Case Report

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Extracranial Glioblastoma Metastasis to The Parotid Gland: Illustrative Case with Review of Literature

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ABSTRACT

Background: Extracranial Glioblastoma (GBM) metastasis is rare but has been reported in the lungs, lymph nodes and vertebrae. Herein, we present a case of GBM metastasizing to the parotid gland.

Observations: A 65-year-old male with a left temporal lobe GBM underwent standard treatment protocol. Five months after resection, a new subgaleal metastasis was resected despite no primary tumor recurrence. Seven months later, he returned with a newly developed left-sided facial nerve palsy, swollen parotid gland and palpable upper jugular group lymph nodes. The parotid gland mass was initially treated for infectious parotitis but enlarged and a subsequent biopsy revealed extracranial GBM. Considering his good treatment response to the primary GBM and high-performance status (Karnofsky 90%), we partially removed the tumor for cytoreduction and released the facial nerve. The patient underwent an uneventful surgery; however, he expired 2 weeks later from status epilepticus. Seventeen other cases of GBM metastasis to the parotid gland have been reported in the literature.

Conclusion: Younger age and prior surgical resection are risk factors for developing extracranial GBM metastasis. Prognosis for these patients is poor, but early diagnosis and aggressive treatment should be considered, especially in patients with high performance status and stable primary disease.

Keywords: Glioblastoma, Extracranial metastasis, Parotid gland

Abbreviations: GBM: Glioblastoma Multiforme; MRI: Magnetic Resonance Imaging; CNS: Central Nervus System; BBB: Blood Brain Barrier

1. Introduction

Glioblastoma (GBM) represents the most common and aggressive type of primary malignant brain tumor, accounting for roughly 16% of all cases[1]. Despite advances in therapeutic approaches, the prognosis of GBM remains poor at a median survival of 12 to 14 months[2]. Extracranial metastasis of GBM is rare, reported in only 0.4% to 2% of patients with GBM[3]. Metastatic lesions have been reported in various organ systems in the body but more prominent in the lungs, lymph nodes and vertebrae[4]. In this study, we report the case of a patient diagnosed with GBM metastasizing to the parotid gland along with a comprehensive review of these cases in the literature. We also provide an up-to-date review on the factors and mechanisms behind extracranial GBM metastasis.

2. Illustrative Case

Here we report a case of a 65-year-old male patient presented with diffuse headaches that had progressively worsened over the prior three weeks, along with morning episodes of vomiting and confusion. Neurological evaluation showed a positive pronator drift of the right arm. Magnetic Resonance Imaging (MRI) of the brain revealed an intra-axial lesion of the left temporal lobe with a cystic component and marked vasogenic edema, as well as heterogeneous contrast enhancement on T1 post-contrast sequence, suspicious for a high-grade glioma (Figure 1A and B). The patient underwent resection of the lesion through a left pterional craniotomy. A xanthochromic fluid was aspirated from the cyst within the temporal lobe and a gray to pink hemorrhagic capsule with a pseudo-cleavage plane was removed. Postoperative imaging confirmed gross total resection (Figure 1C and D). Pathological analysis of the tumor sample showed anaplastic cells associated with microvascular proliferation and necrosis. Immunochemistry confirmed glial-cell lineage GFAP positive. The patient underwent a standard treatment protocol for GBM, including adjuvant radiotherapy and concomitant chemotherapy with temozolomide[5].

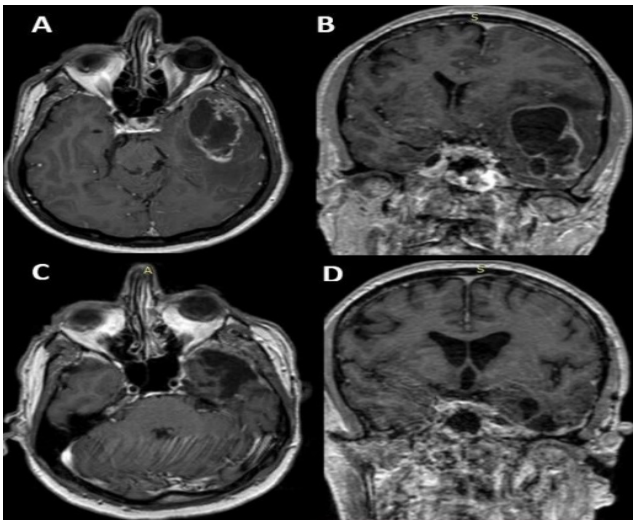

Figure 1: (A) Axial and (B) coronal T1 post-contrast MRI sequences showed a left temporal mass with a cystic component demonstrating heterogeneous contrast enhancement. (C) Axial and (D) coronal T1 post-contrast MRI sequences show gross total resection of the lesion and no recurrence at 1 month postoperatively.

