



Leptomeningeal Carcinomatosis as Primary Presentation in Gastrointestinal (GI) Malignancies - Case Series and Review of Literature

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Abstract

Background: Leptomeningeal Carcinomatosis (LC) is a serious complication of cancer where the malignant cells spread to the meninges of Central Nervous System (CNS) involving the pia mater and arachnoid mater. LC heralds a poor prognosis with limited treatment options.

Case Presentation: We intend to report three cases of LC which is commonly the secondary event to Gastrointestinal (GI) malignancies and is very rare to be the presenting symptom. We present case series of three patients diagnosed with LC. Clinical examination of Case 1 revealed that he had left hemiparesis. Case 2 had neck rigidity with no other focal deficit. Upper GI endoscopy of Case 2 revealed ulcerated lesion in stomach and biopsy revealed infiltrating adenocarcinoma of stomach. Case 3 revealed cerebellar signs on clinical examination.

Conclusion: LC is a less common manifestation and is rare to be the presenting symptoms. Prudent and detailed evaluation is mandatory.

Keywords: Leptomeningeal carcinomatosis; Gastrointestinal malignancies; Magnetic resonance imaging; Histopathology; Clinical examination

Background

Leptomeningeal Carcinomatosis (LC), is defined as escalation of the disease to the meninges surrounding the central nervous system. It is an uncommon disease but usually a catastrophic issue observed in late stages of cancer. LC is detected in around 5% of patients suffering with recurrent cancer [1,2] however, lines of evidence suggest that it can be identified in around 20% of cases in autopsy cases [3]. LC is most frequently linked to skin cancer, breast cancer, lung cancer and gastrointestinal cancer. Furthermore, it is also detected in solid tumours including brain tumours and blood cancer [1,2,4]. The prognosis of LC is poor and it has been reported that patients who are not given treatment survive only for a month to a little more. Treatment increases the life span of these patients to approximately 2-3 months [5,6]. Several mechanisms have been proposed for LC development including (1) direct intrusion through surrounding structures; (2) spread of cancer most often through blood stream; and, lastly, (3) choroid plexus tissue producing Cerebrospinal Fluid (CSF) gets perforated which ultimately allows transport of solutes [7,8].

LC is commonly the secondary event to Gastrointestinal (GI) malignancies and is very rare to be the presenting symptom [9,10] The clinical presentation is usually subacute in nature, but acute presentation is rare and usually has a poor clinical outcome [11,12]. The most common manifestation of LC includes headache, nausea and vomiting, changes in gait, seizures, problems with vision, hearing loss, and others. Studies have shown that therapeutic interventions including radiation therapy, systemic therapy combined with intrathecal therapy, and chemotherapy may improve outcomes in patients diagnosed with LC.

Case Series

Case 1

A 77 years old male, a known case of hypertension presented with 7 days history of headache, fever, vomiting and altered sensorium and one day h/o seizure. He had no history of vomiting,

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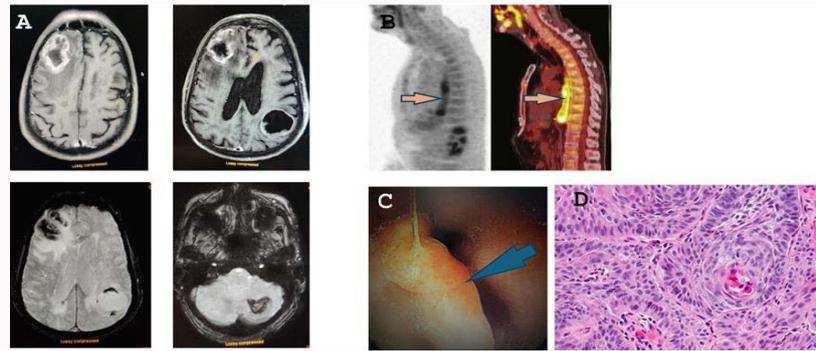


Figure 1: (A). MRI brain with contrast was done which showed multiple T1 hypo-intense and T2 heterogeneously hyperintense lesions with central necrotic areas in the right frontal, bilateral parietal, left posterior temporal and occipital lobes with extensive peri-lesional edema having intralesional hemorrhage showing thick irregular peripheral enhancement. MR spectroscopy revealed prominent choline peak in the peripheral solid portion and lipid peak in the central necrotic areas. (B). PET CT revealed necrotic circumferential wall thickening involving mid thoracic esophagus [orange arrows]. (C). GI consultation was taken, and upper GI endoscopy was performed which showed ulcerated lesion in esophagus, biopsy was taken [blue arrow]. (D). HP study showed poorly differentiated squamous cell carcinoma.

loose stools or fever. Patient had history of sudden onset of left sided weakness associated with involuntary movements. Patient attendant gave history of symptomatic improvement of left sided weakness. On clinical examination, patient was conscious but disoriented to time, place and person, obeying simple verbal commands. He had Left sided hemiparesis. His MRI brain with contrast showed multiple T1 hypo and T2 heterogeneously hyperintense lesions with central necrotic areas in the right frontal, bilateral parietal, left post temporal and occipital lobes with extensive perilesional oedema having intralesional haemorrhage showing thick irregular peripheral enhancement (Figure 1). His MRS showed prominent choline peak in the peripheral solid portion and lipid peak in the central necrotic areas. PET CT revealed necrotic circumferential wall thickening involving mid thoracic oesophagus. HP study showed poorly differentiated squamous cell carcinoma. Patient was advised to continue his previous medications as before and was treated symptomatically.

