

# AMYOTROPHIC LATERAL SCLEROSIS AS A PARANEOPLASTIC MANIFESTATION OF GASTRIC ADENOCARCINOMA – A RARE CASE REPORT

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## ABSTRACT

Motor neuron diseases (MND) have been reported as a rare paraneoplastic syndrome of a systemic neoplasm. Amyotrophic Lateral Sclerosis as a paraneoplastic manifestation of gastric carcinoma is even rarer. We present a patient with amyotrophic lateral sclerosis (ALS) in association with adenocarcinoma of stomach. A 48 year old man presented with a four months history of progressive dysphagia, spastic dysarthria and marked fasciculation in his atrophic tongue. Gag reflexes were diminished bilaterally. There was significant atrophy in thenar and hypothenar areas of both hands and dorsum of both feet. Electromyography result was compatible with diffuse motor neuropathy with active denervation. MRI brain showed classical findings of ALS. Upper GI endoscopic study showed ulcerated mucosa in body of stomach. Histological biopsy of stomach confirmed the presence of adenocarcinoma. The importance of considering a paraneoplastic syndrome in a patient with presentation of ALS is that it can lead to searching for underlying neoplasm before its apparent signs and symptoms develop and a scope to initiate treatment for primary carcinoma. Again treating the underlying neoplasm may halt or even resolve the neurologic signs and symptoms.

**Key Words:** Motor Neuron Diseases; Amyotrophic Lateral Sclerosis; Paraneoplastic Syndrome; Gastric Adenocarcinoma

## Introduction

Amyotrophic lateral sclerosis is a rapidly progressive, fatal neurodegenerative disorder for which there is no effective treatment. The diagnosis is based upon the clinical presentation and consistent electrodiagnostic studies. Typically, there is a combination of upper and lower motor neuron signs, clinically. Electrodiagnostic studies are indicative of motor neuron disease. MRI brain and MR spectroscopy help in clinching the diagnosis in appropriate clinical settings. Paraneoplastic etiology is a rare but established etiology of ALS. Here we report a case of ALS which originated as a paraneoplastic manifestation of gastric adenocarcinoma.

## Case Report

A 48 year old non-diabetic, non-hypertensive smoker male presented with a four months history of progressive dysphagia. Dysphagia was more towards liquids than solids. He also suffered from recurrent choking episodes during taking foods. He was also troubled by progressive difficulty in speech and hoarseness. His dysphagia and hoarseness had no diurnal variability or fatigability. No history of any diplopia or dribbling of saliva from angle of mouth. Also no history of any bladder-bowel or cortical or meningeal or sensory involvement was there. He gave history of recent onset anorexia and unintentional weight loss of 6 kg in last 3 months. On general examination, significant pallor was noted. No lymphadenopathy or

icterus was noted. On neurological examination, spastic dysarthria and marked fasciculation in his atrophic tongue were noted. Gag reflexes were diminished bilaterally. Other cranial nerves were intact. In muscle testing there was significant atrophy in thenar and hypothenar areas of both hands and dorsum of both feet without involvement of trunk or extraocular muscles. However no fasciculations were seen either proximally or distally. Muscle power testing showed power of distal muscles of both upper and lower limbs as 3/5 and proximally 5/5 according to Medical Research Council grading; Tendon reflexes were brisk bilaterally. No other abnormality in neurological examination was noted. Chest exam revealed ejection systolic murmur in pulmonary area. Other system examination revealed no abnormality. Laboratory evaluation showed that he had normocytic, normochromic anaemia. Urea, creatinine, sodium, potassium, calcium, phosphate were within normal limits. Creatine Phosphokinase was normal. Nerve conduction study showed diminished Compound Motor Action Potential (CMAP) with normal conduction velocity in both median, ulnar and peroneal nerves. Sensory Motor Action Potential, F-wave, H-reflex were normal. Electromyography showed fibrillation and positive sharp waves in all tested small muscles of hands and feet suggestive of MND. MRI brain showed hyperintensity in bilateral corticospinal tract in the region of internal capsule which was classical of ALS. CSF study was normal. Vit B12, HIV serology, thyroid hormone were also normal. MRI cervical spine ruled out any compressive myeloradiculopathy. Upper GI endoscopic

study showed ulcerated mucosa in body of stomach, Histological biopsy of stomach confirmed the presence of adenocarcinoma. In spite of intensive care, the patient continued to deteriorate and succumbed to massive aspiration and resulting severe pneumonia and sepsis.

