

A rare case report of metastasis to duraplasty site in a patient with esophageal carcinoma

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ABSTRACT

A 76-year old man presented with dysphagia, decreased response and generalised weakness. He had acute subdural hematoma two years back and decompressive craniectomy was done for that. After one year, he had poorly differentiated carcinoma of esophagus with multiple metastases in right supraclavicular lymph node, lung, liver, and left ilium. The stage was T3N1M1 and grade was G3. Contrast enhanced computed tomography brain showed an enhancing extraaxial swelling in the craniectomy site, compressing the brain. He went into coma. He was ventilated. Surgical decompression was done. Fleishy pink growth in the plane of duraplasty was excised. He developed myocardial infarction and succumbed. Histopathological result came as metastasis form adenocarcinoma. Immunohistochemistry showed strong membrane positivity for Pan-cytokeratin. This is the first report of a metastasis in duraplasty done for an earlier surgery, although there are lots of reports of metastases in duramater.

Abbreviations

BBB	Blood- brain barrier
BiPAP	Bilevel positive airway pressure
ECOG	Eastern cooperative oncology group
CECT	Contrast enhanced computed tomography
PRBC	Packed red blood cells
FFP	Fresh frozen plasma
ECG	Electrocardiogram
EF	Ejection fraction
BP	Blood pressure
HPR	Histopathological result
Pan-CK	Pan-Cytokeratin
IHC	Immunohistochemistry

Introduction

Incidence of metastases in dura mater in patients with advanced malignancy is 8–9 % [1–3]. The malignancies which commonly metastasize to the dura are the cancers of the breast, prostate, lung, kidney, thyroid, gastrointestinal tract, and head and neck, and hematologic malignancies such as lymphoma and leukemia. The methods of spread are direct extension from skull metastases or haematogeneous spread.

Extension from the skull metastasis is into the epidural space. Haematogenous spread is usually into the subdural space. The presentations are progressive neurological deficits, subdural hematomas, and subdural fluid collection [1–10]. The subdural haemorrhage is presumably due to obstruction of dural vessels by neoplastic cells. The presenting symptoms are headache, multiple cranial neuropathies, aphasia, hemianopia, focal weakness, sensory loss, mental symptoms and or convulsions [1–3]. Headache may be due to traction on the dura, invasion of venous sinuses, or raised intracranial pressure. Sensorimotor deficits arise from either compression of brain or brain invasion. Radiological findings may mimic a meningioma or subdural hematoma [1,2,11–17]. The imaging findings may be a single nodular or biconvex dural lesion, localised thickening, diffuse dural involvement, dural tail, vasogenic edema, brain invasion, and or involvement of venous sinuses [2]. Rapid invasion and progression of symptoms can occur [3]. The treatment modalities are surgical removal and chemotherapy [1,2]. The chance of survival is poor, because of advanced malignancy. But the response to chemotherapy is better than brain or leptomeningeal metastases, because the dural metastases are outside the blood- brain barrier (BBB). Currently there is no report of metastasis in duraplasty done for other surgeries.

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Case report

A 76-year old man was admitted with dysphagia, seizure, decreased response, and generalised weakness. He had acute subdural hematoma two years back, after falling at home. He underwent decompressive craniectomy, evacuation of hematoma, and duraplasty. After one year, he had dysphagia. There was no history of malignancy in the family. Endoscopy showed neoplastic polypoidal growth in the lower esophagus. Biopsy was reported as poorly differentiated carcinoma. Chemoradiotherapy was done as he had metastases in right supraclavicular lymph node, lung, liver, and left ilium. The stage was T3N1M1 and grade was G3. On examination he had dyspnea, excessive secretions, poor feeding, fatigue and altered sensorium. He was on bilevel positive airway pressure (BiPAP) ventilation. Eastern cooperative oncology group (ECOG) performance status was 4 with complete disability.

Contrast enhanced computed tomography (CECT) brain showed left subdural hematoma, and extra axial swelling in the craniectomy site with enhancement of contrast, and compression of brain, suggestive of metastasis (Fig. 1a,b,c,d).

