# Commentary

Meningeal carcinomatosis (MC) is a malignant infiltration of the leptomeninges and subarachnoid space and can be a devastating complication of a systemic malignancy. Meningeal carcinomatosis was first reported in 1970 and occurs most commonly in adults with a history of breast carcinoma, lung carcinoma and melanoma. MC was an infrequent sign of dissemination of malignant neoplasia.

The incidence of MC appears to be increasing, perhaps due to longer survival of patients with malignant neoplasms and the improvements in neuroimaging techniques. MC has been reported to occur in to 2% to 25% of patients with malignancy or cancer,[1-3] and typically has a poor prognosis even with an aggressive treatment.[1] Although often found in patients with known metastatic malignancies, MC can also be the initial manifestation of an underlying malignancy. Clinical manifestations of MC are heterogeneous and symptoms are typically widespread, involving multiple levels of the central and peripheral nervous system.[4] Signs of meningeal irritation, confusion, headache, cranial nerve deficits and seizures may indicate the leptomeningeal dissemination of a neoplasm, primarily located outside the CNS.[3] Traditionally, to establish a definitive diagnosis, the presence of malignant cells in cerebrospinal fluid (CSF) is required. It has been reported that an initial cytologic examination is the only diagnostic in approximately 50% of causes, but increases with serial CSF examinations;[5] several lumbar punctures may be required to establish the diagnosis.[3] Neuroimaging is an additional tool to assess for MC. Computed tomography (CT) and magnetic resonance imaging (MRI) without contrast[3] is suboptimal for the detection of MC. An MRI with gadolinium is the only imaging modality, necessary to confirm diagnosis in suspected neoplastic letpomeningeal disease.[2] In comparing 3 MRI sequences, contrast-enhanced fast FLAIR sequences are less sensitive than standard contrast-enhanced T1-weighted MR sequences in detecting intracranial neoplastic leptomeningeal disease. However, Nardone[2] reports that, MRIs of the brain may also appear completely normal.

Hypoglycorrhachia was long time considered as typical for bacterial meningitis, mainly tuberculous meningitis. But, low sugar content of the fluid was found in past in a number of occasions, and this has also been observed in fluids when tumor cells had not been found during life although necropsy later showed a carcinomatous meningitis.[6] Very low hypoglykorrhachia was referred in patients with MC and gastrointestinal carcinomas also in the last time. That is why we recommend for intensifying of the search for malignant cells if the glucose content is found to be low.[7]

The pathophysiology of the hypoglycorrhachia is still an unsolved problem. Some authors suggested an increased utilization rate of CSF glucose by tumor cells infiltrating meningeal membranes. This theory, however, does not explain the low level of CSF glucose, frequently observed in combination with a moderate pleocytosis in tuberculous or sarcoid meningitis or, conversely, the normal levels of CSF glucose in aseptic meningitis despite the presence of a marked pleocytosis. Fishman[8] hypothesized that the lowering of CSF glucose occurs after an abnormality of the transport mechanism of the glucose molecule across the blood-brain barrier. Periodic triphasic sharp waves in electroencephalograph (EEG), described in referred case study,[9] are highly suggestive of CJD and have been very rarely described in patients with MC. In spite of the fact that it was found in patient with MC, we cannot regard it as typical for this diagnosis.

The treatment of meningeal carcinomatosis is decided on the basis of the patient`s general condition and the control status of the primary lesion, but is still having a poor prognosis. Radiotherapy, systemic chemotherapy, and palliative therapy are used. Overall, current treatments offer a poor outcome. Systemic chemotherapy is significantly associated with longer survival time than local treatment modalities. But, without therapy, the median survival is 4 weeks to 6 weeks, which can be extended to 4 months to 6 months with chemotherapy, in selected patients.

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