Five months after resection, the patient presented to our clinic with concerns of a new lump on the left scalp. The lump was round with a hard consistency, mildly painful and non-mobile and fixed to the scalp. MRI of the brain showed a subgaleal lesion that was concerning for metastatic tumor seeding versus inflammation (Figure 2A and B). The rest of the imaging was unremarkable, showing expected post-surgical changes without evidence of tumor recurrence. The scalp mass was followed clinically for three weeks, however it was expanding fast, as such the decision was made to proceed with resection. Intra-operatively, the mass was found to be subgaleal and was resected without complications (Figure 2C). Pathological analysis confirmed the lesion to be an extracranial GBM (Figure 3).

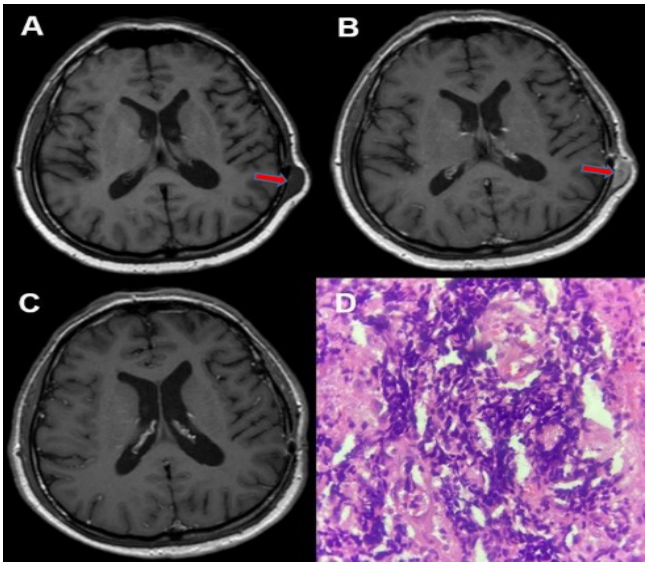

Figure 2: (A) Axial T1 pre- and (B) post-contrast MRI sequences showing a subgaleal lesion located on the left parietal region demonstrating marked contrast enhancement. Physical exam located a lump on the left parietal region with hardy-stone consistency. (C) Postoperative axial T1 post-contrast MRI sequence imaging confirming gross total resection. (D) Microscopic findings of pseudopalisading necrosis from the lesion, consistent with GBM.

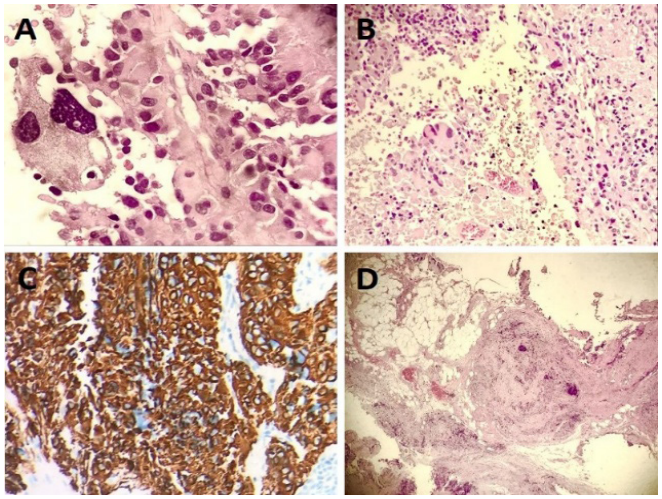

Figure 3: Microscopic and immunohistocytochemistry pathology analysis of the subgaleal metastasis (40x magnification). (A) H&E stains showing fibro fatty tissue being infiltrated by a tumor composed of tumor cells showing moderate and focal marked nuclear pleomorphism, (B) as well as areas of tumor necrosis and mitotic figures. Immunohistochemistry showed the tumor cells to be (C) positive for GFAP and (D) (E) necrosis, consistent with a diagnosis of glioblastoma.

