Case Series

Covid in Disguise – A Series of Neurological Presentations

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We present three patients of COVID19 who presented to the emergency with neurological derangements. On admission fever, cough, sorethroat or contact history were notably absent in the first two that led to initial confusion about the diagnosis. We hereby stress on keeping SARSCoV2 infection in the differential diagnosis if patients present during this pandemic with neurological symptoms.

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Key words **: COVID19, Neurological Derangements, Differential Diagnosis.**

The ongoing COVID19 pandemic has overwhelmed the medical fraternity with its expanse and virulence. The novel coronavirus is classically said to present as a febrile illness involving the respiratory tract predominantly. However,neurological presentations with or without typical features are being encountered especially in the elderly[1].

Case 1 :

A 72 year old hypertensive, nondiabetic male presented with the history of headache and insidious onset of drowsiness for the last two days. There was no history of seizures vomiting, limb weakness, head trauma, addictions or known liver, kidney or pulmonary disease. On admission, his relatives denied any history of fever, cough, sorethroat or respiratory distress.

On examination at admission —

The patient had a GCS of E3M3 V2. There was mild anaemia, no jaundice, edema cyanosis clubbing lymphadenopathy. Pulse was 100/min, BP 150/90mm Hg, respiratory rate 20/min, oxygen saturation 96% in room air. There was neck rigidity along with a positive Kernig's sign. No cranial nerve palsies, tone and reflexes were normal. Power and sensation could not be tested. Other systemic examinations were non-contributory.

Preliminary investigations reveal Hb% 11gm/dl, TC 12000/cu mm DC N 92% L6 %E 1%M1%, ESR 70mm/hr, platelet- 70,000/ cu mm, Na 123meq/litre, K 4.5 meq/liter,urea30 mg/dl creatinine 0.9mg/dl, LFT- bilirubin -1mg/dl, ALT64meq/l, AST50meq/l . ALP, Albumin, Globulin were normal. CTScan Brain came out to be normal. CSF was sent for evaluation.

After six hours —

The patient had deteriorated. GCS E1M1V1, pulse rate 130/ min, BP 90/70, respiratory rate 34/min and , saturation 68% on oxygen. He was febrile, breathless and unconscious.Chest examination revealed bilateral scattered crepitations. Other findings were similar as before.

ABG revealed a Type 1 respiratory failure. Chest x ray was done , showing bilateral interstitial infiltrates. CT Chest bilateral ground glass appearance. The CSF demonstrated a cell count of 8 (all lymphocytes) and protein 84mg/dl, other parameters being normal. Prothrombin time, D Dimer and RT PCR for SARSCoV2 were sent. All of the reports were abnormal i.e the patient was diagnosed to be suffering from COVID19 with a presentation resembling acute encephalitic syndrome (AES) in the absence of preceding history of fever, cough or dyspnoea.

Editor's Comment :

- **COVID 19 patients with severe disease may manifest neurological features like stroke.**
- **During the pamdemic of COVID-19, patients presenting with neurologic manifestations, should prompt clinicians to consider SARS-CoV-2 infection as a differential diagnosis.**

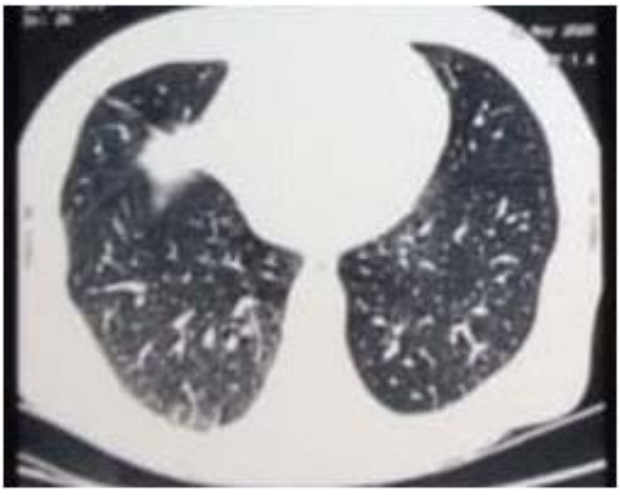

Fig 1 — CT Scan Thorax shows bilateral rounded opacities

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Case 2 :

