

Letter to the Editor

Squamous cell carcinoma of the scalp causing cortical venous thrombosis and Intraparenchymal hematoma



Dear Editor,

We present a case of squamous cell carcinoma (SCC) of the scalp with intracranial invasion causing cortical venous thrombosis and symptomatic intraparenchymal hemorrhage. We discuss the factors influencing operative versus supportive management that arise in this unique situation.

A 74-year-old man presented with altered mental status of unknown duration. He had undergone a Mohs procedure for resection of SCC of the frontal scalp with negative margins at an outside hospital ten months prior, and was lost to follow-up before pursuing further treatment. History was notable for hypertension, diabetes, mild diastolic heart failure, chronic kidney disease, and prior smoking. Examination disclosed dense expressive aphasia, mild right upper extremity pronator drift, and a 8 x 7 cm exophytic mass arising from the left frontal scalp. Laboratory studies were notable for fever, leukocytosis, and acute renal failure.

Noncontrast computed tomography (CT) of the head revealed a large intraparenchymal hematoma in the left frontal lobe with associated mass effect (Fig. 1A, B). Magnetic resonance imaging (MRI) showed the extracranial enhancing mass invading through the frontal bone to the level of the meninges, with dural enhancement suggestive of dural invasion or reactive change (Fig. 1C-E). The intraparenchymal hematoma was subjacent to the mass, with surrounding T2 hyperintensity and diffusion restriction. Curvilinear sulcal enhancement along the hemorrhage suggested congestive vascular changes (Fig. 2A, B). MR venogram demonstrated patency of the superior sagittal sinus (Fig. 2D, E). These findings suggested a cortical venous thrombosis leading to intraparenchymal hemorrhage with possible surrounding vasogenic and cytotoxic edema.

Staging workup showed parotid gland lymphadenopathy concerning for metastasis, without other systemic sites of involvement. Anticoagulation was withheld due to the invasive mass and possibility of surgical intervention being required to relieve intracranial hypertension. Fluid resuscitation, broad-spectrum antibiotics and hyperosmolar therapy were undertaken. Although his mental status and aphasia improved, he required additional treatment for pulmonary edema and hypoxic respiratory failure. Interdisciplinary discussion between the plastic and reconstructive surgery, neurosurgery, otolaryngology, oncology and palliative care teams was undertaken. Wide local excision of extracranial disease with calvarium and scalp reconstruction was felt to be technically achievable. However, meaningful oncologic resection was deemed unlikely due to the extensive intracranial invasion with unclear margins and metastatic focus. Prognosis was felt to be guarded even with multimodal treatment due to the patient's advanced age and multiple comorbidities. A consensus recommendation to pursue palliative treatment instead of aggressive surgical resection was offered. The patient's family agreed, and after one week he returned to his outside oncology providers in medically

stable condition for further treatment.

SCC represents approximately 16% of scalp tumors [1]. Overall, SCC is significantly more common in the oropharyngeal mucosa than the skin. Whereas smoking is a risk factor for mucosal lesions, risk factors for developing cutaneous SCC include age, ultraviolet light exposure, chronic scarring, history of ionizing radiation, androgenetic alopecia (in men), and immunosuppression. The rates of bone invasion in cutaneous SCC have not been well established in the literature. However, in one retrospective study, 20 out of 53 immunocompromised patients with cutaneous SCC of the scalp were found to have bone invasion [2]. Bone invasion is associated with poor prognosis [3].

First line therapy for localized cutaneous SCC is surgical excision via either standard excision, curettage and electrodesiccation (C&E), or Mohs micrographic surgery (MMS) [4]. Radiotherapy is an effective adjuvant for tumor control in select patients [4]. The five-year local recurrence rate for primary cutaneous SCC following MMS was 3.1% compared with 3.7% for C&E and 8.1% for standard excision [5]. Success rates for combined surgical excision and radiotherapy are not well established. Treatment of invasive or metastatic cutaneous SCC may include surgical resection with or without adjuvant radiotherapy, epidermal growth factor inhibitors, or cisplatin-based chemotherapy; these strategies are primarily based on retrospective data, owing to the rarity of metastatic disease [4].

Cortical vein thrombosis (CoVT) has been most frequently described in association with thrombosis of the dural venous sinuses and/or other large cerebral veins. Isolated CoVT has been reported only in case reports, due to the rarity of the condition. In a systematic review of isolated CoVT, Coutinho and colleagues reported that the majority of patients are women, with a mean age of 41 years. Common risk factors include pregnancy, oral contraceptive use, or infection; approximately 5% of patients have associated systemic malignancy leading to a hypercoagulable condition [6]. Workup for these risk factors upon initial history and physical examination is critical, particularly to exclude conditions such as meningitis that require urgent treatment. Diagnosis is challenging due to the small caliber and variable, often asymmetric anatomy of cortical veins. Cerebral angiography remains the gold standard imaging modality, though sensitivity and specificity are unknown; MRI or CT venography may also be employed [7]. Noncontrast MR venography was utilized in lieu of contrast-enhanced CT angiography or venography in this case due to the patient's renal failure. To our knowledge, CoVT due to an invasive extracranial mass has not been previously reported.