The patient remained clinically and radiologically stable for another 7 months, after which he returned for evaluation of newly developed facial nerve palsy and swelling on his neck. On examination the patient had developed a House-Brackmann grade IV facial nerve palsy on the left. A swollen parotid gland along with palpable upper jugular group lymph nodes were found on the left (Figure 4). Palpation of the parotid gland revealed a hard, non-mobile mass. He was initially treated for infectious parotitis, however there was continued enlargement of the mass. A biopsy of the lesion was performed, which returned positive for GBM, indicating metastatic spread to the parotid gland.

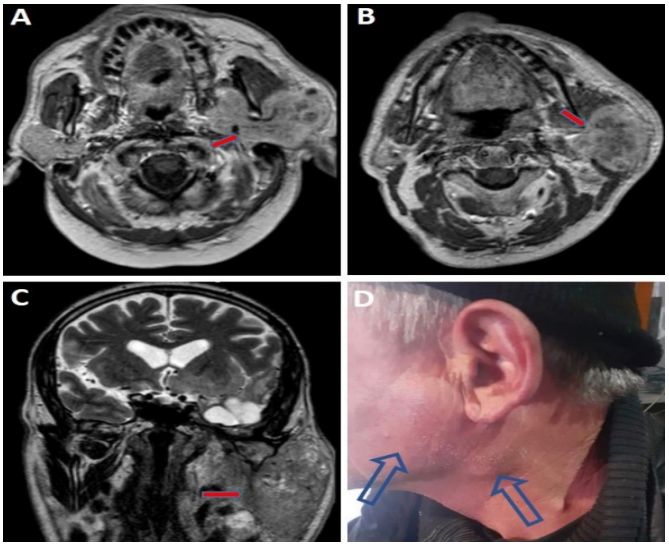

Figure 4: (A, B) Axial and (C) coronal T1 post-contrast MRI sequences showing the lesion infiltrating the left parotid gland with extension into the pterygopalatine fossa. (D) A photograph of the patient shows a diffuse swollen parotid gland along with submandibular and palpable lymph nodes.

We discussed the case with our Oncology board and considering the patient's good treatment response to the primary GBM and his high-performance status (Karnofsky 90%), we decided to partially remove the tumor for cytoreduction and to release the facial nerve causing the peripheral facial palsy. The patient underwent an uneventful surgery and was discharged home on postoperative day 2. Unfortunately, the patient had a re-admission at a later date and eventually expired.

3. Discussion

Extracranial metastasis of GBM is very rare, with a reported incidence in the literature ranging between 0.4 and 2%[3]. The first case was published in 1928 by Davis et al.[6] Published in 2006, the most comprehensive review to date reported 128 cases of GBM extracranial metastasis, with even more reports published since then[7]. Overall, 70% of reported cases are males, with a mean age at presentation of 38 years old[4]. Metastasis has been shown to be more common in the lungs, lymph nodes, vertebrae and liver[8] and rarely in soft tissues[9]. Among the lymph node metastases, 62% are cervical and often ipsilateral, although bilateral have also been reported[10]. GBM spreading to the parotid gland has also been reported in the literature. To our knowledge, including the case from the current study, there are only 18 reported cases of GBM metastasis to the parotid gland (Table 1)[3,11-25].

From our review of the literature, we found that the average age of patients diagnosed with GBM metastasis to the parotid gland was 46 years old, 72% of which were male. The primary site of GBM was the temporal lobe in 55% of patients and distant metastases were found to be ipsilateral in 16 out of 18 patients. These demographics are well correlated with prior literature reports of patients with GBM metastasis[7]. No clear association was found between the progression of the primary GBM and the extent of the metastatic disease. In our patient, the primary GBM remained stable despite progression of the metastasis mass. The reported median overall survival starting from the diagnosis of the metastasis varies widely, ranging from 5 to 16 months[26]. Overall, studies have shown that the prognosis when GBM metastasizes to the neck is better when compared to metastatic disease to the lung and liver[7]. This could be explained by the cosmetic nature of enlarging neck masses, leading to an earlier notice by the patient and the accessibility of these lesions for tissue biopsy, facilitating prompt diagnosis and a faster pathway to potential treatment. Biopsy of parotid gland masses can be achieved using different methods, including parotid or cervical lymph node biopsy, fine needle aspiration cytology or even parotidectomy.

Table 1: Reported Cases of GBM Metastasis to the Parotid Gland.

