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Infectious Disorders of the Central Nervous System Epidemiology

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Introduction

Despite the development of antibiotics, vaccinations, and other medical treatments, infections of the central nervous system (CNS) still cause major morbidity and mortality throughout the world. Meningitis, encephalitis, spinal and cranial abscesses, discitis, epilepsy, and other serious problems can be brought on by the pathogenic organisms, which include bacteria, viruses, parasites, fungi, and prions. Infection with neurocysticercosis (NCC), which is on the rise in developing countries, is the primary cause of avoidable epilepsy in the developing world. Due to growing migration and tourism, drug-resistant organisms, and immunosuppressed people, the rise of additional CNS illnesses is still a worry. While most CNS infections require medical attention, some may also require neurosurgical intervention for biopsy, debridement, decompression, or reconstruction. Meningiomas make up 36% of the intracranial lesions that neurosurgeons treat, making them one of the most prevalent intracranial pathological disorders. Although the majority of these lesions are benign, the potential for meningioma development and recurrence has not been well reflected by the traditional classification of malignancies by histological type or World Health Organization (WHO) grade. Numerous targeted therapies have been unsuccessful in having a long-lasting impact on malignant cancers. The knowledge of the illness has recently undergone a fast transformation as a result of numerous groundbreaking researches analysing the genomes of intracranial meningiomas. There are at least six different mutations, as shown by the significance of NF2 (neurofibromin 2), TRAF7 (tumour necrosis factor [TNF] receptor-associated factor 7), KLF4 (Kruppel-like factor 4), AKT1, SMO (smoothened), PIK3CA (phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha), and POLR2 (RNA polymerase) [1].

About the Study

With a median SCNA rate of 12.3 percent and greater frequencies of chromothripsis (large chromosomal rearrangements), higher-grade meningiomas resembled several of the characteristics of other aggressive malignancies. Numerous mutations that had not before been reported were discovered in addition to the NF2, AKT1, and SMO prevalent mutations. Despite the fact that several of these genes have been linked to cancer in the past, it was unknown how they affected meningiomas, necessitating further research. Additionally, 8% of tumours displayed changes in epigenetic modifiers, which on a genomic level had not been investigated in meningiomas and may be crucial. As opposed to other research, which focused on low-grade meningiomas, this analysis looked at 134 high-grade meningiomas to compare the prevalence of NF2 mutation, chromosomal abnormalities, and other unique alterations with low-grade meningioma rates. either whole-exome or whole-genome sequencing [2]. Tumor suppressor p53, a crucial DNA-repair regulating protein, and defective DNA repair pathways were two mechanisms proposed to support the high degree of genomic disarray in highgrade meningiomas. Only 23% of mutations were shared generally across high-grade meningiomas, indicating significant heterogeneity of mutations. In several samples collected over time, heterogeneity was also seen within the specific tumours of particular patients. Genomic analysis of tumour specimens from patients who had multiple resections over time revealed that recurrent lesions had significantly different genomes from the previously resected lesions, indicating that recurrences were actually caused by distinct, unresected tumours rather than the parent tumor's residual tissue [3-5].

The parasagittal, falcine, torcular, and intraventricular areas had a higher likelihood of harbouring high-grade meningiomas than the skull base. even though there are fewer targets. A fascinating and emerging subject is the radiological evaluation of meningiomas to predict histology class, WHO grade, or genetic patterns. In predicting tumour behaviour, Coroller assessed 15 quantitative and 10 qualitative radiological parameters of 175 meningiomas; these variables were more reliable than clinical features. Higher-grade lesions were predicted by increased necrosis, intratumoral heterogeneity, non-sphere-like form, and bigger volumes. The aggressiveness of meningiomas has been predicted using diffusion MRI, MR elastography, and MR spectroscopy, among other cutting-edge imaging modalities. Genomic classification may be used in conjunction with imaging studies to influence surgical choice. A number of ongoing clinical trials are attempting to enhance therapy by focusing on a specific mutational class as a result of the introduction of meningioma genomics. Patients with residual or progressing meningioma are being enrolled by the National Cancer Institute and the Alliance for Clinical Trials in Oncology Group for testing for AKT1, SMO, or NF2 mutations and for therapy with AKT, SMO, or FAK inhibitors, respectively. Vismodegib, an inhibitor of SMO, has previously been licenced for the treatment of basal cell carcinoma and is presently being tested for the treatment of meningiomas with SMO mutations. Nivolumab, a BRAF inhibitor, as well as dual mTORC1 and mTORC2 inhibitor trials are now being started in meningiomas. There are also now being tested immunomodulating treatments, such as the pembrolizumab inhibitor of programmed cell death 1 [2].

Conclusion

It has been demonstrated that many mutations, including those in NF2, TRAF7, KLF4, AKT1, SMO, PI3KCA, and POLR2A, are significant. Genomic subtypes with implications for tumour location and a potential cancer stem cell source that resembles an embryo have been discovered. More genomic disarray and NF2 mutations were found in aggressive, high-grade meningiomas than in lower-grade tumours. Methylation patterns and six distinct subclasses have demonstrated superior patient outcome prediction compared to conventional WHO grading. The upcoming round of WHO tumour classification will probably be impacted by these genetic discoveries. Currently running clinical studies are starting to employ genetic subtype information to guide medication. In the end, it will be up to time to determine how, not if, these discoveries will be applied in clinical practise.

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