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Histology of Secondary Central Nervous System Lymphoma

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Short Communication

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DESCRIPTION

Systemic lymphoma can lead to central nervous system lymphoma. Patients with systemic lymphoma develop clinical or pathologic indications of cerebral involvement in between 7% and 29% of cases, according to large studies. Almost every occurrence is linked to relapsed or progressing systemic disease; solitary CNS relapse is not only uncommon, but it's frequently followed by systemic relapse.

A variety of indicators have been proposed to predict a higher risk of CNS relapse based on data from retrospective multivariate analysis. Unfavorable histology (Burkett's and lymphoblastic NHL), high LDH, stage IV disease, and the presence of 'B' symptoms are just a few of them (fever, weight loss, sweats) [1]. A higher risk of acquiring cerebral disease may be linked to certain areas of systemic involvement.

The meninges of the brain or spinal cord are virtually always involved in secondary central nervous system lymphoma, with involvement of the cerebral parenchyma being less prevalent.

As a result, secondary central nervous system lymphoma's presenting symptoms and signs mirror this characteristic. As a result, headaches, meningitis, cranial nerve palsies, mental state changes, sensory and motor impairments, and nerve root palsies develop in patients [2].

Except for the locations of illness, secondary central nervous system lymphoma has the same radiologic appearance as PCNSL. MR scanning, with or without gadolinium contrast enhancement, can improve meningeal disease resolution and should be considered in the evaluation of a patient with suspected secondary cerebral or spinal lymphoma, especially if the CT scan is unsatisfactory [3]. In the identification of meningeal illness, other investigative modalities such as myelography are not as sensitive as MR scanning.

According to the Working Formulation criteria, the majorities of cases of secondary cerebral NHL are high grade lymphomas or diffuse large B cell lymphoma. Only about 2% of low-grade lymphomas have secondary brain involvement. Due to the proclivity of secondary central nervous system lymphoma to involve the meninges, up to 50% of patients have positive CSF cytology [4].

There are no studies that sufficiently describe appropriate therapy, so treatment should be tailored to the patient's specific needs. For a patient with a good performance status who requires an aggressive treatment approach, therapy should comprise at least a mix of systemic chemotherapy and some sort of cerebral illness treatment. While it would be reasonable to base cerebral component therapy on that utilised for PCNSL within a certain hospital, given the high prevalence of meningeal involvement, intrathecal chemotherapy may be the best option [5].

The function of CNS prophylaxis in individuals at high risk of developing secondary cerebral NHL is less well-defined than in patients with acute lymphocytic leukemia, which can also cause intracerebral involvement. In high-risk individuals with high-grade systemic NHL, standard care includes CNS prophylaxis, such as intrathecal methotrexate. Bernstein, et al. looked at the frequency, natural history, and risk factors for CNS relapse in individuals with severe non-Hodgkin lymphoma [6]. The SWOG 8516, a randomised trial published in 1993, was used to investigate the efficacy of CNS prophylaxis in patients with aggressive NHL. The cumulative incidence of CNS relapse was 2.8 percent after 20 years, compared to 55.0 percent for non-CNS relapse.

16/25 individuals acquired CNS relapse while on chemotherapy treatment for their systemic NHL or 1 month after treatment; 11/25 patients had isolated CNS relapse; 10/25 patients had both systemic and CNS relapses. The 2-year survival rate was 0% against 30% (p 0.0001), with a median survival time of 2.2 months versus 9 months (non-CNS relapses). CNS relapse was predicted by the number of extranodal sites and the International Prognostic Index. Assuming a 6% chance of CNS relapse, which can be lowered to 2% with prophylaxis, we'll need to treat 960 people to benefit 40. As a result, high-risk patients with a high to high/intermediate IPI score at diagnosis should have their CSF evaluated, and if the CNS is involved, treatment rather than prophylaxis should be given.

In patients with BM involvement at diagnosis, there was no significant advantage of CNS prophylaxis; however, the power of this analysis is restricted due to the small number of events. Patients with secondary central nervous system lymphoma appear to have a lower overall survival rate than those with PCNSL. Only about 15% of individuals survive a year after being diagnosed with secondary cerebral NHL. Many of the former die of growing systemic disease rather than their CNS sickness, as these authors have recognized. It's possible that CNS disease is essentially a symptom of widespread systemic disease, and that this, rather than an inherent trait of secondary cerebral NHL, is to blame for the dismal prognosis [7,8].

REFERENCES

1. Campbell R, et al. Why don’t patients do their exercise? Understanding non-compliance with physiotherapy in patients with osteoarthritis of the knee. J Epidemiol Community Health. 2001; 55:132-138.
2. Gomara-Toldra N, et al. Physical therapy after spinal cord injury: A systemic review of treatments focused on participation. J Spinal Cord Med. 2014; 37: 371-379.

3. Maddison J, et al. Owner compliance in veterinary practice: Recommendations from a roundtable discussion. Companion Anim. 2021; 26:4-10.
4. Noreau L, et al. Participation after spinal cord injury: The evolution of conceptualization and measurements. J Neurol Phys Ther. 2005; 29: 147-156.
5. Kashluba S, et al. A longitudinal, controlled study of patient complaints following treated mild traumatic brain injury. Arch Clin Neuropsychol.2004;19:805–16.
6. Hou R, et al. When a minor head injury results in enduring symptoms: A prospective investigation of risk factors for postconcussional syndrome after mild traumatic brain injury. J Neurol Neurosurg Psychiatry. 2012;83:217–23.
7. Maas AIR, et al. Traumatic brain injury: Integrated approaches to improve prevention, clinical care, and research. Lancet Neurology 2017;16: 987–1048.
8. Sinha S, et al. Workforce planning in neurosurgery. Br J Neurosurg. 2020:34:3–8.