

Disabling tinnitus and third nerve palsy following pontine haemorrhage: Application of ICF framework.

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Introduction

Spontaneous intracerebral hemorrhage (ICH) accounts for 10-20% of strokes worldwide, with hypertensive vasculopathy being the most common underlying cause [1]. Brainstem hemorrhages represent one-third of ICH and often arise in the pons [2]. Vital sensorimotor pathways and cranial nerve nuclei traverse this region. Thus, characteristic signs of pontine ICH include hemiparesis, cranial nerve palsies, and impaired consciousness [3].

Oculomotor nerve involvement causes ipsilateral ptosis and pupillary dilation with preservation of extraocular movements due to sparing of the superior branch [4]. Abducens palsy and internuclear ophthalmoplegia reflect medial longitudinal fasciculus damage [2]. Beyond focal deficits, brainstem hemorrhages frequently precipitate headaches and tinnitus due to vascular irritation of pain and auditory pathways [5]. Persistent, troublesome tinnitus impairs concentration and quality of life [6].

The International Classification of Functioning, Disability, and Health (ICF) codifies the multifactorial impacts of health conditions like stroke [7]. The ICF enhances rehabilitation by elucidating specific limitations in body structures/functions, activities, participation, and environmental interactions. ICF-based assessment informs goal setting and interventions to optimize functioning and societal participation.

This report presents an ICF profile of a patient with tinnitus and oculomotor palsy following pontine hemorrhage. MRI confirmed the hemorrhagic lesion. ICF components were examined to capture the breadth of disability. Tinnitus management and multidisciplinary therapies were tailored accordingly to promote recovery.

Case presentation

A 31-year-old male with untreated hypertension presented with right-sided involuntary movements followed by disorientation and unconsciousness. Examination revealed left ptosis, anisocoria, right hemiparesis, and extensor plantar response. Brain MRI revealed a hemorrhage in the right pontine tegmentum with extension into the midbrain and medulla (Figure 1).

The neurological deficits indicated a left-third nerve palsy and proper corticospinal tract involvement. During the hospitalization, the patient developed constant high-pitched ringing in both ears. Tinnitus onset coincided temporally with the hemorrhage, implicating vascular disruption of central auditory pathways as the likely cause. He also experienced a persistent headache attributed to irritation of pain-modulating brainstem nuclei by the hemorrhage.

An ICF profile was created to characterize the myriad sequelae (Table 1). Impaired body structures and functions include the oculomotor nerve, corticospinal tracts, and central auditory system. Activity limitations comprised impaired vision, communication, self-care, and mobility. Tinnitus hindered concentration and sleep. Environmental factors like social support and rehabilitation access were facilitators, while personal factors such as young age and hypertension control motivation enabled progress.

Specific management approaches were implemented based on the ICF profile. For tinnitus, the patient underwent tinnitus retraining therapy involving sound therapy and counseling. Vision rehabilitation and physical/occupational therapy also focused on facilitating activities and participation. The ICF profile will continue guiding multidisciplinary care during outpatient rehabilitation.

The patient was biomedical diagnosis:

1. Acute cerebrovascular accident, hemorrhagic infarct in the right pons extending to the midbrain and bilateral medulla.
2. Systemic Hypertension
3. Vascular headache secondary to brainstem hemorrhage
4. Tinnitus
5. Left third nerve palsy with horizontal gaze palsy
6. Right-eye exposure keratitis

Discussion

This patient's disabling tinnitus (b2400) and third nerve palsy (b2152) resulted from a hypertensive pontine hemorrhage. The ICF framework enhanced understanding of the myriad neurological impacts. The brainstem houses vital sensorimotor infrastructure, including cranial nerve nuclei (s11051), corticospinal/corticobulbar tracts, and auditory/oculomotor pathways [2]. Accordingly, lesions cause deficits referable to involved structures.

Oculomotor nerve fascicles originate from midbrain subnuclei before coalescing to exit the interpeduncular cistern [4]. This anatomy explains the partial third nerve palsy with ptosis and mydriasis but spared extraocular movements. Medial longitudinal fasciculus damage produced a horizontal gaze palsy. Corticospinal tract involvement caused contralateral hemiparesis.

Tinnitus was a significant source of disability not directly attributable to focal damage. Instead, it likely arose from vascular irritation of central auditory networks [5]. Similar central mechanisms induce headaches through the stimulation of pontine pain modulators. Refractory tinnitus and headaches represent major complications impacting the quality of life after brainstem hemorrhages.

Applying the ICF framework enabled a granular, patient-centered characterization of health-related domains. Beyond documenting neurological impairments, the ICF profile revealed resultant limitations in diverse activities and variable environmental influences two times, directly after the stroke; this could help follow the progress, obtain a prognosis, and make decisions according to future interventions. This understanding guided interventions targeting specific participation goals. Ongoing use of the standardized ICF language facilitates clear multidisciplinary communication and goal-driven rehabilitation [7].

