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Commentary

Tumors of the Central Nervous System with Primitive Neuroectodermal Subtypes

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DESCRIPTION

Neoplasms known as Primitive Neuroectodermal Tumours (PNET) have medulloblastoma as their model. These are tiny cell, malignant embryonal tumours with divergent differentiation along neuronal, glial, or, in some cases, mesenchymal lines to varying degrees [1].

The following describes the most recent WHO classification of embryonal tumours:

- Medulloblastoma
- Primordial Neuroectodermal Tumour of the CNS (PNET)
- Brain neuroblastoma
- Ganglioneuroblastoma of the CNS
- Medulloepithelioma
- Ependymoblastoma
- Rhabdoid/atypical teratoid tumour

Here, only CNS malignancies are covered. Primitive neuroectodermal tumours in the periphery are thought to be separate entities.

PATHOPHYSIOLOGY

PNET of the CNS can be roughly separated into supratentorial tumours and infratentorial tumours (medulloblastoma or iPNET).

Regarding the histogenesis of these tumours, there is a great deal of disagreement. Initially, it was believed that these dense, cellular embryonal tumours differed mainly in their location, kind, and degree of differentiation and had a common genesis from primitive neuroectodermal cells. However, several of these tumours are allocated a separate niche in the updated World Health Organization (WHO) classification under the presumption that these embryonal tumours could also develop from cells already committed to differentiation [2-5].

Despite the debate, these tumours are referred to as supratentorial and infratentorial (medulloblastoma). The latter are more common in young adults than infratentorial tumours and occur less frequently. The most typical method of PNET metastatic spread involves spinal dissemination through Cerebrospinal Fluid (CSF). By lowering peritumoral edoema, preoperative steroid therapy can aid with some of the signs and symptoms of Primitive Neuroectodermal Tumours (PNET).

RADIATION THERAPY

Radiation therapy should be administered under the guidance of a radiation oncologist and is typically administered adjuvantly. Numerous studies document a direct, dose-dependent link between local tumour control and postoperative radiation. A 5-year event-free survival rate of 50-70% is achieved with adjuvant radiation alone when posterior fossa doses of 5000 cGy and neuraxis doses of 3000 cGy are used. Lower radiation therapy dosages are less effective, at least when chemotherapy is not used. For individuals with spinal dispersion, cranial-spinal axis radiation is employed.

Newer techniques including high fractionation radiotherapy and stereotactic radiosurgery are being studied. These treatments prevent radiation-related side effects in children, such as cognitive impairment or growth retardation, which are typically observed with conventional radiotherapy. They also confine the radiation dose to the local locations [6].

CHEMOTHERAPY

A medical oncologist should be consulted before starting any chemotherapy. Various medication combinations, including but not limited to lomustine, vincristine, cisplatin, etoposide (VP-16), and cyclophosphamide, are utilised to treat these tumours. A number of trials, including those from the Children's Cancer Group (CCG) and the Pediatric Oncology Group (POG), are in progress and are examining various combinations of radiotherapy and chemotherapy. The Children's Hospital of Pennsylvania research, which reported an 80% 5-year event-free survival rate among 51 children, has so far

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produced the best results. It has been established that the benefits of CCNU are modest, especially in patients with high risk.To increase survival and outcomes; high-dose chemotherapy combined with stem cell rescue is being tested.

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