The initial plan was to continue palliative care in view of advanced malignancy. He was given conservative supportive care with antiedema and anticonvulsant medications. But he had progressive deterioration into coma. So, surgical decompression was planned at high risk. He had anaemia, thrombocytopenia, and elevated prothrombin time (Haemoglobin-6.4 g%, packed cell volume- PCV-19.6 %, Platelet-0.39lakh/cumm, prothrombin time- PT test-17.28, international normalised ratio- INR-1.36). Four packed red blood cells (PRBCs), four fresh frozen plasmas (FFPs) and eight platelet concentrates were given over 24 h, along with frusemide 40 mg. He was ventilated.

Surgical decompression was done. Large flap was raised including the previous flap in centre. Fleshy pink growth in the plane of duraplasty was excised (Fig. 2a). The duraplasty was opened and excised. There was an organised hematoma adherent to brain. The hematoma was removed.

Thrombin-gelatin (FloSeal) was kept for hemostasis. The new duraplasty was done with Polypropylene patch (G-patch). The wound was closed in two layers. Scalp was lax after the procedure.

He had hypotension. Blood pressure (BP) was 90/60 mmHg. Noradrenaline infusion was started. Hemoglobin was 10 g%. PCV was 30.3 %. Platelet count was 1.1 lakh/cumm. PTINR was 1.39. Gave two FFPs. CT brain was repeated and showed decompressed status (Fig. 2b). Electrocardiogram (ECG) showed ST segment elevation in V4, V5, V6. Echocardiography was suggestive of myocardial infarction (MI). Ejection fraction (EF) was 40 % only. Troponin I (Trop I) was 2870 ng/ml. Started aspirin 75 mg, clopidogrel 75 mg, and enoxaparin 40mg bd. He worsened further hemodynamically on fifth day and dobutamine also was started. But he succumbed.

Histopathological result (HPR) came as metastasis from adenocarcinoma. Section showed dural tissue with an infiltrative neoplasm composed of cells arranged in nests and cribriform pattern (Fig. 2c). Cells were cuboidal with moderate cytoplasm, and pleomorphic vesicular nucleus with prominent nucleoli. Numerous mitosis were seen. Stroma showed lymphocytic infiltration. Immunohistochemistry (IHC) showed strong diffuse membrane positivity for Pan-Cytokeratin (Pan-CK) in tumour cells (Fig. 2d).

Discussion

Although there are many reports of metastases to dura mater, there is no report of metastases to duraplasty from other sites. There are reports of presentation with subdural hematoma. The mechanism of formation of subdural hematoma is the invasion of vessels by tumor cells. The probable mechanism of metastasis in the duraplasty also is the invasion of new vessels growing on the graft. The reason for the initial subdural hematoma of the patient is not clear, as it was before the diagnosis of esophageal carcinoma. The diagnosis of metastasis in duraplasty was made only because of high index of suspicion and performance of the CECT. A non-enhanced CT would have missed it.

The prognosis of a patient with extensive metastases is dismal. So, palliative and supportive care is needed. But surgical decompression can be attempted in life threatening conditions. Here, the patient had coagulopathy, anaemia, and thrombocytopenia. Surgical decompression was done after the correction of these. But he developed MI. There are reports of extended survival for 6 months to 1 year after surgery for dural metastases, after control of the primary malignancy. This report can be an alert for early detection and efficacious treatment of such cases. Further research is needed to know the mechanism of implantation of tumor cells in the graft.

Conclusion

This is the first report of a metastasis in duraplasty done for an earlier surgery, although there are lots of reports of metastases in dura mater. The patient had disseminated metastases from esophageal carcinoma. There was rapid progression into coma due to compression of the brain. Surgical decompression was done after the correction of anemia, coagulopathy and thrombocytopenia. But he succumbed due to MI. This case report alerts about the possibility of metastasis in duraplasty which may mimic a subdural hematoma.

CRedit authorship contribution statement

Ajaya Kumar Ayyappan Unnithan: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Anu Jose Markose:** Supervision, Methodology, Investigation, Formal analysis, Data curation.

