CASE REPORT
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THE ANATOMY BEHIND THE SIGNS: NEUROANATOMICAL, CLINICAL AND RADIOLOGICAL APPRAISAL OF BENEDIKT'S SYNDROME

Neurology

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ABSTRACT

Background: Benedikt's syndrome is a rare midbrain neuro-ophthalmological syndrome caused by lesions involving the paramedian midbrain tegmentum.It results from combined involvement of the oculomotor nerve fascicles, red nucleus, and adjacent cerebellothalamic pathways, cerebral peduncle. The classical constellation includes ipsilateral third cranial nerve palsy with contralateral hemiparesis and contralateral cerebellar ataxia and/or Holmes tremor and/or choreoathetosis.Due to dense anatomical organization of midbrain structures, relatively small lesions can produce characteristic crossed neurological syndromes.**Case Presentation:**A 75-year-oldfemale who is a diabetic patient, with incidental diagnosis of hypertension presented with sudden onset diplopia and drooping of the right eyelid associated with left upper and lower limb weakness. Neurological examination demonstrated ipsilateral third cranial nerve palsy and contralateral motor deficit and gait instability. MRI brain revealed an acute infarct involving the paramedian midbrain. The patient was treated according to ischemic stroke protocol with antiplatelet therapy and risk-factor optimization.**Conclusion:** Benedikt's syndrome is an uncommon but distinct midbrain vascular syndrome. Recognition of its characteristic clinical features allows precise neuroanatomical localization and timely management.

KEYWORDS

Benedikt's Syndrome, Midbrain Infarct, Oculomotor Palsy, Red Nucleus, Posterior Cerebral Artery, Brainstem Stroke.

INTRODUCTION

Brainstem infarctions often present with diverse and complex neuro-ophthalmological manifestations due to the dense anatomical arrangement of cranial nerve nuclei and long tracts within a compact region[1,2].

Benedikt's syndrome represents one of the classical "crossed brainstem syndromes" that contributed significantly to the early understanding of clinical neuroanatomical localization. First described in the late 19th century by the Austrian neurologist Moritz Benedikt, this syndrome demonstrated that discrete lesions within the brainstem could produce predictable combinations of cranial nerve and long tract deficits [3].

Prior to the advent of modern neuroimaging, such clinicopathological observations were essential for mapping functional neuroanatomy. The association of ipsilateral oculomotor nerve palsy with contralateral involuntary movements and motor weakness enabled neurologists to localize lesions to the midbrain tegmentum, involving oculomotor nucleus and adjacent motor and cerebellar pathways.

With the development of magnetic resonance imaging, these classical syndromes have been anatomically validated. Benedikt's syndrome is now known to result most commonly from lesions affecting the paramedianmidbrain due to infarction in territory of posterior cerebral artery [4]. Despite advances in imaging, recognition of its characteristic clinical features remains crucial for rapid bedside localization in acute neurological practice.

In this report, we present a case demonstrating classical clinical and radiological features of Benedikt's syndrome, highlighting the continued relevance of clinico-radiological correlation in modern neurology.

CASE PRESENTATION:

The patient was an elderly female with a history of diabetes and incidentally diagnosed with hypertension presented with sudden onset of diplopia and drooping of the right eyelid. This was associated with weakness of the left upper and lower limb.On examination, the patient was conscious and oriented

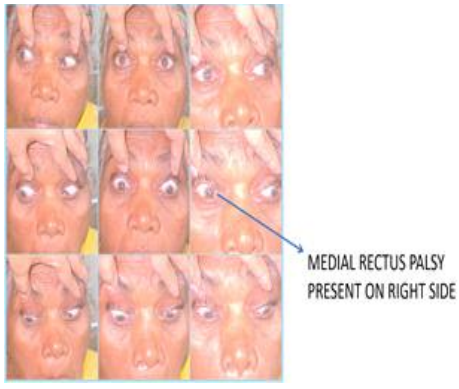


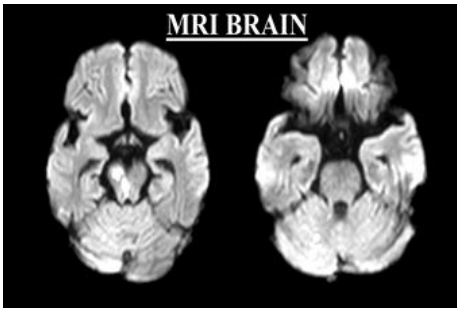
Fig 1:Nine gaze photographs showing ptosis in the right eye with restricted adduction of extraocular movements on right side.

Cranial nerve examination revealed right eye ptosis with right medial rectus palsy. Extraocular movements were restricted in adduction in right eye. Motor examination demonstrated left hemiparesis with extensor plantar response. Cerebellar examination revealed finger to nose test was impaired and rapid alternating hand movements were impaired on both sides along with gait instability.The clinical findings were consistent with Benedikt's syndrome.

Magnetic resonance imaging (MRI) of the brain demonstrated an acute infarct at right cerebral peduncle in inferior part of midbrain and right cerebellar hemisphere. Routine laboratory investigations revealed hypertriglyceridemia and Hba1c-10.1%. 2D echo was normal. Carotid Doppler revealed diffuse atheromatous changes involving bilateral carotid arterial system. The patient was managed conservatively with antiplatelet therapy, statins, and blood pressure control. Supportive

rehabilitation therapy was initiated. Clinical improvement was observed over time.

