Venous thromboembolism in glioblastoma

Venous thromboembolism encompasses deep venous thrombosis (DVT) and pulmonary embolism (PE). The exact mechanism of development of VTE in patients with glioblastomas (GB) is unclear, but predisposing factors include hemiparetic limbs, chemotherapy, older age, and corticosteroid therapy [1)].

Because of the possibility of intracranial hemorrhage (ICH), there is reluctance to treat patients who have GB with anticoagulants. However, the incidence of ICH in patients who receive anticoagulation is approximately 2%, which is not significantly different from the incidence in patients with brain tumors who do not receive anticoagulants [2)].

Chemical prophylaxis of VTE during the postneurosurgical period remains one of the major dilemmas in modern neurosurgical practice due to a potential increased risk of devastating intracranial hemorrhage in the setting of anticoagulation [3)].

Despite a low incidence of intracerebral hemorrhage (ICH), even with anticoagulation therapy, physicians are reluctant to administer anticoagulants to GB patients with VTE. The optimum treatment for GB patients with VTE has not been established; however, the benefits of anticoagulation therapy may outweigh the risks of ICH. A prospective clinical trial to evaluate the potential benefits for deep venous thrombosis prophylaxis in GB patients is warranted [4)].

Case series

Four hundred fourteen glioblastoma patients with isocitrate dehydrogenase (IDH) wild type status were identified. VTE was documented in 65 patients (15.7%). The median time from tumor diagnosis to the occurrence of VTE was 1.8 months, and 27 patients were diagnosed with VTE postoperatively (within 35 days) (42.2%). History of prior VTE was more common in patients who developed VTE than in those who did not (p=0.004). Bevacizumab treatment at any time during the disease course was not associated with the occurrence of VTE (p=0.593). Most patients with VTE (N=61, 93.8%) were treated with therapeutic anticoagulation. Complications occurred in 14 patients (23.0%), mainly intracranial hemorrhages (N=7, 11.5%). Overall survival did not differ between patients diagnosed with VTE and those who had no VTE (p=0.139). Tumor progression was the major reason for death (N=283, 90.7%), and only 3 patients (1.0%) died associated with acute VTE.

VTE occurred early in the disease course, suggesting that the implementation of primary venous thromboembolism prophylaxis during first-line chemoradiotherapy could be explored in a randomized setting [5)].

1)
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2)
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4)

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5)

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