Case 2

A 65 years male, presented with episodic throbbing headache with sweating, vomiting each lasting for few minutes and multiple times in a day. On clinical examination he had neck rigidity with no other focal deficit. MRI Brain + C showed leptomeninges having diffuse increased T2 flair signal involving sulco-cisternal spaces (Figure 2). Cytology revealed presence of atypical cells having Signet Ring cell suggestive of metastatic poorly differentiated neoplasm. CECT chest and abdomen was reported to have eccentric wall thickening in mid body of stomach involving a segment of 3.6 cms with maximum wall thickness of 11 mm. CECT abdomen was S/o- diffuse circumferential thickening in distal antrum and pyloric region of stomach with mild luminal narrowing with serosa invasion and mild peri gastric fat stranding with lymphadenopathy. Upper GI Endoscopy (UGIE) revealed ulcerated lesion in stomach and biopsy revealed infiltrating adenocarcinoma of stomach.

Case 3

A 67 years male presented with multiple episodes of vomiting, head reeling and generalized weakness for 3 days. Clinical examination revealed cerebellar signs. MRI Brain+ C showed Lt cerebellar multiple ring and nodular enhancing lesions showing iso to hypo on T2 signal/ FLAIR and hypointense on T1W with disproportionate oedema along with patchy meninges and leptomeninges in both temporal lobe and adjacent to perisylvian cortex (Figure 3). UGIE presented

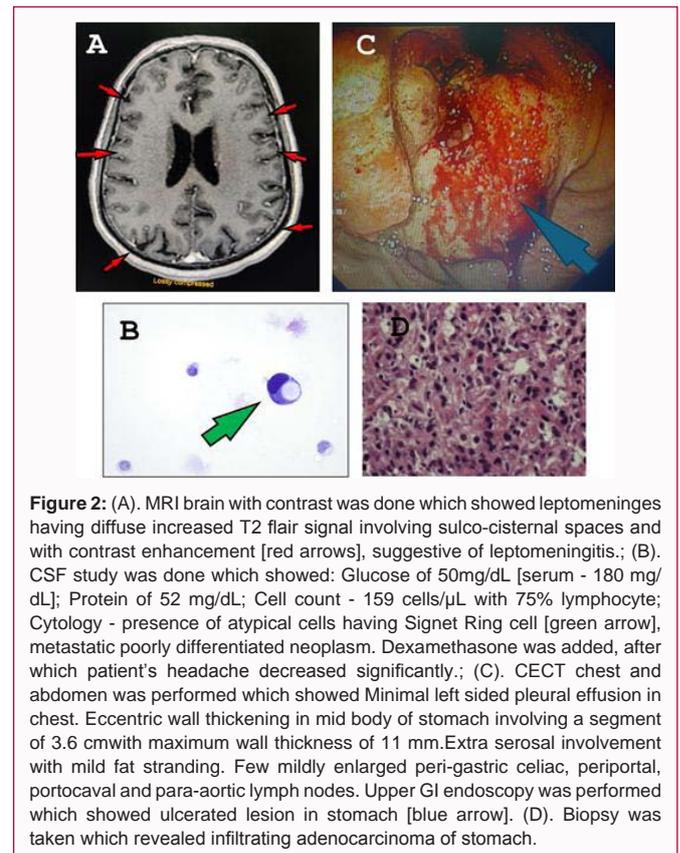


Figure 2: (A). MRI brain with contrast was done which showed leptomeninges having diffuse increased T2 flair signal involving sulco-cisternal spaces and with contrast enhancement [red arrows], suggestive of leptomeningitis.; (B). CSF study was done which showed: Glucose of 50mg/dL [serum - 180 mg/dL]; Protein of 52 mg/dL; Cell count - 159 cells/ μ L with 75% lymphocyte; Cytology - presence of atypical cells having Signet Ring cell [green arrow], metastatic poorly differentiated neoplasm. Dexamethasone was added, after which patient's headache decreased significantly.; (C). CECT chest and abdomen was performed which showed Minimal left sided pleural effusion in chest. Eccentric wall thickening in mid body of stomach involving a segment of 3.6 cm with maximum wall thickness of 11 mm. Extra serosal involvement with mild fat stranding. Few mildly enlarged peri-gastric celiac, periportal, portocaval and para-aortic lymph nodes. Upper GI endoscopy was performed which showed ulcerated lesion in stomach [blue arrow]. (D). Biopsy was taken which revealed infiltrating adenocarcinoma of stomach.

antral growth and histopathology study showed Adenocarcinoma of stomach.

