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Harlequin Syndrome Associated with Multilevel Intercostal Nerve Block

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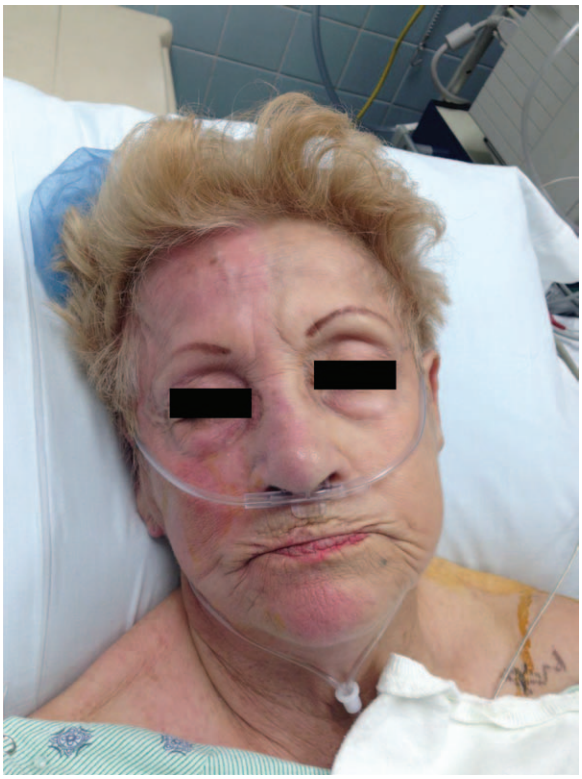

HARLEQUIN syndrome is characterized by clearly demarcated unilateral facial flushing and sweating caused by an injury to the sympathetic fibers to the face.[1,2] Lesions creating the characteristic finding occur along the pathway connecting the hypothalamus, intermediolateral column of the spinal cord, cervical sympathetic ganglia, and postganglionic sympathetic fibers. Most of the sudomotor and vasomotor fibers innervating the face exit the spinal cord at the T2 to T3 level and continue along both the internal and external carotids.[3] Injury along this pathway leads to facial anhidrosis and a loss of sympathetically mediated vasodilatation. The "normal" appearing side is actually the injured side. The contralateral side is thought to overcompensate creating the characteristic findings.[1] Upper extremity involvement implies concomitant injury to superior thoracic sympathetic ganglia.[3]

The patient in the figure developed Harlequin syndrome with upper extremity flushing and diaphoresis in the postanesthesia care unit 1 h after receiving left T3 to T11 intercostal nerve blocks with 0.25% bupivacaine for postoperative analgesia for thoracoscopic resection of the left lower lobe of the lung. Her vitals were at baseline, and she was neurologically intact. Her symptoms completely resolved in less than 12 h.

Harlequin syndrome may be seen after multilevel high thoracic intercostal nerve blocks. In the absence of hemodynamic instability or new neurologic findings, a central neurologic insult can be excluded, and reassurance is all that is required.[3] We present this figure to educate fellow clinicians of this rare presentation of a typically benign, self-limited complication after multilevel high thoracic intercostal nerve block.

Competing Interests

The authors declare no competing interests.

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