CoVT can lead to intraparenchymal hemorrhage in close to 50% of cases, localized parenchymal edema in approximately one-third of cases, and localized subarachnoid hemorrhage in a minority of cases. Symptoms may include headache, focal neurological deficits or seizures [6,9,10]. Treatment of CoVT typically involves anticoagulation with heparin followed by oral anticoagulation; this is not specific for CoVT,

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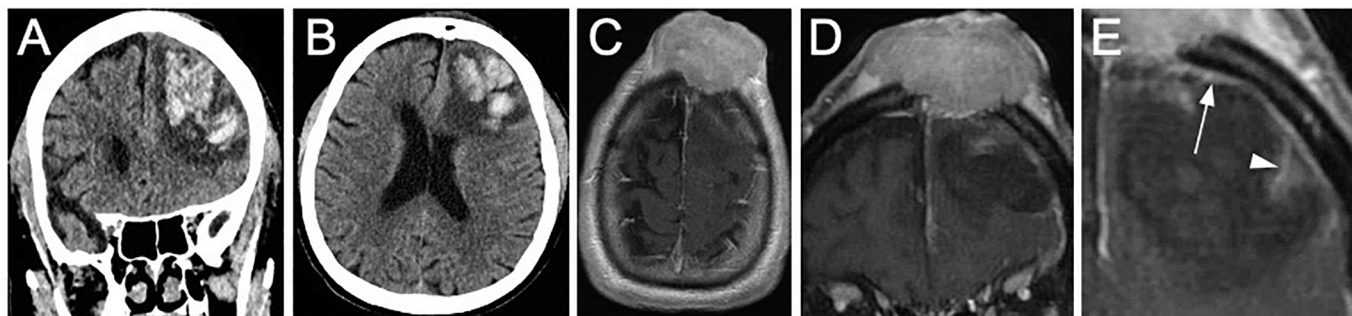


Fig. 1. (A, B) Noncontrast computed tomography scan of the head in the coronal (A) and axial (B) planes shows a large mixed density intraparenchymal hematoma with surrounding edema and mass effect. (C-E) T1-weighted gadolinium-enhanced magnetic resonance images of the brain in the axial (C) and coronal (D, E) planes shows a large, well-circumscribed extracranial mass eroding through the frontal bone to the level of the meninges, adjacent to the hematoma. Enhancement of the dura adjacent to the mass (arrow) and sulci surrounding the hematoma (arrowhead) is noted.

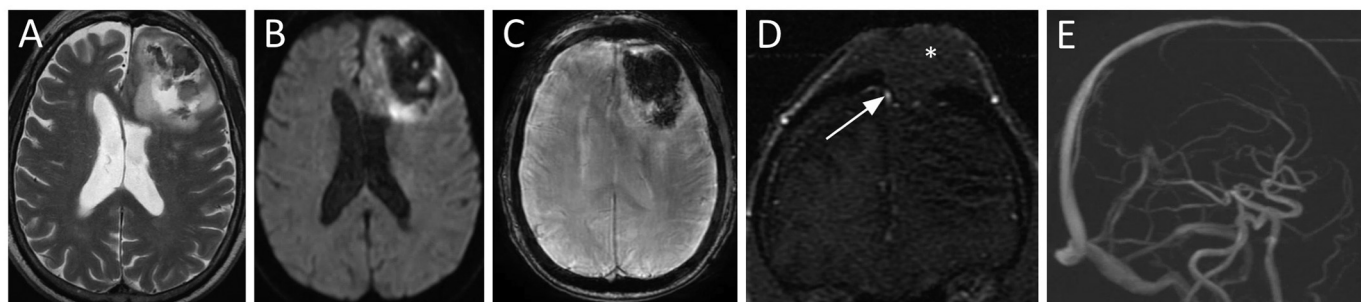


Fig. 2. (A, B) Axial noncontrast MRI T2-weighted (A) and diffusion-weighted (B) images show hyperattenuation around the hematoma suggestive of vasogenic and cytotoxic edema, respectively. (C) Axial MRI susceptibility-weighted angiography (SWAN) sequence shows high magnetic susceptibility consistent with hemorrhage. (D, E) Magnestic resonance venogram coronal two-dimensional time-of-flight (D) and oblique three-dimensional reconstruction (E) show patency of the superior sagittal sinus (arrows) along its entire course, including adjacent to the invasive mass (asterisk).

but rather derived from venous sinus and cerebral vein thrombosis treatment algorithms [8,9]. Treatment must also be tailored to the specific clinical circumstances and associated pathologies. Management of associated intracranial hypertension may include both medical measures and decompressive surgery in refractory cases [6,9,10].

In conclusion, we describe a unique case of recurrent invasive cutaneous SCC causing isolated CoVT. This case highlights the need for close follow-up and adjuvant radiotherapy in high-risk cutaneous SCC cases, as well as diagnostic pearls and management considerations for the rare entity of isolated CoVT.

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