Discussion

Leptomeningeal Carcinomatosis (LC) is the penetration of leptomeninges by the cancerous cells, which is a cataclysmic complication of solid tumours or blood cancers [13]. It is reportedly diagnosed clinically in 2% - 4% of all cancer patients. Gastric cancer usually presents with weight loss, persistent pain abdomen, nausea, dysphagia, melena. Gastric Cancer induced LC is less common 0.14% - 0.24% among all gastric cancers. There are very rare cases, LC presents as initial manifestation of asymptomatic gastric adenocarcinoma

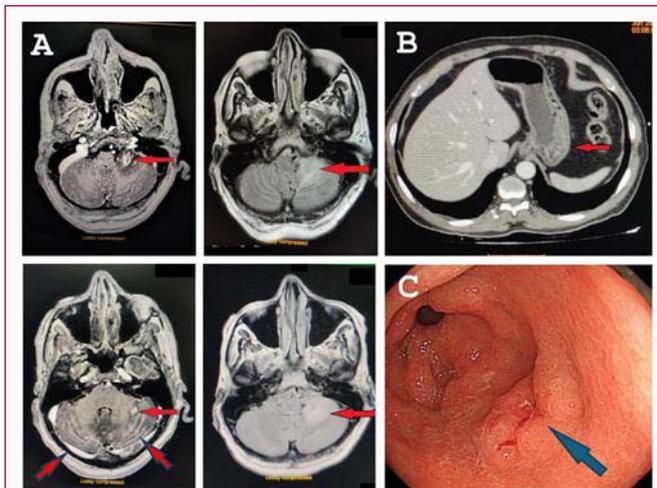


Figure 3: (A). MRI brain was done which revealed left cerebellar hemisphere multiple ring and nodular enhancing lesions (2 in number) showing iso to hypointense on T2 signal / FLAIR and hypointense signal on T1W of sizes ~ 9x9 mm and 10 x 9 mm with disproportionate edema around. The edema component showing mild diffusion restriction, T2/FLAIR signal. Cerebellar folia of both cerebellar hemisphere and basal cisterns increased T2/FLAIR signal with enhancement. Patchy meninges and leptomeninges in both temporal lobe and adjacent to perisylvian cortex similar increased T2/FLAIR signal and enhancement - likely Neoplastic etiology (? metastasis) with leptomeningeal /patchy meningeal spread. However rare possibility of chronic granulomatous lesion with meningitis cannot be ruled out. (B). CECT thorax and abdomen was done CT thorax did not reveal any abnormality. CECT abdomen was S/o- diffuse circumferential thickening in distal antrum and pyloric region of stomach with mild luminal narrowing s/o malignancy with serosal invasion and mild perigastric fat stranding with lymphadenopathy [red arrow]. (C). GI consultation was taken, and UGI endoscopy was performed which was S/O antral growth CA Stomach [blue arrow]. Biopsy was taken which was suggestive of adenocarcinoma of stomach.

where metastasis to peritoneum and liver generally occur first. The mortality rate of these patients is high and their prognosis is desolate. The incidence of LC is prevalent in all types of systemic cancers but mostly found associated with lung cancer [14].

Clinical manifestation of LC is multifaceted with multifocal neurologic deficits. This is due to the fact that malignant cells can easily penetrate the nerve roots of CNS, can directly intrude the brain or spinal cord, causing obstructive hydrocephalus. As a result, the patient shows symptoms of headache, nausea/vomiting, changes in gait, seizures, problems with vision, hearing loss, feebleness in the legs, backpain, and bowel/bladder dysfunction [15].

A thorough clinical history and examination is essential to discriminate a patient diagnosed with LC from other diagnoses that show similar manifestations. Understanding the clinical history of a patient allows for precise and timely treatment, and increasing the life span of the patient. Both MRI (gadolinium-enhanced) and CSF cytology are employed to confirm the diagnosis of LC. In LC patients which are suspected to have metastatic malignancies that can involve the complete CNS, an MRI of the brain with whole-spine imaging with T1- and T2-weighted sequences with contrast is recommended [15]. However, MRI itself is not sensitive enough to confirm the diagnosis of leptomeningeal disease. Reports indicate that MRI is only 65% to 75% sensitive in these patients [16]. Therefore, other cytological tests which involve the detection of cancerous cells in CSF, are conducted to accurately diagnose and confirm LC. However, the cytological test detects 50% false-negative results [17]. Hence, sampling of CSF in serial manner is conducted in order to improve sensitivity [18].

Since MRI and cytological CSF sampling when conducted alone yield inaccurate diagnosis of LC, it is advised to consider clinical examination along with MRI findings or serial CSF analysis together to get a clear picture of LC diagnosis [19].

Furthermore, the prognosis of LC is poor with a remaining life span of three to four months after detection [20]. Therapeutic interventions that include anticancer therapies and immunotherapies that penetrate the blood-brain barrier need to be personalized for each patient.

Conclusion

Leptomeningeal carcinomatosis is less common manifestation and is rare to be the presenting symptoms. Prudent and detailed evaluation is mandatory and those cases can be missed at places with limited facilities. High degree of suspicion can be rewarding.

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