neurologic symptoms, the triggers likely act via subtle effects on the undamaged ipsilateral and contralateral brain regions that subserve poststroke plasticity [14].

New neurologic deficits are usually mild to moderate in severity and do not exceed the previous stroke deficits. Symptoms can start abruptly but can be remedied completely within hours or days after the resolution of the trigger [11].

Interestingly in this case patient was very well and fully independent post initial stroke 28 years ago. Despite stroke recrudescence due to UTI, his swallowing problems persisted and he ended up with PEG insertion.

This case highlights the possibility of new symptoms which can be persistent despite treating the PSR trigger. This phenomenon was not fully mentioned in the literature and will need further research.

Learning Points/Take Home Messages

1. Chronic infarcts can present with recurring/new symptoms due to local or systemic risk factors (Neurological Decompensation).
2. Neurological diseases that can present with parkinsonian symptoms need careful consideration with an expert neurological opinion.
3. Although Imaging can be helpful in identifying progressive supranuclear palsy, it is mainly done to exclude other diagnoses, as progressive supranuclear palsy remains largely a clinical diagnosis based on slow and progressive onset of symptoms.

Conflict of Interest

No conflicts of interests.

Patient Consent

Obtained.

References

1. Kwan J, Hand P (2006) Early neurological deterioration in acute stroke: clinical characteristics and impact on outcome. Journal of the Association of Physicians 99(9): 625-633.
2. Castillo J (1999) Deteriorating stroke: diagnostic criteria, predictors, mechanisms and treatment. Cerebrovascular Diseases 9(Suppl 3): 1-8.
3. Wityk R and Llinas R (2007) Stroke. 1st (Edn.) Philadelphia: American College of Physicians, 88.
4. Harrop JS, Hunt GE, Vaccaro AR (2004) Conus medullaris and cauda equina syndrome as a result of traumatic injuries: management principles. Neurosurgical Focus 16(6): 1-23.
5. Graber JJ, Staudinger R (2009) Teaching NeuroImages: “Penguin” or “hummingbird” sign and midbrain atrophy in progressive supranuclear palsy. Neurology 72(17): e81-e81.
6. Litvan I, Agid Y, Calne D, et al. (1996) Clinical research criteria for the diagnosis of progressive supranuclear palsy (Steele-Richardson-Olszewski syndrome) report of the NINDS-SPSP international workshop. Neurology 47(1): 1-9.
7. Troost BT, Daroff RB (1977) The ocular motor defects in progressive supranuclear palsy. Annals of Neurology: Official Journal of the American Neurological Association and the Child Neurology Society 2(5): 397-403.
8. Krishnamurthi RV, Feigin VL, Forouzanfar MH, et al. (2013) Global and regional burden of first-ever ischaemic and haemorrhagic stroke during 1990–2010: findings from the Global Burden of Disease Study 2010. The Lancet Global Health 1(5): e259-e281.

9. Benjamin EJ, Blaha MJ, Chiuve SE (2017) Heart Disease and Stroke Statistics-2017 Update: A Report From the American Heart Association. Circulation 135(10): e146-e603.
10. Lozano R, Naghavi M, Foreman K, et al. (2012) Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. The Lancet 380(9859): 2095-2128.
11. Topcuoglu MA, Saka E, Silverman SB, et al. (2017) Recrudescence of deficits after stroke: clinical and imaging phenotype, triggers, and risk factors. JAMA Neurology 74(9): 1048-1055.
12. Chamorro A, Urra X, Planas AM (2007) Infection after acute ischemic stroke: a manifestation of brain-induced immunodepression. Stroke 38(3): 1097-1103.
13. Hug A, Dalpke A, Wieczorek N, et al. (2009) Infarct volume is a major determiner of post-stroke immune cell function and susceptibility to infection. Stroke 40(10): 3226-3232.
14. Nudo RJ, Wise BM, SiFuentes F, et al. (1996) Neural substrates for the effects of rehabilitative training on motor recovery after ischemic infarct. Science 272(5269): 1791-1794.