Cataplexy Secondary to Brain Lesions: A Systematic Review and Case Report of Pontine Infarction

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BACKGROUND

Secondary narcolepsy has been described in association with structural lesions of the central nervous system, most often presenting with the full narcoleptic phenotype.

In contrast, isolated cataplexy is rarely reported but provides a valuable pathophysiological model to disentangle the mechanisms of REM atonia generation from those mediated by the orexinergic system.

Understanding these selective dissociations offers insight into the functional anatomy of emotion–motor integration and REM sleep control

OBJECTIVES

The aim of this study was to conduct a systematic review of published cases of secondary cataplexy associated with structural brain lesions and to report a new singular case of isolated cataplexy following pontine ischemic injury.

METHODS

A systematic search of PubMed (updated May 2025) was performed using "cataplexy" combined with brain-injury terms (infarct, tumor, encephalitis, demyelination, trauma).

Eligible reports included human single-case studies describing cataplexy secondary to structural brain lesions documented by imaging.

Exclusions were animal studies, primary narcolepsy without lesion, reviews, duplicates, and insufficient clinical data.

For each case, we extracted lesion location and etiology, SOREMP, EDS, sleep paralysis, hallucinations, RBD, treatment, and outcome.

Lesions were grouped into major neuroanatomical categories (pons—tegmentum/basis/paramedian, midbrain, medulla, thalamus, hypothalamus, basal ganglia, cerebellum, cortex).

Outcomes were harmonized (Improved/Stable/Worsened/Deceased).

Descriptive analyses summarized distributions and proportions; Fisher's exact or chi-square tests explored lesion–symptom associations.

Results were expressed as proportions with 95% Wilson confidence intervals.

Data synthesis followed PRISMA 2020 and CARE case-report standards.

RESULTS

Thirty-four published single-case reports met inclusion criteria, each describing cataplexy secondary to a structural CNS lesion confirmed by imaging.

The most frequent etiologies were neoplastic lesions (30%), followed by vascular (26%), inflammatory/demyelinating (18%), and traumatic or iatrogenic causes (26% combined).

Lesions were predominantly located in the hypothalamus, brainstem (especially the pons), and suprasellar region.

Across all cases, SOREMP were documented in 26 of 36 patients (79%, 95% CI 63–91), excessive daytime sleepiness (EDS) (58%, 95% CI 41–74), sleep paralysis (36%, 95% CI 21–53), REM sleep behavior disorder (RBD) (21%, 95% CI 10–38), and hypnagogic/hypnopompic hallucinations (18%, 95% CI 8–35).

A small subgroup (n = 8; 23%) presented isolated cataplexy without EDS, typically after pontine or midbrain ischemic lesions.

No statistically significant association was observed between pontine localization and the presence of SOREMP (OR 0.21; 95% CI 0.03–1.61; p = 0.19) or RBD (OR 1.33; 95% CI 0.25–7.08; p = 1.00).

Similarly, the overall association between lesion site (multi-category variable) and RBD frequency was non-significant ($\chi^2 = 2.91$; p = 0.82).

A combined REM-dissociative cluster—defined as ≥2 features among SOREMP, RBD, sleep paralysis, or hallucinations—was observed in 41% of patients but showed no correlation with lesion topography ($\chi^2 = 0.66$; p = 0.72).

Despite the lack of formal statistical significance, brainstem (particularly pontine) lesions tended to show higher frequencies of REM-related phenomena (71%) compared with supratentorial locations (38%), supporting a potential functional link between pontine structures and REM atonia control.

CASE REPORT

We report the first documented case of isolated cataplexy following a paramedian pontine ischemic lesion.

A 76-year-old man developed partial cataplexy triggered by positive emotions, in the absence of daytime sleepiness, sleep paralysis, hallucinations, or sleep-related behaviors.

Polysomnography showed fragmented sleep with preserved REM atonia, while MSLT revealed two SOREMPs.

The case supports the concept of a selective disruption of pontine REM atonia pathways, distinct from orexinergic dysfunction, and exemplifies a rare human model of isolated cataplexy.

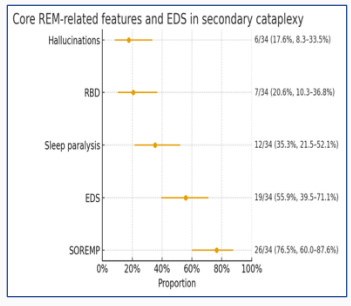
DISCUSSION AND CONCLUSION

This systematic review highlights the clinical and anatomical heterogeneity of secondary cataplexy.

The present case represents, to our knowledge, the first documented instance of isolated cataplexy following a paramedian pontine ischemic lesion.

This observation supports the existence of a REM sleep motor inhibitory circuit within the pons, functioning independently of hypothalamic orexinergic pathways.

Overall, these findings suggest that the orexin-centric hypothesis of cataplexy should be broadened: while orexin deficiency remains the principal mechanism in primary narcolepsy, direct disruption of pontine REM atonia networks can also produce cataplexy, indicating a more complex, circuit-based pathophysiology...



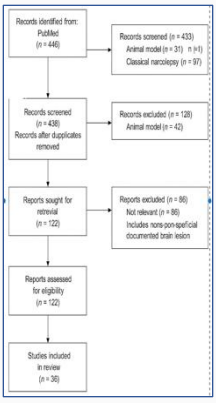


Figure 1. Brain MRI: median pontine ischemic outcome (FLAIR, sag)

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