A middle-aged man was admitted with symptoms of excessive daytime somnolence (EDS) of 1-year duration. He used to have sudden and irresistible bouts of sleep, occurring six to seven times a day, amounting to 18–20 hours of sleep daily. He had immense difficulty carrying out his daily activities and felt perennially exhausted. He was not able to concentrate at work. However, the patient did not give a history of sleep paralysis, cataplexy, or hallucinations. On examination, he was not obese or overweight. Neurological examination was normal.

All laboratory tests were normal. Thyroid function assessment was normal. Magnetic resonance imaging (MRI) brain with contrast showed a presence of calcified arteriovenous malformation (AVM) in the superior colliculus of the dorsal midbrain extending into the rostral pons inferiorly and vermis of cerebellum posteriorly (Figs 1 and 2). Nocturnal polysomnogram with multiple sleep latency tests did not show features suggestive of narcolepsy.

Excessive daytime somnolence is said to be the difficulty in remaining awake during the day, resulting in undesirable spells of drowsiness or sleep.  

Excessive daytime somnolence or hypersomnia, not associated with the other components of narcolepsy, is commonly seen in many neurological conditions. The anatomical substrates responsible for hypersomnia appear to be heterogenous and variable.

A meta-analysis on 116 cases of symptomatic narcolepsy/EDS was done in 2005. Genetic disorders (34%), tumors (29%), head trauma (16%), and demyelinating diseases (9%) were the commonly reported...
etiologies. Strokes, tumors, cysts, abscesses, hematomas, vascular malformations, and multiple sclerosis plaques were some of the uncommon causes of EDS.\(^1\)

The superior colliculus has intricate connections to many brain regions which encompasses the periaqueductal gray, thalamus, cortex, brainstem, and spinal cord.\(^2\)

The superior colliculus–posterior thalamus region is concerned with the onset of non-rapid eye movement sleep, rapid eye movement sleep, and wakefulness in mammals.\(^2\)
Mathis et al. emphasized the importance of the medial tegmentopontine area in the pathophysiology of EDS. Focal lesions in this area might lead on to various atypical narcoleptic syndromes. Symptomatic EDS has rarely been reported in patients with isolated lesions in the diencephalon, hypothalamus, midbrain, medial pontine tegmentum, and exceptionally in the ventral pons, a region pertaining to the hypocretin projection pathway.

This patient who had a calcified AVM in a strategic site extending from the superior collicular region of the midbrain to rostral pons was suffering from symptomatic or secondary EDS. This highlights the clinical correlation of sleep disorders with anatomical substrates. Sleep disorders, which have a complex heterogeneity, can rarely be the presenting manifestations of many neurological conditions, and awareness of this association facilitates early recognition and prompt management.

REFERENCES


