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Case Report

Complex Presentation of Acute Ischemic Stroke with Small Haemorrhagic Foci and Cranial Nerve Involvement in the Background of Diabetes, Hypertension, and Ischemic Heart Disease

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Abstract

This case describes a patient who developed an acute stroke in the presence of several health problems. Sudden onset of dizziness followed by a fall with head injury. After this, he developed slurred speech, weakness of the left arm, and deviation of the mouth to the left. Symptoms appeared suddenly, did not progress, and there was no loss of consciousness. He was admitted to the neurology department for evaluation. On admission, investigations showed haemoglobin 13.2 g/dL, white blood cell count 9200, and platelets 3.19 lakh. However, blood clotting studies showed increased prothrombin time of 16.3 and INR of 12.6 on some days, indicating delayed clotting. His medical history revealed hypertension for five years, type 2 diabetes mellitus, and ischemic heart disease for ten years. He had undergone coronary angioplasty earlier and was taking medicines like enalapril, metformin, glimepiride, atorvastatin, and clopidogrel. He had quit smoking ten years ago after smoking 5–10 cigarettes daily for 15 years. He did not consume alcohol. During admission, he was treated conservatively as advised by neurosurgery, cardiology, and ophthalmology teams. Antiplatelet medicines were stopped due to the presence of intracranial bleeding. He received intravenous antibiotics (ceftriaxone), stomach protection (pantoprazole), an antiemetic (ondansetron), mannitol to reduce brain swelling, vitamin injections, and insulin for blood sugar control. Other medicines included bisoprolol, enalapril, atorvastatin, modafinil, and baclofen. Supportive care included physiotherapy for limb and speech recovery, restricted salt intake below 2 g/day, and limited fluid intake below 1.2 L/day. This case highlights the role of careful conservative management and the importance of long-term rehabilitation in a patient with multiple risk factors such as hypertension, diabetes, and heart disease.

Keywords: Ischemic Stroke; Hypertension; Coronary Angioplasty; Diabetes Mellitus

Introduction

Brainstem strokes account for about 10% of ischemic strokes and most commonly involve the pons, a vital structure responsible for breathing, heart rate, balance, and cranial nerve functions. The clinical presentation is often vague, with symptoms such as dizziness, nausea, vomiting, and visual disturbances, which are nonspecific and may lead to delayed diagnosis. Early and accurate identification is critical, as prompt treatment improves recovery and reduces the risk of severe disability [1]. Stroke remains a major global health problem and is the second leading cause of death and disability worldwide, accounting for nearly 10.5% of global deaths, while about 5% of survivors live with permanent disability [2]. Stroke occurs when the cerebral blood supply is abruptly interrupted, either due to vascular occlusion in ischemic stroke or vessel rupture in haemorrhagic stroke, leading to rapid neurological deficits such as weakness, slurred speech, or paralysis, which may result in death or long-term impairment if untreated [3]. Medically, stroke is defined as a sudden-onset neurological syndrome caused by a vascular event, resulting in focal or global loss of brain function lasting more than 24 hours or leading to death, with acute ischemic stroke being the most common subtype. Established risk

factors include hypertension, diabetes mellitus, ischemic heart disease, atrial fibrillation, and cigarette smoking, while emerging risk factors such as vitamin B12 deficiency, low folic acid levels, and elevated homocysteine are increasingly recognised and are clinically important because they are preventable [4]. Most stroke patients, particularly elderly individuals, have pre-existing chronic illnesses; comorbidity refers to the presence of one additional disease, while multimorbidity indicates two or more chronic conditions, and only about 6% of stroke patients have no comorbidity. Conditions such as hypertension, diabetes, and heart disease often coexist, share common pathophysiological mechanisms, and significantly increase stroke risk. Vitamin B12 deficiency impairs folate metabolism, leading to hyperhomocysteinemia, which causes endothelial damage, promotes thrombosis, and increases the risk of ischemic stroke [5]. Stroke may also present with cranial nerve involvement, including facial palsy due to seventh cranial nerve damage, which may be central or peripheral, and other conditions, such as neurocysticercosis, can mimic stroke, particularly in endemic regions [6,7]. Complications like hyponatremia and cerebral vasospasm may follow head injury or intracranial haemorrhage and worsen neurological outcomes [8,9]. This case is significant because it highlights the complex interaction between acute ischemic stroke, haemorrhagic components, cranial nerve involvement, and multiple metabolic and cardiovascular risk factors, underscoring the importance of recognising preventable causes such as vitamin B12 deficiency and adopting an individualised, multidisciplinary approach, making it a valuable contribution to clinical understanding and stroke prevention strategies.

