Cranial Nerve Palsy in Tuberculous Meningitis *Versus* Acute Bacterial Meningitis

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The cornerstone in the diagnosis of meningitis is examination of the cerebral spinal fluid (CSF), while clinical feature seems to be an unreliable value; however, some previous studies evaluated diverse clinical presentations and post complications as prognostic indicators in order to differentiate diverse types of meningitis (1,2).

Since now, a wide spectrum of neurological symptoms has been described as the most common complication in meningitis survivors. The incidence of neurological sequels varies with the severity of the meningitis and the organism type.

In a recently interesting published paper in the Acta Medica Iranica, entitled "Cranial nerve palsy as a factor to differentiate tuberculous meningitis from acute bacterial meningitis" (3), several neurological features including seizure, level of consciousness, stroke, focal neurologic deficit and cranial nerve palsy at the time of admission have been investigated in a multivariate regression, of which only cranial nerve palsy has been shown as a positive predictive factor with Odds Ratio of 1.980 (1.161-3.376) and *P* value of 0.006 (3).

Cranial nerve involvement is associated with high mortality and disability in meningitis patients (4). In some previous studies, the incidence, predictors and the prognostic significance of cranial nerve involvement in patients with tuberculosis meningitis has been compared with other causative agents of meningitis (1). Meningitis with slowly evolving pathogen and basal exudate collection is often related with cranial nerves involvement (5,6). Since some unusual bacterial agents, fungal pathogens and non tuberculosis mycobacterium (NTM), also known as atypical mycobacterium may present as a chronic meningitis and lead to cranial nerve palsy, they should be considered in the differential diagnosis of tuberculous meningitis (TBM). Meningovascular syphilis, which commonly occurs within the first year after primary infection, is kind of basilar meningitis which accompanied by cranial nerve involvement (7).

Multiple cranial nerve involvement, which is indicative of basilar meningitis, has previously been reported in patients with Streptococcus pneumonia meningitis (8,9). There are also several case reports which illustrate multiple cranial nerve abnormalities in Cryptococcus meningitis in immunocompetent and immunosuppressed individuals (10,11).

Taken together, by specifying the type of organism involved, the chance for nerve involvement and probability for cranial nerve palsy could be better estimated.

In the mentioned manuscript by Moghtaderi *et al* (3), the neurological manifestations were only observed at base line and the patients were not followed for further change in neurological symptoms. In this regard, Sharma *et al* who evaluated retrospectively the incidence of cranial nerve palsy in TBM and followed the patients for nine months, revealed complete improvement of cranial nerve abnormality in some survivors in contrast in some others development of new cranial involvement has been detected (4). The improvement of neurological sequel varies depending on which nerve is involved.

Therefore, for a better estimation in comparison of cranial nerve palsy between TBM and acute bacterial meningitis (ABM), the type of involved nerve should be ascertained on admission, while the patients should be followed-up during a 6-9 month period for any further neurological complication.

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