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Case Report

Imaging findings in the progression of a giant cell glioblastoma

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ABSTRACT

First described in 1909, giant cell glioblastoma (GC) is a histologic variant of glioblastoma multiforme (GBM) that accounts for 1% of cases of primary GBM. It is characterized by a predominance of bizarre giant cells with abundant eosinophilic cytoplasm, and may portend an improved prognosis over classic GBM. Due to the rarity of GC, there is a paucity of reports that describe its associated radiologic findings. This case report chronicles the progression of a GC that was incidentally discovered in a 74-year-old male with coincident subdural hematoma and empyema. Serial brain imaging was obtained as part of this patient's continued work-up that documents the radiologic characteristics of the GC over a period of months. To our knowledge, this manuscript is the most extensive radiologic documentation of the progression of GC to date.

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Introduction

Giant cell glioblastoma (GC) is a rare histologic variant of glioblastoma multiforme (GBM) characterized by a predominance of bizarre multinucleated giant cells with abundant eosinophilic cytoplasm [1]. It was first described as a “monstrocellular” tumor by Schmincke in 1909, and has since been reported to account for 1% of cases of primary GBM [2]. GC may portend an improved prognosis over GBM [2]. Yet, despite having been first described in the literature over 100 years ago, GC has been incompletely characterized to date due to its rarity. We present a case of GC coincident with a subdural hematoma

and empyema that radiologically details the development of this rare neoplasm.

Case report

A 74-year-old male presented to the ED with sudden onset altered mental status and aphasia. His medical history was notable for hypertension, atrial fibrillation for which he was on warfarin, and recurrent oral cavity squamous cell carcinoma (OSCC) for which he had undergone right mandibulectomy with neck dissection and a partial glossectomy. The patient had a seizure in the ED and was admitted to the ICU for further workup. On hospital day (HD) 2, a noncontrast MRI

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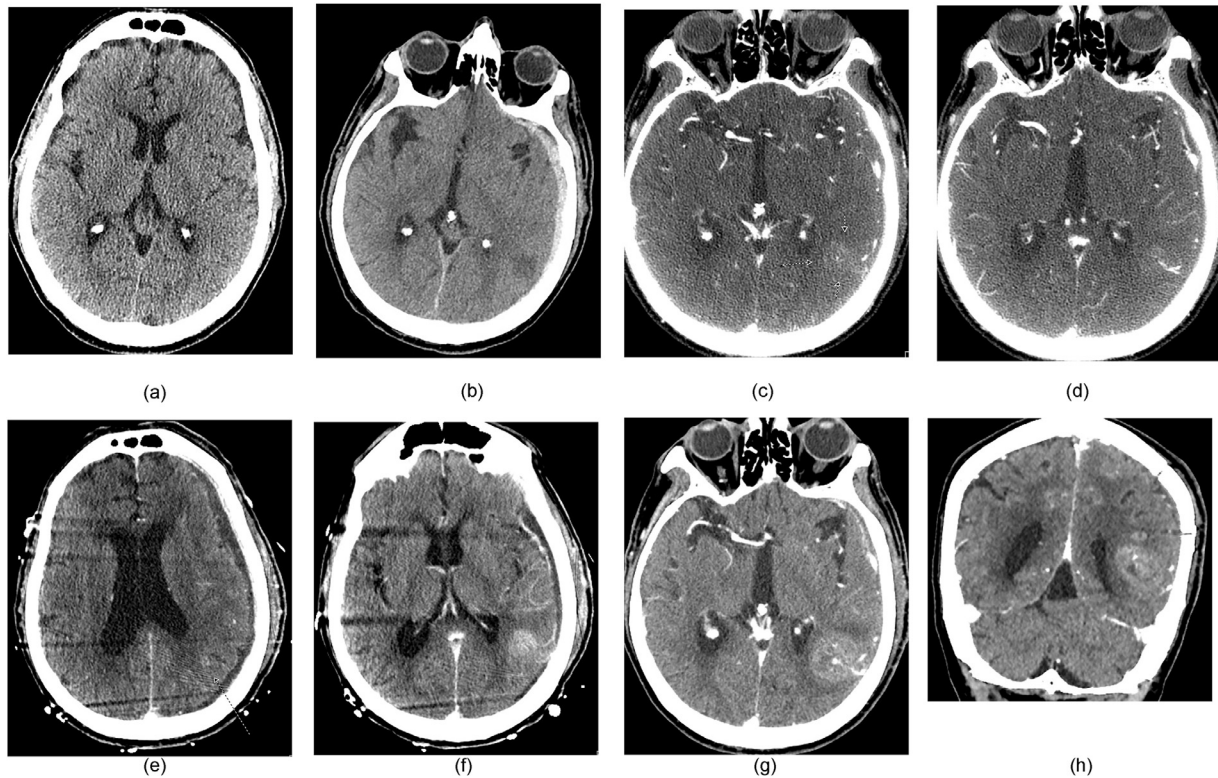