Case Presentation

A 70-year-old male was admitted with a sudden onset of complaints. While at the market, he developed dizziness followed by a fall with head trauma. Immediately afterwards, he was noted to have slurring of speech, weakness of the left upper limb, and deviation of the mouth to the left side. His family reported that the symptoms were acute, non-progressive, and he remained conscious throughout. There was no history of loss of consciousness, seizures, tongue bite, drooling, involuntary passage of urine or stool, or abnormal jerky movements. He also gave a two-month history of cough with yellow sputum and breathlessness, which was progressive and worse in the mornings.

His past medical history included hypertension for five years on enalapril 2.5 mg, type 2 diabetes mellitus on metformin 500 mg with glimepiride 2 mg, and ischemic heart disease for ten years, for which he underwent percutaneous transluminal coronary angioplasty (PTCA). He was on atorvastatin 20 mg and clopidogrel 75 mg regularly. He had stopped smoking ten years ago after a 15-year habit of 5–10 cigarettes daily and denied alcohol consumption. His personal history revealed normal appetite, mixed diet, and sound sleep, with regular bowel and bladder habits.

On examination, he was conscious, obeying commands, and moderately built and nourished. His GCS score is 3 (eyes open to noise) and 4 (eyes open spontaneously). His vitals were as follows: temperature, 99.8°F; pulse, 73 beats/min; blood pressure, 130/80 mmHg; respiratory rate, 14 cycles/min; SpO_2, 98% on room air; weight, 60 kg; height, 170 cm; and BMI, 21. Random blood sugar was 254 mg/dL (Table 1). General examination showed no pallor, icterus, clubbing, cyanosis, oedema, or lymphadenopathy. The cardiovascular system examination revealed normal S1 and S2 sounds without murmurs. The respiratory system had bilateral normal breath sounds, and the abdomen was soft and non-tender. Central nervous system examination revealed aphasia, left facial weakness with loss of nasolabial fold, left-sided hypotonia, reduced motor power (upper limb 3/5, hand grip 2/5, lower limb 3/5), extensor plantar response on the left, and speech disturbance. Other reflexes were variably reduced, and gait, coordination, sensory, and higher functions could not be assessed due to his condition.

Investigations showed haemoglobin 13.2 g/dL, TLC 9200, platelets 3.19 lakh, urea 22 mg/dL, creatinine 0.6 mg/dL, sodium 137 mEq/L, potassium 4.5 mEq/L, and urine with nil albumin and sugar 1+. MRI brain revealed an acute infarct in the frontal and right insular cortex with a small haemorrhage in the right anterior putamen and caudate head, partial thrombosis of the M1 segment, and complete thrombosis of the M2/M3 segment of the right MCA, a small gliotic focus in the right cerebellar region, and mild age-related cerebral and cerebellar atrophy. ECG showed T wave inversion in I, aVL, V2–V6. Chest X-ray showed increased bronchovascular markings. 2D echocardiography revealed left ventricular ejection

fraction (LVEF) of 40%, severe hypokinesia of mid and apical walls, thinning of the anterior wall and apex, IVC of 13 mm collapsing with inspiration, and no clot or vegetation. USG abdomen showed bilateral grade 1 renal parenchymal changes (Table 2).

During hospitalisation, he was treated conservatively as advised by neurosurgery, cardiology, and ophthalmology teams. Ophthalmology ruled out papilledema or retinopathy. He was managed with intravenous ceftriaxone 1 g twice daily, pantoprazole 40 mg once daily, ondansetron 4 mg twice daily, mannitol 100 ml thrice daily, Vitcofol C 2 cc IV, RT feeds 200 ml every three hours, insulin for glycemic control, bisoprolol 2.5 mg once daily, enalapril 5 mg once daily, modalert 100 mg once daily, baclofen 10 mg stat, and atorvastatin 40 mg at night. Antiplatelet drugs were withheld due to intracranial bleeding. Supportive measures included physiotherapy for limb and speech recovery, salt restriction below 2 g/day, and fluid restriction below 1.2 L/day.

