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TAC-tic syndromes: a case series on the complex overlap between trigeminal autonomic cephalalgias and trigeminal neuralgia

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Introduction

Trigeminal autonomic cephalalgias (TACs) and trigeminal neuralgia (TN) are classified as distinct entities by the ICHD-3, yet their co-occurrence as TAC-tic overlap syndromes is increasingly reported. When these conditions coexist, two distinct phenotypes must be recognized and independently treated, despite shared features of strict unilaterality and trigeminal territory involvement.

Case presentation

We describe five women, aged 47 to 84 years, with TAC-tic overlap syndromes: two SUNCT-tic, two cluster-tic, and one paroxysmal hemicrania-tic (PH-tic). All patients presented a TN component, characterized by brief shock-like paroxysms in the trigeminal territory, and a TAC component, characterized by longer, severe unilateral attacks with ipsilateral cranial autonomic features. In Case 1, TN in V2 preceded SUNCT by 11 years; later, left V1/V2 attacks lasting 45–90 seconds, occurring up to 10 times per hour with tearing, conjunctival injection, and rhinorrhea, developed in recurrent bouts. Partial control was achieved with carbamazepine and lamotrigine, and after neurovascular compression was identified, microvascular decompression led to complete 2-year remission. In Case 2, right periorbital/temporal attacks lasting 2–3 minutes, up to more than 15 per day, with profuse tearing and conjunctival injection, were classified within the SUNCT/SUNHA spectrum with SUNCT phenotype, although at the longer end of the ICHD-3 duration range. Nine months later, a distinct TN phenotype emerged, with 1-second V1/V2 shocks triggered by eating. A vascular loop was later identified, and microvascular decompression led to complete remission. In Case 3, infraorbital TN shocks coexisted with cluster attacks lasting 90–120 minutes, with rhinorrhea, tearing, conjunctival injection, and agitation; control was achieved with nerve blocks, carbamazepine, and verapamil. In Case 4, cluster-like supraorbital attacks lasting 40–60 minutes coexisted with tactile-triggered TN paroxysms and resolved after nerve block, carbamazepine, and verapamil, with sustained remission after withdrawal. In Case 5, TN preceded PH by 26 years; explosive attacks lasting 15–20 minutes, 4–8 per day, with tearing, periorbital edema, agitation, and suicidal ideation, were markedly reduced by indomethacin, while TN required neuromodulator adjustment.

Discussion

SUNCT-tic cases showed neurovascular compression and remission after decompression, supporting a relevant peripheral mechanism. In cluster-tic and PH-tic cases, absence of compression and differential responses to verapamil, nerve blocks, indomethacin, and neuromodulators suggest interaction between central trigeminal-autonomic dysfunction and peripheral trigeminal mechanisms.

Conclusion

TAC-tic syndromes are rare, likely underrecognized, and diagnostically complex. Dual-phenotype characterization based on ICHD-3 criteria with treatment directed at each component independently enabled satisfactory control in all five cases.

Keywords:
Trigeminal neuralgia
Trigeminal autonomic cephalalgias
SUNCT syndrome
Cluster headache
Paroxysmal hemicrania

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