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CEREBELLAR MANIFESTATION SECONDARY TO RECTAL NEOPLASIA

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ABSTRACT
The condition known as paraneoplastic cerebellar degeneration (PCD) is among the most disabling examples of a cancer's remote effects. Serology for paraneoplastic antibodies, clinical suspicion, and a targeted search for malignancy all play a role in the diagnosis. Tests for neuronal autoantibodies aid in diagnosis and point the way for the search for malignancy. Unfortunately, even with an early diagnosis, existing therapeutic approaches only have little success. Here we present the case of a young male who developed cerebellar syndrome secondary to rectal neoplasia.

KEY WORDS: Cerebellar syndrome, Rectal mass, Colorectal carcinoma, Paraneoplastic

INTRODUCTION
Paraneoplastic syndromes are a group of rare disorders that have a variety of neurological manifestations secondary to underlying malignancy. Main pathophysiology is an autoimmune response to a tumor when they express neuronal proteins or by immunological disturbance by tumor rather than a metastatic complication.[1] It is commonly an initial manifestation of an occult malignancy and affects 1-3% of all cancer patients. Any malignancy can cause a paraneoplastic syndrome and can affect any part of nervous system.[2]

Paraneoplastic cerebellar degeneration occurs predominantly in patients with neoplasia of ovary, uterus, breast, lungs or Hodgkin's lymphoma.[3,4] It is mediated by antibodies formed against tumor antigens and these proteins resembles with those found within cerebellum resulting in cross reaction causing cerebellar injury and dysfunction.[5] Mostly specific antineuronal antibodies detection precedes diagnosis of malignancy. In approximately 40% of patients, no antibodies are identified and it does not exclude the likelihood of malignancy.[6]

Colorectal malignancy rarely presents with neurological paraneoplastic disorder; few cases have manifestation of vasculitis and sensory neuropathy. Here we report a case of seronegative paraneoplastic cerebellar degeneration associated with rectal adenocarcinoma.

CASE PRESENTATION
A 40-year-old male, having no previous co-morbidities, plumber by occupation, presented with history of diarrhea and vomiting two months back. He was initially treated at local setup but had episodes of loss of consciousness that were not associated with convulsions or sphincteric incontinence. After that he developed speech disturbance along with head tremors, and difficulty in looking upward and walking for 20 days, leading to bed-bound state. He also gave history of fresh rectal bleeding.

At the time of presentation in ER, he was hemodynamically stable with no signs of respiratory distress. Glasgow coma scale (GCS) 15/15, Pupils were bilateral equal and reactive, upward gaze palsy, head titubation, dysarthric speech, power 4/5 in upper extremities, +4/5 in lower extremities. Plantars were bilateral downgoing. Cerebellar signs were positive i.e. nystagmus, past pointing and dysdiadochokinesia; severe limb and gait ataxia were noted. Sensations and sphincters were intact. Modified Rankin scale score was 5 at time of admission.

On investigation, blood complete picture showed total leucocyte count of 7480/mm^3, Hemoglobin of 13.5g/dl, platelet count of 245000/mm^3. Metabolic and coagulation profile were normal, hepatitis B &C screening was negative. Plain CT scan brain was unremarkable. Cerebrospinal fluid routine examination showed WBC count of 5 cells/mm3 with 90% lymphocyte predominance and CSF glucose of 83mg/dl and protein of 59mg/dl. MRI brain with contrast & MRV was unremarkable. Antinuclear antibody and Serum autoimmune encephalitis panel were negative (Table 1).

Table 1: Paraneoplastic antibodies panel of the patient

Name of antibody	Results
Anti Yo	Not Detected
Anti Hu	Not Detected
Anti Ri	Not Detected
Anti Amphiphysin	Not Detected
Anti-ANNA	Not Detected
Anti Ma2	Not Detected
Anti-Recoverin	Not Detected
Anti CRMP 5	Not Detected

Erythrocyte sedimentation rate was 2 mm/hr. IgG anti gangliosides antibodies and celiac disease screening were negative. Thyroid profile and serum vitamin B12 levels were normal. Stool routine examination was normal. HIV screening was negative. Nerve conduction studies / Electromyography were unremarkable. Ultrasonography abdomen and pelvis showed altered hepatic echotexture with hyperechoic area in fundal region of gall bladder, likely polyp (incidental finding). Computed tomography scan Abdomen and Pelvis with contrast showed circumferential thickening of rectum with pararectal stranding not involving mesorectal fossa extending from S3 vertebral body till coccyx, findings suggestive of Rectal carcinoma.

Initially treatment started on the lines of CNS Infection with broad spectrum antibiotics in meningitis dose along with DVT prophylaxis. No improvement was observed. Then pulse therapy (1g intravenous methylprednisolone) for 5 days was given along with antibiotics. Still no improvement in symptoms was observed, Plasma Exchange five sessions were then done along with vigilant monitoring for autonomic dysfunction. Slight improvement in symptoms of ataxia and head tremors were observed. Patient was discharged with Modified Rankin scale of 4 and referred to General surgery department for urgent further evaluation and management of rectal carcinoma.

DISCUSSION

In a 10-year Mayo Clinic retrospective study, out of 115,081 cancer patients, only 58 patients suffering from paraneoplastic neurological syndrome, of which only five patients had colon carcinoma.[7] Peripheral neuropathy, cerebellar syndromes, limbic encephalitis and p-ANCA-positive vasculitis, and myoclonus were reported as paraneoplastic manifestations.

For our patient, we were not able to identify an associated autoantibody. Guidelines for paraneoplastic neurological syndrome diagnosis states that in the setting of a classical syndrome, the presence of onco-neuronal antibodies is not required.[8] No intrathecal chemotherapy and surgery made our patient symptom free suggested that his clinical neurological phenomenon was indeed paraneoplastic in nature.

All the authors have approved the final version of the article, and agree to be accountable for all aspects of the work.

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Author's contribution:
Soban Khan; concept, case management, data collection, data analysis, manuscript writing
Zaid Waqar; case management, data collection, data analysis, manuscript writing
Maryam Khalil; case management, data collection, data analysis, manuscript writing
Bushra Khalid; case management, data analysis, manuscript revision
Sajid Ali; case management, data analysis, manuscript revision
Ijaz Ali; case management, data analysis, manuscript revision
Muhammad Tariq; case management, data analysis, manuscript revision
Muhammad Adil; case management, data analysis, manuscript revision
All the authors have approved the final version of the article, and agree to be accountable for all aspects of the work.