Neurological deficit and subsequent improvement were assessed through serial structured neurological examinations during hospitalisation and follow-up. Assessment focused on level of consciousness, speech and language function, cranial nerve involvement, motor power, muscle tone, deep tendon reflexes, plantar response, and functional mobility. Motor weakness was objectively graded using the Medical Research Council (MRC) scale, which documented left-sided hemiparesis and allowed comparison over time. Formal documentation using standardised stroke scales such as the National Institutes of Health Stroke Scale (NIHSS), Barthel Index (BI), and modified Rankin Scale (mRS) was not performed at admission due to the acute clinical setting, mixed ischemic–hemorrhagic pathology, and limited patient cooperation during the early phase. Functional improvement was therefore monitored clinically by observing changes in speech output, limb movements, response to physiotherapy, ability to maintain posture, and participation in daily activities. We acknowledge that the use of NIHSS, BI, and mRS would have provided more objective and comparable outcome measures and recognise this as a limitation of the present case report. Future similar cases will incorporate these standardised scales to enhance the quality and reproducibility of neurological assessment. On discharge dated the patient's condition was stable. He continued to have residual neurological deficits but was hemodynamically stable. He was referred back to ESI hospital for further follow-up, physiotherapy, and rehabilitative care with the same medication plan and lifestyle advice. This case highlights the occurrence of an acute cerebrovascular accident in the setting of multiple comorbidities, the role of conservative management in the presence of intracranial bleed, and the importance of long-term rehabilitation for functional recovery.

Table 1: Baseline data.

DATE	BP	PR	SPO2	RR	GRBS	TEMP
06/08/2025	130/80	73	98	14	254	98.1
07/08/2025	152/76	72	95	16	255	98.3
08/08/2025	142/80	80	98	16	143	98.1
09/09/2025	124/80	80	98	14	139	98.7
10/08/2025	100/80	78	98	16	137	99.1
11/08/2025	120/68	83	95	14	137	98.4
12/08/2025	122/70	70	98	18	152	98.1
13/08/2025	130/98	68	97	18	150	98.1
14/08/2025	122/80	80	98	16	130	98.8

Table 2: Investigation Reports.

Lab data	06/08/2025	07/08/2025	08/08/2025	09/08/2025	10/08/2025	11/08/2025	12/08/2025	13/08/2025
Hb	13.2	14.8	15.4	15.2	14.5	13.9	14	15.1
PCV	39.8	43.9	47.8	47.9	43.3	42.5	42.4	45.8
RBC	4.99	5.61	5.92	5.99	5.46	5.35	5.37	5.85
Platelets	3.19	3.92	3.84	3.45	3.46	3.50	3.66	4.03
WBC	9200	17800	13580	14000	10990	10410	11180	13080
Neutrophiles	65	72	71	73	72	71	68	66
Lymphocytes	28	20	20	19	19	23	23	27
Eosinophiles	01	01	01	00	01	00	01	01
ESR	48	46	40	38	34	34	30	32
Urea	22	39	56	56	41	34	34	35
Creatinine	0.6	0.7	0.7	0.9	0.6	0.6	0.6	0.6
Sodium	137	131	129	129	131	128	133	138
Potassium	4.5	4.3	4.4	4.0	3.8	3.8	3.7	4.1
Chloride	103	99	99	97	101	110	101	102
Prothrombin time	16.3	16.3	41.7	16.6	16.1	16.5	16.9	16.2
INR	12.6	3.29	1.29	4.56	2.56	4.98	1.76	1.54

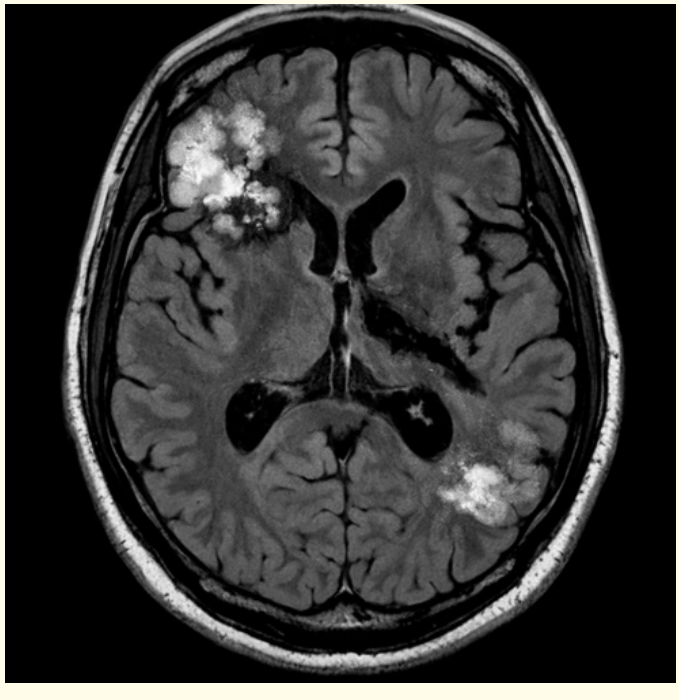

Figure 1: MRI of the brain showing an infarct and thrombosis.

