Intratumoral hemorrhage

Etiology

Intratumoral hemorrhage is thought to originate from abnormal newborn vessels that traverse necrosis areas [1] or from tumoral invasion of large vessels [2] , leading to thinning and rupture of the vessels walls. Another potential mechanism would be relatively weak tumor vessels, which are not well invested with a glial meshwork; this may contribute to reduced resistance to the shearing forces of the brain [3]. Endothelial proliferation with subsequent obliteration of the lumen or presence of intratumoral arteriovenous fistulae are alternate explanations for intratumoral bleeding [4]. Thus, hemostasis often cannot be easily achieved at hematoma removal; this finding indicates the possibility of brain tumors as a cause of bleeding [5].

Epidemiology

There have been known risk factors of intratumoral hemorrhage. Hemorrhage more often develops in malignant tumors such as glioblastoma multiforme and brain metastasis [6] [7] [8]. The incidence of tumor bleeding in malignant astrocytoma in one study was 6% [9] while that in glioblastoma and metastatic brain tumors were 6.5-8% and 7-9%, respectively). Among benign neuroepithelial tumors, the incidence of hemorrhage from mixed glioma and oligodendroglioma was much higher than the other tumors [10] [11]. On the other hand, pituitary neuroendocrine tumor and meningiomas have the high risk for developing intratumoral hemorrhage among benign non-neuroepithelial tumors [12]. The location of bleeding depends on the different site of brain tumor even though intratumoral hemorrhage usually develops in the atypical location of hypertensive intracerebral hemorrhage and the patients often have no history of hypertension [13] [14].

Intratumoral hemorrhage occurs most frequently in pituitary neuroendocrine tumor among all types of brain tumors [15].

Diagnosis

Radiological studies with contrast material usually distinguish tumors from hemorrhage, as the border between the tumors and hemorrhage is usually clear [16]. In contrast, if the tumors are compressed by a large hemorrhage, or the border between the tumors and hemorrhage is unclear, intratumoral hemorrhage may be indistinguishable from spontaneous ICH, even though contrast material is used [17]. Thus, a CT with contrast cannot exclude underlying pathologies that may cause ICH, especially if the patient has a history of hypertension, and the location is typical for hypertensive ICH. MRI with gadolinium in the early follow-up period would likely have lead to earlier detection of the tumors in the present case. However, Inamasu et al. [18] suggested that in terms of cost effectiveness, it is controversial to have every patient presenting with typical hypertensive ICH undergo MRI with gadolinium to rule out intratumoral bleeding.

Case reports

A 63-year-old gentleman who had right-side severe sensorineural hearing loss on MRI showing a right cerebellopontine angle tumor (volume 4.96 cm3) with an internal acoustic meatus extension. He underwent GKRS with the prescription dose of 12 Gy to the 50% isodose line, covering 4.66 cm3 (i.e., 94%) of the tumor. Ten days later, he experienced a symptomatic intra-lesional hemorrhage with a mass effect over the brainstem. When symptoms did not resolve after an initial conservative approach, he underwent surgical decompression of the lesion. Postoperatively, the patient had facial palsy but was free of disabling vertigo and ataxia. At the 6-month follow-up, he was doing well without any other complaints. Although rare, an intralesional bleed can occur after GKRS in Vestibular Schwannoma and should be suspected when new severe symptoms develop immediately after therapy [19].

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