tumor is rare. However, their comment that there are only 13 such cases documented in the literature is incorrect.

Achani and Colover reported two cases of pathological laughter due to compression of the brain stem by tumor. Their first case had a 3-year history of pathological laughter as the chief complaint. This patient had an epidermoid tumor in the cerebellopontine angle and its removal cured the laughter. Their second patient’s symptom was thought to be due to an intrinsic brain-stem tumor.

Recently, Lal and Chandy reported a case of pathological laughter in an 18-year-old woman. The laughter was due to an intrinsic brain-stem mass lesion in the pontomedullary region. Computerized tomography-guided stereotactic biopsy confirmed that the lesion was a glioma. Her symptoms had improved at the end of 8 months following radiotherapy and chemotherapy.

References

RESPONSE: I am indeed grateful to Dr. Gopinath for bringing to my attention the references mentioned in his letter. I had overlooked the report of 1976. As to the report of 1992, my article had already been submitted before this particular article was published.

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Idiopathic Hypertrophic Cranial Pachymeningitis

TO THE EDITOR: Drs. Mamela, et al., describe three cases of “idiopathic hypertrophic cranial pachymeningitis” (Mamela AN, Kelly WM, Davis RL, et al: Idiopathic hypertrophic cranial pachymeningitis. Report of three cases. J Neurosurg 79:270-276, August, 1993). They state that this form of granulomatous pachymeningitis had been reported in only six cases prior to their paper. My perusal of their documentation and review of the literature prompts this collegial admonition.

In 1949, Naffziger and 1 reported the case of a 40-year-old woman with obstructive hydrocephalus and dramatically thickened dura mater ensheathing the cerebellar hemispheres in which resection was followed by slow recovery. A review of the literature disclosed a wealth of publications pertinent to the subject of possible etiologies and pathogenesis. My calling this paper to the attention of Dr. Mamela and his colleagues may find a hospitable reception at their hands. But perhaps more important is the purpose to alert the many who write in our journals that comprehensive scholarship involves thorough searches into the world of literature, much of which predates any computer service and often includes jewels of information not only in English but in other languages. There are more reports available than the authors cite to enrich their contribution.

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RESPONSE: I read with great interest the recent article by Drs. Mamela and coworkers (Mamela AN, Kelly WM, Davis RL, et al: Idiopathic hypertrophic cranial pachymeningitis. Report of three cases. J Neurosurg 79:270-276, August, 1993). I would like to share my experience of having seen a patient with a similar presentation in 1988 at our Institute of Medical Sciences. A young man of about 25 years of age presented with a 6-week history of diplopia and inability to close the right eye. There was no history of fever, headaches, diabetes, ear discharge, trauma, or exposure to vaccinations. He was a well-built man with a normal ear, nose, throat, and systemic examination. He had bilateral sixth and right seventh and 10th cranial nerve palsies. The rest of his neurological examination was normal, and there were no meningeval signs. A complete blood count, blood biochemistry, collagen profile, venereal disease tests, chest x-ray film, and computerized tomography head scan were normal. Electrophysiological tests (visual evoked responses, brain-stem auditory evoked responses, and nerve conduction velocity tests) were normal. Routine cerebrospinal fluid examination for cytology and malignant cells was negative. Bacterial and fungal staining and culture were negative. Over the course of 3 to 4 weeks, the patient developed decreased hearing in the right ear with minimal left facial weakness. The patient left the hospital with a diagnosis of multiple cranial nerve palsies. Immunosuppressant therapy was not given because the condition could not be recognized. The short duration of evolution is an odd feature in this case as compared to the recent report. I would like to invite comments from the authors.

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RESPONSE: We appreciate the comments of Dr. Stern and Dr. Kouk, and read them with great interest. We thank Dr. Stern for bringing his article to our attention and acknowledge his admonition that our review did not cite the entire literature of the subject of pachymeningitis. Dr. Stern’s point concerning the need for a thorough study of all pertinent articles on a subject when preparing a review of this nature, including those written in foreign languages and journals, is well taken. The article he mentions on chronic pachymeningitis is
a fine example of a truly complete review.\textsuperscript{1} Furthermore, we wholeheartedly agree that it is worthwhile to seek out the "jewels of information" that may be found in the less recent neurological literature.

However, we do not believe our failure to reference some articles yielded a faulty or incomplete analysis. The patient described by Naffziger and Stern\textsuperscript{1} suffered from obstructive hydrocephalus without any cranial neuroptihic neuropathies. In contrast, every patient in our series, as well as all other reported cases of idiopathic hyperpertrophic pachymeningitis, suffered from progressive cranial nerve palsies or cerebellar symptoms, but not hydrocephalus. Their patient had repeated febrile episodes, and the authors themselves concluded that repetitive infections were the most likely cause of pachymeningitis. None of the patients in our series and none of the cases of idiopathic pachymeningitis we cited from the literature suffered from febrile illnesses. In addition, most of these patients had progressive disease, whereas Naffziger and Stern's patient was cured by dural excision. Idiopathic pachymeningitis is clearly distinct from hemorrhagic pachymeningitis interna or purulent pachymeningitis, and these differences were duly mentioned in our article. Finally, granulomatous illnesses such as sarcoid were not excluded in much of the older literature on this subject, making a diagnosis of idiopathic pachymeningitis impossible.

The primary goal of our report was to define the idiopathic form of pachymeningitis and to describe the clinical characteristics, diagnostic assessment, and treatment options. We did not intend to review all etiologies of pachymeningitis or the historical development of this subject. We believe the literature we cited reflects the intended focus of our paper.

The clinical history and diagnostic workup in the case described by Dr. Koul strongly suggests pachymeningitis but the normal computerized tomography scan (CT) argues against a diagnosis of the idiopathic form; contrast-enhanced CT scans or magnetic resonance images would be useful in such a case. The relatively short time course is atypical, but we do not think this by itself excludes a diagnosis of idiopathic pachymeningitis. A dural biopsy remains essential, as all other causes of pachymeningitis, such as sarcoid, granulomatosis, or infection, must be excluded before idiopathic pachymeningitis can be diagnosed. If follow-up review of his patient is possible, we recommend a dural biopsy followed by a course of steroids. We would be interested to learn of the outcome.

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