Figure 2: DWI-MRI-brain image showing acute infarct in right midbrain, right cerebral peduncle in inferior part of midbrain and right cerebellar hemisphere



DISCUSSION:

Benedikt's syndrome is a rare neuro-ophthalmological syndrome resulting from lesions of the midbrain tegmentum, involving a complex interplay of cranial nerve nuclei and ascending and descending tracts. The characteristic clinical features arise due to involvement of the oculomotor nerve fascicles, red nucleus, medial lemniscus, and cerebellothalamic pathways, which are anatomically clustered within the midbrain [1,2].

Differentiation from other midbrain syndromes is essential for accurate anatomical and clinical diagnosis. Midbrain vascular syndromes form a spectrum that includes Weber's syndrome, Claude's syndrome, and Benedikt's syndrome, each defined by specific anatomical involvement and clinical presentation. Weber's syndrome involves the cerebral peduncle leading to contralateral hemiparesis without significant cerebellar signs, whereas Claude's syndrome is characterized predominantly by ataxia without prominent motor weakness. Benedikt's syndrome represents an intermediate pattern where both cerebellar and motor pathways are affected [1,2].

The vascular supply of the midbrain tegmentum is primarily derived from paramedian perforating branches of the posterior cerebral artery supplying the midbrain tegmentum, and occlusion of these vessels leads to focal infarction producing the characteristic clinical syndrome[4].

The classical presentation consists of ipsilateral oculomotor nerve palsy manifested as ptosis, ophthalmoplegia, and diplopia, along with contralateral cerebellar signs such as ataxia, dysmetria, and tremors due to involvement of the red nucleus and superior cerebellar peduncle [5,6]. In some cases, extension of the lesion into the cerebral peduncle leads to corticospinal tract involvement, resulting in contralateral hemiparesis, as observed in the present case.

Less commonly, hemorrhage, tumors, infections or demyelinating lesions may produce similar clinical syndromes [6,7]. MRI remains the gold standard for diagnosis, demonstrating lesions in the midbrain and associated structures, thereby confirming clinical localization[8].

Management follows standard protocols for ischemic stroke, including antiplatelet therapy, statin therapy and control of risk factors [9].

A review of available literature suggests that Benedikt's syndrome is an uncommon entity, with most cases reported as isolated case reports, highlighting its rarity and the importance of clinical recognition and localization, neuroimaging and management[6-10].

In the present case, the combination of clinical findings and radiological correlation enabled accurate diagnosis and appropriate management.

CONCLUSION:

Benedikt's syndrome, though rare, offers a striking example of how precise anatomical localization can be achieved through bedside clinical examination. The presence of "crossed" neurological deficits-ipsilateral oculomotor nerve palsy alongside contralateral motor and cerebellar dysfunction-serves as a classic yet often under-recognized signature of midbrain involvement. In this case, the midbrain declared itself even before imaging,through the language of clinical signs. Ultimately, this case highlights that the eye sees what the mind knows. Timely recognition of such distinctive clinical signatures can transform diagnostic uncertainty into targeted intervention, significantly influencing patient outcomes. Thus, even in the era of advanced imaging, it is the synthesis of clinical signs that continues to guide precise localization and optimal care.

REFERENCES

1. Jankovic J, Mazziotta JC, Pomeroy SL, Newman NJ, editors. Bradley and Daroff'sNeurology in Clinical Practice. 8th ed. Philadelphia: Elsevier; 2021.
2. Splittgerber R. Snell's Clinical Neuroanatomy. 8th ed. Philadelphia: WoltersKluwer; 2019.
3. Benedikt M. Tremblementavecparalysiecroisée du moteuroculairecommun. Bull Med (Paris). 1889;3:547-548.
4. Tatu L, Moulin T, Bogousslavsky J, Duvernoy H. Arterial territories of human brain: brainstem and cerebellum. Neurology. 1996;47(5):1125-1135. doi:10.1212/WNL.47.5.1125.
5. Liu GT, Crenner CW, Logigian EL, Charness ME, Samuels MA. Midbrain syndromes of Benedikt, Claude, and Nothnagel: setting the record straight. Neurology. 1992;42(9):1820-1822. doi:10.1212/WNL.42.9.1820.
6. Adhikari A, Bhattarai AM, Pandit A, Rokaya P, Khadka M, Shahi M, et al. Benedikt syndrome in a 74-year-old hypertensive woman: a case report. Clin Case Rep. 2022;10(12):e6767. doi:10.1002/ccr3.6767.
7. Paidakakos NA, Rokas E, Theodoropoulos S, Dimogerontas G, Konstantinidis E. Posttraumatic Benedikt's syndrome: a rare entity with unclear anatomopathological correlations. World Neurosurg. 2012;78(6):715.e13-715.e15. doi:10.1016/j.wneu.2012.03.028.
8. Kumral E, Bayulkem G, Akyol A, Yunten N, Sirin H, Sagduyu A. Mesencephalic and associated posterior circulation infarcts. Stroke. 2002;33(9):2224-2231. doi:10.1161/01.STR.0000027438.93029.87.
9. Kleindorfer DO, Towfighi A, Chaturvedi S, Cockroft KM, Gutierrez J, Lombardi-Hill D, et al. 2021 guideline for the prevention of stroke in patients with stroke and transient ischemic attack: a guideline from the American Heart Association/American Stroke Association. Stroke. 2021;52(7):e364-e467. doi:10.1161/STR.0000000000000375.
10. Caplan LR. VertebrobasilarIschemia and Hemorrhage: Clinical Findings, Diagnosis, and Management of Posterior Circulation Disease. 2nd ed. Cambridge: Cambridge University Press; 2015.