Finally, the MRI was instrumental in precisely localizing the hemorrhage to the right pontine tegmentum. The correlation between MRI and functional performance confirmed the neurological deficits while excluding competing etiologies. Neuroimaging improves diagnostic accuracy and prognostication in ICH. However, optimal outcomes require complementing imaging with holistic frameworks like ICF that capture lived experiences.

Conclusion

This case exemplifies multifaceted disability following pontine hemorrhage. The patient exhibited left-third nerve palsy and right hemiparesis from damage to the relevant midbrain and corticospinal tracts. Additionally, he developed centralized tinnitus and headaches due to vascular irritation of auditory and pain

pathways. These complications added significant disability not explained by structural injury alone. MRI precisely localized the hemorrhage and elucidated the neurological manifestations.

Comprehensive application of the ICF framework codified impairments in body structures/functions, limitations in activities/participation, and the impacts of environmental/personal factors. This understanding informed interventions, including tinnitus retraining therapy and physical/occupational rehabilitation, to address discrete participation goals. Ongoing use of the ICF structure and terminology will optimize multidisciplinary care. Neuroimaging and holistic paradigms like ICF are essential for accurate diagnosis and patient-centered management following turning off brainstem hemorrhages.

Consent Statement.

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

VP and IN analyzed and interpreted investigative data. IN, SH, and VP performed a literature review and contributed to writing the manuscript. All authors read and approved the final manuscript.

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Figures

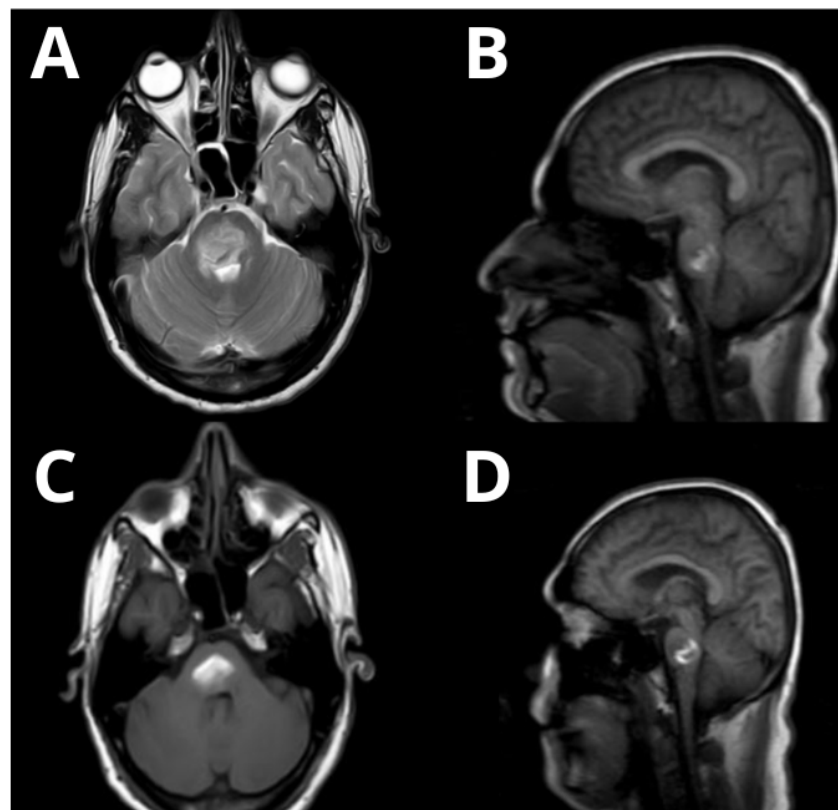


Figure 1 . - T1/T2 hyperintensities with adjacent perilesional edema are noted in pons extending to involve the right midbrain and superior part of the medulla oblongata, causing mild narrowing of the fourth ventricle. Areas of diffusion restriction and blooming were noted. Suspicious small feeding vessel noted- Few tiny foci of blooming noted in the bilateral lentiform nucleus and external capsule - likely microhemorrhages. Mild periventricular T2/FLAIR hyperintensities noted - likely vessel ischemic changes

Tables

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image2.emf available at <https://authorea.com/users/657406/articles/662226-disabling-tinnitus-and-third-nerve-palsy-following-pontine-haemorrhage-application-of-icf-framework>

Table 1 ICF functioning profile: b: Body function; s: Body structures; d: Activity and participation; e: Environmental factors; prof: health professional or other professional; 0 no impairment; 1= mild impairment; 2 moderate impairment; 3 severe impairment; 4= complete impairment; G: Intervention Goal; P=

performance of...; C= capacity in. In environmental factors: +4: Complete facilitator; +3: Substantial facilitator; +2: Moderate facilitator; +1: Mild facilitator; 0: No barrier/ facilitator; 1: Mild barrier; 2: moderate barrier; 3: Severe barrier; 4: Complete barrier. MD: Doctor of Medicine; PT: Physical therapy OT: Occupational Therapy; AuD: Audiologist.

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ICF Table.xlsx available at <https://authorea.com/users/657406/articles/662226-disabling-tinnitus-and-third-nerve-palsy-following-pontine-haemorrhage-application-of-icf-framework>