

Malignant Gliomas in Adults

TO THE EDITOR: In their Medical Progress article, Wen and Kesari (July 31 issue)¹ review the molecular pathogenesis of gliomas yet do not mention the role that DNA tumor viruses may play in tumor formation. In the same issue, Prins et al.² provide evidence that strongly supports prior research findings that link cytomegalovirus to glioma. Other DNA viruses, such as JC virus, have been implicated in glioma pathogenesis.³ In this regard, it is also important to note that detection of atypical nuclei and high-proliferation indexes in a brain biopsy led to, in at least one instance, the misdiagnosis of a grade III astrocytoma; subsequent polymerase-chain-reaction studies led to a diagnosis of progressive multifocal leukoencephalopathy related to JC virus.⁴ Further research is therefore needed to understand the link between DNA viruses and glioma.

Michael A. Meyer, M.D.

University of Buffalo
Buffalo, NY 14215
michaelandrewmeyer@yahoo.com

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THE AUTHORS REPLY: Because of space limitations, our review was largely restricted to information that was strongly supported by data. We agree that the studies regarding the potential role of

tumor viruses in the pathogenesis of malignant gliomas are intriguing. However, much more research is required to confirm such a link. Although some studies indicate the presence of cytomegalovirus in malignant gliomas,¹ other studies have failed to confirm these findings.^{2,3} In addition, the detection of viruses such as cytomegalovirus in malignant gliomas may not necessarily indicate that these viruses contributed to the transformation of the tumor. An alternative explanation could be reactivation of virus as a result of immunosuppression related to the tumor, corticosteroids, or chemotherapy.² With the advent of genomewide analytic tools and the recent advances characterizing the glioblastoma genome,⁴ we may now be able to address the issue of the presence of infectious agents in human cancer by using newer approaches, such as genomic subtraction technologies.⁵

Patrick Y. Wen, M.D.

Santosh Kesari, M.D., Ph.D.

Dana-Farber/Brigham and Women's Cancer Center
Boston, MA 02115
pwen@partners.org

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Case 20-2008: Abdominal Pain and Weakness after Gastric Bypass Surgery

TO THE EDITOR: In the discussion of the Case Record by Bonkovsky et al. (June 26 issue)¹ about a patient with porphyria who became symptomatic after gastric bypass surgery, there was no mention of the patient's nutritional care, which may have played a major contributory role. The persistent ketonuria before hospitalization indicates a daily carbohydrate intake of less than 100 g, where-

as the very low serum creatinine level on admission indicates limited meat intake and a marked reduction in skeletal muscle, reflecting severe protein-calorie malnutrition.² These findings are consistent with limited energy and protein intake postoperatively, which was at least 18 days before a subsequent hospital admission that was more than 5 weeks in duration. If the patient remained