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Journal search and commentary

Article reviewed: Papilledema and obstructive sleep apnea syndrome[☆]

Gerald B. Rich

Pacific Sleep Program, Portland, OR, USA

Objectives

To discuss the finding of papilledema in patients with obstructive sleep apnea syndrome, and to review its pathogenesis and clinical features in this sleep disorder.

Study design

Case review

Study population

Four patients were investigated, each presenting with visual loss. Bilateral papilledema was found. All four patients were reported to have obstructive sleep apnea syndrome.

Methods

Detailed ophthalmologic investigations were performed. Neuroimaging studies, cerebrospinal pressure measurements and fluid analysis, complete blood counts, and weights were also obtained. Neither full neurological examination nor polysomnography results were indicated.

Results

Bilateral papilledema that was often asymmetric was found, with varying degrees of visual impairment. Three individuals appeared to have morbid obesity, while one was said to be of normal weight. The latter was said to have an unspecified anatomic variation of the pharynx. CSF pressures were either normal or mildly elevated, taken while the patients were awake; while fluid composition was indicated as within the normal range. Computed tomography and magnetic resonance imaging studies were reported as normal. One patient underwent 24-h intracranial pressure monitoring; this demonstrated increases in intracranial pressure to as high as 48 cm H_2O during clinically apparent sleep, associated with oxygen desaturation episodes. The latter were thought to be indicative of apneic events; there was no indication that actual measures of sleep or of airflow were conducted. Two of the patients were known to have sleep apnea prior to the finding of papilledema; the third was reported as having been diagnosed as having sleep apnea three years after initially presenting with papilledema; the fourth was reported to have been diagnosed as having sleep apnea at an unspecified date after his ophthalmologic evaluation. One individual was treated by tracheostomy, with prompt resolution of his visual abnormalities. Another had more rigorous application of nasal CPAP along with weight reduction, associated with slow visual improvement. The third had visual improvement with weight reduc-

[☆] Purvin VA, Kawasaki A, Yee RD, (Arch Ophthalmol 2000, 118:1626–1630).

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tion as well as nasal CPAP, and the fourth had visual improvement following upper airway surgery.

Conclusions

The authors conclude that intermittent intracranial pressure elevations may produce persistent papilledema. They feel that the diagnosis of sleep apnea syndrome related papilledema may not be appreciated because of the lack of the usual symptoms of increased intracranial pressure and normal CSF pressure measurements. They propose its pathogenesis is related to cerebral vasodilatation.

Comment

Obstructive sleep apnea syndrome is a complex disorder with a wide range of symptoms. Among these are ones which have been postulated to possibly relate to cerebral vasodilatation and/or increased intracranial pressure. Rather scant attention has been given to visual symptoms in individuals with this disorder. This report is important in helping to focus attention on this potentially serious issue. A small number of reports of papilledema accompanied by increased intracranial pressure and associated with obstructive sleep apnea syndrome have been published over the last 15 years. The frequency of visual symptoms which may be due to intracranial pressure fluctuations as a result of sleep apnea syndrome is entirely unknown. No systematic investigation of this appears to have been conducted. However, this report provides further evidence papilledema should be added to the growing list of the complications of obstructive apnea syndrome. The response of the first case to tracheostomy, in a manner similar to that of other cases reported in the literature, convincingly indicates that increased intracranial pressure occurs in obstructive sleep apnea syndrome and can produce papilledema; reversible by treatment. The completion of continuous intracranial pressure monitoring in this case is extremely valuable in adding to our understanding of the relationship between apneic events and intracranial pressure. This is of potentially central importance to our understanding of the pathogenesis of other symptoms in sleep apnea syndrome besides the visual ones cited here.

Unfortunately, analysis is hampered by a significant limitation in the amount of information provided. Polysomnography results are unreported, so that the intensity of sleep apnea in these cases is unknown. Other cases reported have all had severe sleep apnea, at least according to their respiratory disturbance indices. It would appear that Case 1 may have been severe and the visual symptoms clearly resolved with definitive treatment of the apnea. The relationship of sleep apnea to the development of visual symptoms and papilledema in other cases is unclear, insofar as the diagnosis of sleep apnea had a very unclear temporal relationship to the appearance of visual abnormalities. In other cases in the literature, morbid obesity appears to be a central, although not a required factor.

It is not possible to draw conclusions regarding the pathogenesis of papilledema arising in association with obstructive sleep apnea on the basis of the cases presented here. In particular, the author's speculation relating these cases to pseudotumor cerebri seems unlikely to be correct. The clinical symptoms and findings, do not necessarily fit well, so it is premature to include sleep apnea under this classification. The pathogenesis of pseudotumor cerebri is as yet unknown; the main considerations have been an increase in cerebral blood volume and/or impairments in CSF absorption. Most individuals are female and morbidly obese. What relation the sex of the individual has to this disorder appears to be entirely unknown. A recent report has tied increased intra-abdominal pressure in the morbidly obese to papilledema; producing an increase in intrapleural pressure and secondarily an obstruction of cerebral venous drainage. Both hypoxemia and hypercarbia have been potentially linked to increased intracranial pressure elevations in sleep apnea patients, which could then produce papilledema. Generally, papilledema has been most convincingly associated with conditions producing consistent elevations in intracranial pressure. When CSF pressures have been measured in sleep apnea patients, episodic increases in pressure are observed, linked reasonably directly to individual apneic events. This may relate not only to changes in oxygen and carbon dioxide levels but also to the substantial fluctuations in intra-abdominal and

intrathoracic pressure associated with each apneic events. These pressure changes will by themselves raise intracranial pressure, particularly if there is limited brain compliance permitting adjustments to these pressure changes. It is certainly reasonable to speculate that repetitive increases in intracranial pressure, especially when they are seen with the high frequency expected in individuals with severe sleep apnea, could result in papilledema. This mechanism, however, may be quite different from that seen in pseudotumor cerebri.

It is rightly concluded that ophthalmologists should make appropriate clinical inquiries regarding the possibility of sleep apnea in patients undergoing evaluation of papilledema. The same can certainly be said for all clinicians investigating this physical finding. This is of potentially particular import in cases diagnosed as having pseudotumor cerebri. It is also particularly important for the sleep medicine specialist to consider a funduscopic examination in consultation on patients for sleep apnea syndrome. The presence of visual symptoms may indicate the greater likelihood of severe sleep apnea as well as the need for urgent evaluation and treatment. As indicated in the first case cited in this report, visual disturbances and papilledema should likely be added to the list of indications of poor compliance with nasal CPAP treatment. Although the vast majority of individuals with papilledema from pseudotumor cerebri experience a resolution of their visual symptoms, a significant percentage remained substantially impaired visually. The importance of early intervention in these individuals has been repeatedly emphasized; this is likely to also be true for papilledema and visual impairment associated with the obstructive apnea syndrome.

A more systematic clinical investigation of visual symptoms in patients with sleep apnea is needed. This reviewer routinely conducts funduscopic investigations of all patients undergoing evaluation for sleep apnea; papilledema has been a rare finding. However, it is certainly possible that mild levels of abnormality have gone undetected. It is also possible that those individuals going on to develop papilledema have some other unique vulnerability, as yet unidentified. Combining clinical information with the results of polysomnography in a larger series would be of substantial interest; especially if features could be found that would identify those individuals who are a particular risk for this complication.