Case No.	Publication	Age	Sex	Location of Primary Intracranial Lesion		Location of Metastatic Lesion
				Lobe	Side	
1	Taha, et al.[11]	33	M	Frontal	L	Ipsilateral
2	Alhoulaiby, et al.[12]	53	M	Temporal	L	Ipsilateral
3	Romero-Rojas, et al.[13]	26	M	Frontal	L	Ipsilateral
4	Kraft, et al.[14]	58	M	Temporal	R	Ipsilateral
5	Moghtader[15]	64	M	Temporal	L	Ipsilateral
6	Ogungbo, et al.[16]	49	F	Multifocal	--	Ipsilateral
7	Waite, et al.[17]	40	M	Frontal	L	Ipsilateral
8	Swinnen, et al.[18]	56	F	Temporal	R	Ipsilateral
9	Taskapilioglu, et al.[19]	30	F	Frontal	L	Contralateral
10	Jie, et al.[20]	47	M	Temporal	R	Ipsilateral
11	Schwock, et al.[21]	52	M	Temporal	L	Ipsilateral
12		42	M	Temporal	L	Ipsilateral
13	Dolman[22]	35	F	Frontal	L	Ipsilateral
14	Park, et al.[25]	25	F	Frontal	R	--
15	Kuhn, et al.[23]	58	M	--	R	Ipsilateral
16	Hamilton, et al.[3]	24	M	Temporal	L	Ipsilateral
17	Frade Porto, et al.[24]	72	M	Temporal	R	Ipsilateral
18	Current case	65	M	Temporal	L	Ipsilateral

The mechanism of extracranial metastasis of primary Central Nervus System (CNS) cancers, such as GBM, is not well understood. Potential mechanisms of distal metastasis include spreading via a lymphatic cerebrospinal fluid drainage system, hematogenous spread or direct invasion through the dura and bone[27]. Risk factors such as age at diagnosis, longer patient lifespan and prior surgical intervention have also been previously discussed. As younger patients have overall better outcomes of GBM than older patients, an increased prevalence of GBM metastasis could be due to a longer lifespan. Moreover, we found that 96% of patients diagnosed with extracranial GBM metastasis had undergone surgery. The longer lifespan in operated patients is likely a result of the benefits provided by surgical resection of GBM, however the possibility of postoperative extracranial extension due to dural violation cannot be excluded. In the case of metastasis to the parotid gland and lymph nodes, tumor spread via the lymphatic system must be considered. However, the central nervous system does not have a proper lymphatic system and the majority of studies reporting lymphatic propagation of GBM occurred in the setting of a prior surgical resection in those patients[28,29].

Hematogenous spread due to a damaged Blood Brain Barrier (BBB) could also facilitate GBM metastatic spread to the parotid gland. Angiogenesis and disruption of the Blood Brain Barrier (BBB) is a potential mechanism for distant metastasis, especially considering that GBM metastasis has been reported in non-operated patients[30,31]. These new vessels are hyperpermeable, lack normal connective tissue and in the presence of high levels of VEGF and matrix metalloproteinases, can facilitate infiltration by tumor cells[32]. In fact, matrix metalloproteinases gelatinase-A (MMP-2) and gelatinase-B (MMP-9) are highly expressed in high grade malignant glioma[33]. Furthermore, a study with 141 patients diagnosed with GBM showed the presence of circulating tumor cells in peripheral blood in 20% of the patients[34]. New diagnostic techniques such as liquid biopsy could prove helpful in early diagnosis of this patient population.

Genetic factors also likely contribute to the extracranial metastasis of GBM. Genetic studies have postulated that a subclone of the primary tumor may have spreading capabilities, especially those with mutations of TP53.[25] Molecular and genetic features such as YKL-40 glycoprotein, lack of EGFR amplification with non-mutant IDH1/2 and absent c-Met upregulation have been previously reported in GBM metastatic lesions[35]. Another study showed gains in chromosomes 3 and 9 and losses in chromosomes 4, 10 and 11 in a patient with osseous GBM metastasis, which altered the immunogenicity of tumor cells by allowing them to avoid specific T-cell responses[36].

4. Conclusion

Extracranial metastasis of GBM to the parotid gland is rare but not uncommon. GBM metastasis should be considered in patients even if the primary disease remains stable. New neck masses in patients with history of GBM require prompt diagnosis with tissue biopsy. Younger age and prior surgical resection are possibly the most important risk factors for developing a metastatic lesion. Prognosis in this patient population is likely poor, but early diagnosis and aggressive treatment should be considered, especially in patients with high performance status and stable primary disease. Further studies are needed to subgroup patients at risk for GBM metastatic disease. Techniques such as liquid biopsy may become helpful in the near future for diagnostic and monitoring purposes.

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