Teaching NeuroImages: Multiple clinical manifestations of a ganglionic sympathetic defect

A 39-year-old woman with longstanding asymmetric facial flushing noticed episodic deformation of her left pupil (figure 1), which 2 months later became miotic. Despite no ptosis, a left Horner syndrome was pharmacologically confirmed (figure 2). She also had left cheek pain, present only at mealtime. Radiologic investigations were normal.

This case illustrates many manifestations of a unilateral sympathetic deficit to the head and eye including harlequin sign,1 tadpole pupil, Horner syndrome, and masticatory pain from parasympathetic overactivation of secretory myoepithelial cells, typically upon the first bite.2 The site of dysfunction localizes to the superior cervical ganglion; the cause remains unknown.

AUTHOR CONTRIBUTIONS
Myriam Ladaique: study concept and design, acquisition of data, analysis and interpretation of data. Aki Kawasaki: study concept and design, acquisition of data, analysis and interpretation of data, study supervision, critical revision of manuscript for intellectual content.

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M. Ladaique reports no disclosures relevant to the manuscript. A. Kawasaki has received book royalties from Cambridge University Press in the past 3 years. Go to Neurology.org for full disclosures.

REFERENCES
Figure 2  Left Horner syndrome pharmacologically confirmed

(A) Baseline anisocoria (pre-apraclonidine) in room light. (B) After instillation of 1 drop of topical apraclonidine in each eye, the miotic left pupil became mydriatic; the right pupil remained unchanged. This reversal of anisocoria is due to adrenergic denervation hypersensitivity of the left iris dilator and confirms a Horner syndrome.
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