Editors' Note: Lekhra gives 2 possible explanations for the wrong side dilated pupil in Wijdicks' case. Commenting on the clinical reasoning case regarding the unusual cause of transverse myelitis, Ghuman and Kaur discuss why the diagnosis of paraneoplastic myelopathy should have been in the differential. Bhargava and Elble explain why they disagree.

—Chafic Karam, MD, and Robert C. Griggs, MD

WRONG SIDE DILATED PUPIL

Om Prakash Lekhra, Indore, India: I read the *NeuroImage* by Drs. Wijdicks and Giannini¹ with interest. There are 2 possible explanations for the wrong side dilated pupil. The first possibility is that raised intracranial pressure caused kinking of the oculomotor nerve over the clivus, just posterior to the clinoid. Another alternative is that supratentorial pressure exerted dynamic axial brainstem distortion leading to contralateral dilated pupil.²

Author Response: Eelco F. Wijdicks, Rochester, MN: We appreciate these additional comments from Dr. Lekhra. In general, the third nerve can be compressed against the clivus, but it is difficult to unmistakably establish that with a routine autopsy procedure. Moreover, a relationship with a wrong side fixed pupil has not been shown.

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CLINICAL REASONING: AN UNUSUAL CAUSE OF TRANSVERSE MYELITIS?

Mandeep S. Ghuman, Chandigarh; Shabdeep Kaur, Shimla, India: Bhargava and Elble¹ excluded the common differentials of longitudinal transverse myelitis but overlooked paraneoplastic myelopathy (PNM). Cord lesions can still have an etiology despite biopsy-proven metastases in the brain. Having made the final diagnosis of small-cell lung carcinoma, the authors should have considered PNM.

Brain metastases are common in lung carcinomas, whereas intramedullary spinal cord metastases (ISCM)

are very rare. PNM may be as rare as ISCM but, when present, PNM is often the presenting manifestation. CSF examination was equivocal. Paraneoplastic antibody workup has its limitations as only 60%–70% will have detectable antibodies.² Regarding immunotherapy, there are no evidence-based recommendations and paraneoplastic syndromes mediated by antibodies are potentially reversible vs those with intracellular target antigens.²

Typical neuroimaging findings in PNM are symmetric, longitudinally extensive tract, or gray matter–specific changes,³ whereas ISCM lesions are usually solitary, in the thoracic spinal cord, located eccentrically within the cord and expanding the cord.⁴ Overall imaging features would favor PNM over ISCM. PET can differentiate between the 2 by demonstrating hypermetabolic foci in ISCM, and can play a pivotal role in certain clinical scenarios: when MRI is contraindicated and when there is suspicion of ISCM with equivocal spinal cord magnetic resonance findings in those who cannot receive IV gadolinium.⁵

Author Response: Pavan Bhargava, Baltimore; Rodger J. Elble, Springfield, IL: The authors thank Drs. Ghuman and Kaur for their comments. In this case, we agree that it was not possible to definitively rule out PNM. However, there are several factors that make this diagnosis unlikely. PNM is commonly associated with moderate CSF lymphocytic pleocytosis, elevated protein, and sometimes oligoclonal bands in the CSF.⁶ Multiple CSF analyses in our patient were normal. The incidence of PNM is far lower than that of spinal cord metastases (SCM), which can occur in 0.1%–0.4% of all cancer patients.⁷ Invoking Occam razor, the simplest explanation for the spinal cord findings would be a SCM. In addition, the MRI spine is consistent with SCM,⁸ especially noting that the cord appears to be swollen at T8 with maximal abnormality in that area.¹ Whereas a PET scan may have helped, studies have shown that PET imaging can miss up to 30% of SCM, so it would not have been definitive.⁹ The lack of response to immunotherapy, presence of multiple brain metastases, and an advanced stage lung cancer make the diagnosis of SCM far more likely than PNM.

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