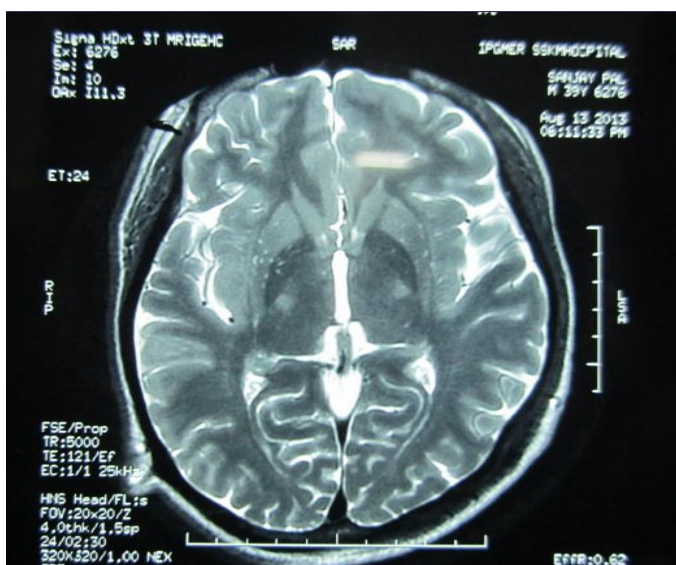


Figure-1: Hyperintensity in both internal capsule on axial T2 flair image



Figure-2: No evidence of cervical myeloradiculopathy in sagittal T1 MRI image

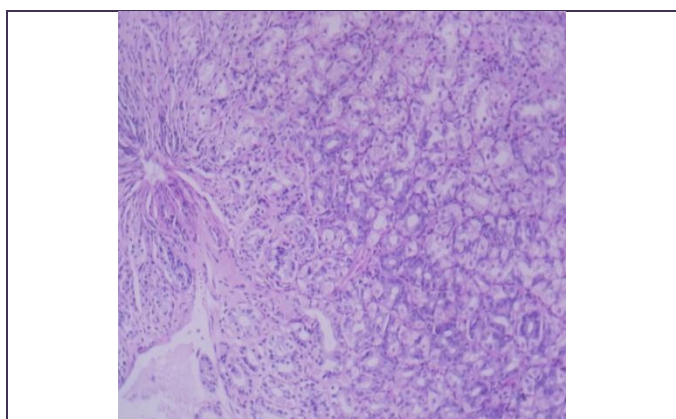


Figure-3: Gastric adenocarcinoma on high power photomicro-

## scopy of gastric biopsy specimen

## Discussion

Paraneoplastic syndromes refer to ability of some tumors to produce signs and symptoms at a site remote from their place of origin. Neurological paraneoplastic syndromes<sup>[1]</sup> first described by Oppenheim in 1888 mainly include limbic encephalitis, stiff person syndrome, peripheral neuropathy, cerebellar degeneration. Lung carcinoma specially the small cell subtype (SCLC) is mostly implicated. The mechanism of such neurological paraneoplastic syndromes is mainly immune mediated. ALS is a rare paraneoplastic manifestation. The mechanism of anterior horn cell damage in ALS is most probably a result of immune cell attack.<sup>[2]</sup> Although SCLC is most common primary carcinoma in ALS, breast carcinoma, lymphoma<sup>[3]</sup>, myeloma, renal cell carcinoma<sup>[4]</sup> also are known for such presentation. Data on ALS arising as a paraneoplastic manifestation from gastric carcinoma is scarce. MND has been reported with esophageal carcinoma.<sup>[5]</sup> Mehrpour et al reported a case of ALS with gastric neuroendocrine tumor.<sup>[6]</sup>

## Conclusion

The purpose of this case report is to stress upon the fact that in an appropriate clinical setting, a paraneoplastic etiology of ALS should be kept in mind.

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