A 66year old lady presented to the emergency with sudden onset weakness of right half of the body and slurring of speech since morning . There was no history of hypertension, diabetes, ischaemic heart disease or dyslipidaemia. In view of ongoing pandemic h/o fever, cough, SOB was taken. No such significant history or h/o Travel or contact. On examination the patient was conscious, with GCS of E4M5V3. Blood Pressure – 160/90 mm Hg , P/R – 110/min , R/R – 32/min. There was evidence of UMN type of facial palsy of the right side.The power on right upper and lower limbs was 4/5 and 3/5 respectively. Tone and jerks were normal on both sides, sensation could not be tested. Plantar was extensor in right side.

Patient was sent for CT Scan Brain. CT brain revealed Left MCA territory infraction. Before admission Resident medical officer checked saturation by Pulse oximeter as per Hospital Protocol. Saturation was 76% at room air but no dyspnoea. Immediately X-ray Chest PA view was done. X-ray Chest showed – bilateral infiltrates. She was admitted, but in same night she had developed shortness of breath. On further enquiry it was evident that she had been suffering from malaise, anorexia, and bodyache for a last few days but no fever. Investigation revealed : Hb% - 11.4 gm%. TLC-5600/dl , ABG – PO2- 66mm Hg , PCO2- 34mm Hg, ECG – normal. Random Blood sugar 112mg/dl . CRP, D-dimer was high . RT-PCR for SARSCoV2 was positive.

Case 3 :

A male patient of 65 years came from a red zone with a history offever for 5 days,cough for 4 days and shortness of breath for 2 days . He had no h/o hypertension, diabetes mellitus, dyslipidaemia, travel or contact . On examination the following findings were noted: pulse rate – 120/min, BP – 140/92 mmHg, respiratory rate– 24/min, oxygen saturation – 86% at room air. Examination of chest revealed few crepitation in both lung bases. Patient was admitted . On investigation it was found that Hb- 12.4bg/dl, TLC was 4500/dl . ABG showed PO2 – 60mmHg, PCo2 – 32mm Hg . Renal function was normal. X-ray Chest showed B/L infiltrates . HRCT thorax showed ground glass opacities. Patient tested for COVID-19 and was positive. Patient was put on treatment as per protocol.

But on 4th day of admission patient became drowsy E3M3V2and the physician noticed decreased movement of left side of body. There was decreased tone in left upper and lower limb. Power 1/5 in all limbs. Plantar was extensor in left side. CT scan brain advised and revealed large infract in right MCA territory.

Discussion

The typical clinical features of COVID 19 are fever cough dyspnoea or diarrhoea. However it is being increasingly recognized that patients may present with atypical variants involving other organ systems. About four fifths of the patients infected by the SARSCoV2 are said to be asymptomatic[2].

The elderly are more prone to develop atypical clinical features .These include dizziness, lethargy, delirium, syncope and falls, nausea vomiting, abdominal pain,hemoptysis, hypotension, conjunctivitis, loss of sense of smell or taste[3-6].

Neurological presentations of COVID 19 may include acute cerebrovascular disease,necrotising hemorrhagic encephalopathy and muscle injuries. It has been documented that these are seen more in elderly and patients of severe disease[1,7].

The underlying pathogenesis put forward is that ACE2 was identified as the functional receptor for SARS-CoV-2, which is present in multiple human organs, including nervous system and skeletal muscles. The expression and distribution of ACE2 may indicate that the SARS-CoV-2 may cause some neurologic manifestations through direct or indirect mechanisms[8].