Discussion

In this case, the patient was treated using a multidisciplinary team approach, where doctors from different specialities worked together. The treatment included several medicines to control infection, reduce symptoms, and improve overall health. He was given intravenous ceftriaxone 1g, which is an antibiotic used to treat bacterial infections. Pantoprazole 40 mg was given to reduce excess stomach acid and protect the stomach lining. Ondansetron 4 mg was used to prevent nausea and vomiting. Mannitol 100 ml was used to remove extra fluid from the body, reduce brain swelling (cerebral oedema), lower intracranial pressure, and help the kidneys by promoting urine excretion of harmful substances. There are very few studies in India that compare vitamin B12, folate, and homocysteine levels and examine their individual or combined effects on the risk of acute ischemic stroke [11]. Managing comorbidities is very important and should be part of a wider plan to reduce the harmful effects of hemorrhagic stroke. Understanding that death risk is linked not only to the stroke itself but also to other existing illnesses can help doctors provide better care by giving more attention to treating these conditions, and not focusing on the stroke alone [3]. Even though the initial MRI confirmed a parietal stroke, this finding alone did not explain the complete left-sided facial droop, which was more suggestive of a peripheral nerve problem [8]. Stroke patients with many health problems (multimorbidity) often need multiple medicines (polypharmacy). This may increase the chance of drug interactions; for example, taking antiplatelets with non-steroidal anti-inflammatory drugs can increase bleeding risk [5]. Overall, the management of this patient was largely conservative, focusing on medicine, diet, and supportive care. Preventive strategies target known risk factors for stroke, including high blood pressure, diabetes, heart disease, sickle cell anaemia, cigarette smoking, and newer risk factors such as high homocysteine, vitamin B12 deficiency, and low folic acid levels [4].

Conclusion

In conclusion, this case highlights the complex nature of acute ischemic stroke when it occurs in patients with long-standing comorbidities such as diabetes mellitus, hypertension, and ischemic heart disease. The presence of a right middle cerebral artery infarct with small haemorrhagic foci made management more challenging, as the use of antiplatelet drugs had to be carefully balanced against the risk of bleeding. The additional finding of right seventh cranial nerve palsy shows how stroke can also affect cranial nerves, leading to functional and cosmetic deficits [We acknowledge that labelling the deficit as a right seventh cranial nerve palsy may not be fully supported by objective documentation. Accordingly, we have revised the manuscript to describe the finding as left-sided facial weakness, consistent with a central facial palsy, rather than definitively diagnosing a peripheral seventh cranial nerve palsy. This limitation has been clearly stated in the revised CNS examination section, and the interpretation has been aligned with the radiological findings of right hemispheric stroke, which can explain the facial weakness through corticobulbar pathway involvement. We appreciate the reviewer's suggestion and have corrected the terminology to improve anatomical accuracy and scientific clarity]. The patient's past medical history, including heart failure with reduced ejection fraction and a history of PTCA, further increased the risk of complications. Conservative management, supportive care, and physiotherapy played an important role in stabilisation and recovery. This case underlines the importance of early recognition, rapid investigation, and multidisciplinary care in reducing disability and improving outcomes in stroke patients with multiple health conditions.

Limitation of the Study

The main limitation of this study is that it describes only a single patient, so the findings cannot be generalised to all stroke cases. The patient had many health problems, such as diabetes, hypertension, and ischemic heart disease, which makes it difficult to say how much each condition directly influenced the stroke outcome. Some advanced tests, like genetic studies and long-term neuroimaging follow-up, were not done due to limited resources and patient preference. The short duration of hospital stay also restricted the observation of long-term recovery and complications. Because of these limitations, the study mainly gives insight into one complex presentation but cannot be applied as a guideline for wider populations.

Source of Funding

None.

Conflict of Interest

None.

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