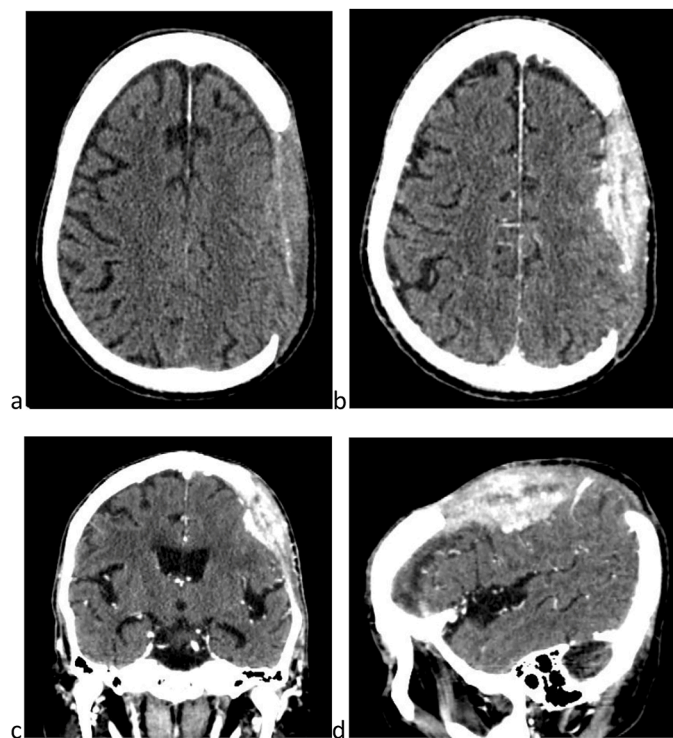


Fig. 1. a. CT scan showing an extraaxial swelling in the craniectomy site with compression of brain. b. Enhancement of tumor with contrast, suggestive of metastasis. c. Coronal view of the contrast enhancing growth. d. Sagittal view of the tumor.

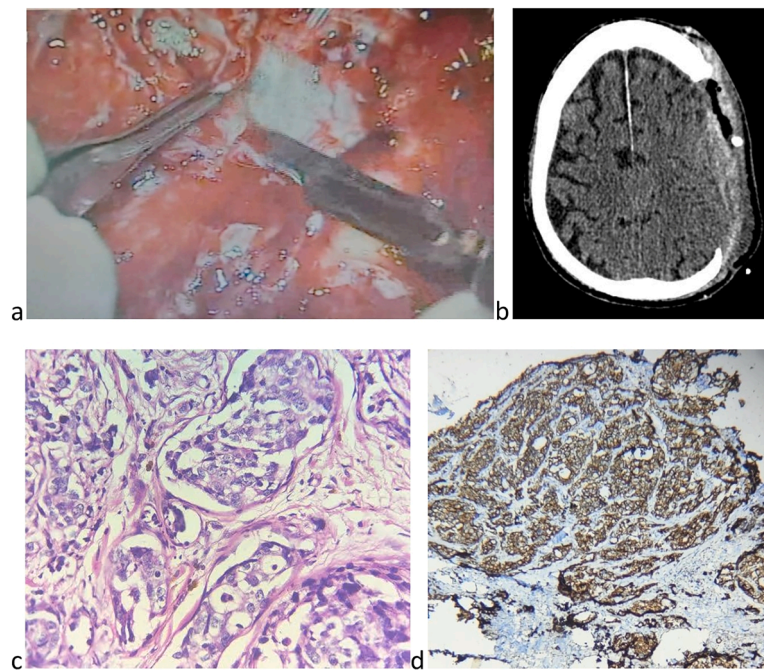


Fig. 2. a. Fleshy pink growth in the plane of duraplasty as seen peroperatively. b. Postoperative CT showing decompression. c. Histopathology showing an infiltrative neoplasm composed of cells arranged in nests and cribriform pattern. d. Immunohistochemistry showing diffuse membrane positivity for Pan-Cytokeratin.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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