Fig. 1 – (a) Initial presentation. CT head in ED shows no abnormality. (b) Initial presentation, hospital day 2. CT head shows acute subdural and temporal hypodensity corresponding to FLAIR hyperintensity. (c and d) 47 days after initial presentation. CTA head obtained for new stroke-like symptoms demonstrates subtle enhancement in this region. There is a central vascular structure running through this area. (e and f) 48 days after initial presentation. CECT obtained for worsening AMS. Purulent drainage from burr hole. MRI was difficult to arrange due to pacemaker. CT head shows enhancing lesion in left posterior superior temporal lobe and additional small satellite focus. (g and h) 78 days after initial presentation. CECT shows likely recurrent subdural empyema and enlarging temporal cortical lesion with prominent central vessel.

brain showed a left convexity subdural hematoma as well as a 1.7 cm rounded focus of transcortical T2/FLAIR hyperintensity in the left temporal lobe demonstrating central susceptibility signal loss, initially thought to represent a venous infarct. Post contrast MRI sequences for further characterization of this lesion showed linear gyriform enhancement and internal microhemorrhage, which supported a diagnosis of subacute venous infarct. The subdural was managed conservatively and the patient was discharged home after 1 week. CTs 1 and 2 weeks out from discharge showed a relatively stable subdural.

Three weeks after discharge (28 days after initial presentation), the patient re-presented to the ED from neurology clinic for acute change in mental status. A noncontrast CT (NECT) showed expansion of the subdural hematoma to 1.9 cm with increased midline shift. Neurosurgery evacuated the hematoma via 2 burr holes and placed a subdural drain, and the patient was discharged to a skilled nursing facility (SNF) 1 week later. The temporal lesion was not visualized or poorly visualized on all the CT scans performed over this time period.

The patient represented a little over a week later (47 days after initial presentation) after a fall at his SNF and was noted

to have facial droop and pronator drift. A NECT showed a new hyperdense collection tracking from the patient's parietal burr hole. The incision over this burr hole was also draining a small amount of purulent fluid raising concern for empyema. A second NECT the same day was unchanged from the first with regard to the parietal collection, but demonstrated interval development of a hyperdense collection in the left posterior temporal region corresponding to the area of hypodensity identified on the CT from his first admission. The region measured up to 3 cm and was concerning for a hemorrhagic transformation of what was still presumed to be a venous infarction. On hospital day 1, additional NECT scans showed slight posterior expansion of the parietal collection and a third contrast-enhanced CT (CECT) showed slight enhancement of the temporal lesion, suggesting blood-brain barrier breakdown, as well as a 2-mm area of satellite enhancement immediately superior to the lesion. On HD 3, contrast-enhanced MRI (CEMRI) confirmed a subdural empyema and demonstrated the temporal lesion as a hyperintense T2/FLAIR signal with enhancement and anterior peripheral restricted diffusion. Concern was raised in the MRI interpretation that, while this area could represent a venous infarct, a hemorrhagic metastasis could also have this appearance. A craniotomy was performed for

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