SARS-CoV-2infection is said to produce a prothrombotic state causing venous and arterial thromboembolism and elevated D-dimer levels.Severe COVID-19 leads to abundance of proinflammatory cytokines which induce endothelial and mononuclear cell activation with expression of tissue factor leading to coagulation activation and thrombin generation. Circulation of free thrombin, uncontrolled by natural anticoagulants, can activate platelets and lead to thrombosis.Although ischaemic stroke has been recognised as a complication of COVID-19 (usually with severe disease)[1], the mechanisms are not yet understood. All patients had large-vessel occlusion; in one ischaemic stroke occurred 4 days after Covid-19 symptom onset, and in the other, during the presymptomatic phase, suggesting that COVID-19 associated ischaemic stroke can occur both early and later in the course of the disease[9].

It is also recently being reported that multiple vascular territories may simultaneously get involved and young individuals (less than 40 years) are also presenting with cerebral strokes.

It has been suggested that COVID-19 might stimulate the production of antiphospholipid antibodies (aPL) as a mechanism of ischaemic stroke, although post-infection aPL are usually transient and not associated with thrombosis[10].

In a Chinese study with 214 patients it was found that seventy-eight patients (36.4%) had nervous system manifestations: CNS [24.8%], PNS [8.9%], and skeletal muscle injury [10.7%]. In patients with CNS manifestations, the most common symptoms were dizziness [16.8%] and headache [13.1%]. In patients with PNS symptoms, the most common reported symptoms were taste impairment [5.6%] and smell impairment [5.1%][1]. It was also found that patients with nervous system involvement had severe disease.

Patients with severe infection had multiple organ involvement, such as serious liver (increased lactate dehydrogenase, alanine

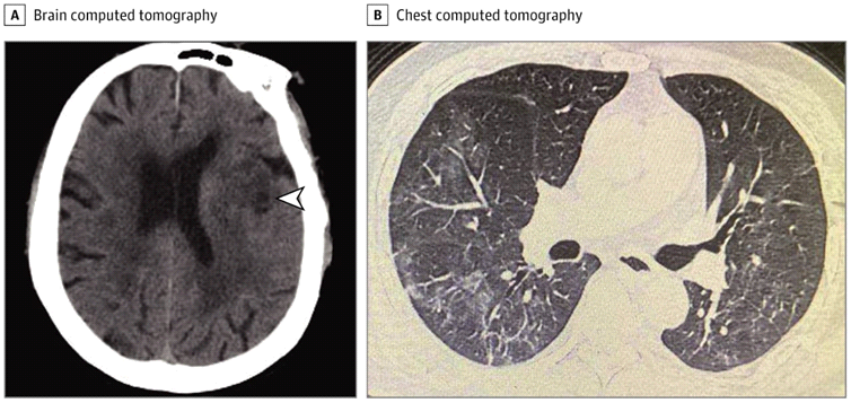

Fig 2 — (A) CT Scan brain shows left MCA territory infarct in case 2, (B) CT S Thorax shows bilateral ground glass opacities in case 3

aminotransferase, and aspartate aminotransferase levels), kidney (increased blood urea nitrogen and creatinine levels), and skeletal muscle damage (increased creatinine kinase levels).

It is documented that patients with severe infection are significantly olderor have comorbidities, especially hypertension and have fewer typical symptoms of COVID-19 such as fever and dry cough .Some patients with fever initially negative for SARSCoV2,several days later,may develop typical COVID-19 symptoms such as cough, throat pain, lower lymphocyte count, and ground-glass opacity appearance on lung CT and have positive test result[1,11].

Autopsy of patients with COVID-19 havedemonstrated that the brain tissue to be hyperemic and edematous and some neurons degenerated[12]. Neurologic injury has been also found in infection of other CoVs such as in SARS-CoV and MERS-CoV. SARS-CoV nucleic acid was detected in the cerebrospinal fluid of those patients and also in their brain tissue on autopsy[13,14].

These cases presented as AES and CVA respectively. All the patients were elderly, and developed severe disease. The point to be highlightedhere is that during the epidemic period of COVID-19, if patients present with neurologic manifestations, clinicians should consider SARS-CoV-2 infection as a differential diagnosis. Early diagnosis will entail rapid isolation, break of the transmission chain and better management .

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