Pseudo Bulbar Palsy a Rare NEURO-COVID Presentation
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ABSTRACT
Background: The Global pandemic of COVID-19 with more than half million people passed away from their families tragically, wide variety of signs and symptoms resulted from this Novel SARS Cov2 virus. Although the respiratory and Gastro-intestinal symptoms are dominant, however, neurological symptoms whether specific or nonspecific have been reported. Till now the extents of knowledge regarding the neurological sequel of COVID-19 infection are finite. Pseudo Bulbar Palsy is a neurological disease characterised by dysarthria, dysphagia, facial and tongue weakness in addition to emotional liability resulted from an upper motor neuron lesion in the corticobulbar tracts. Peripheral neuropathy, Demyelisation and vascularise are underlying aetologies. This will result in deglutition, and speech disturbance.

Case presentation: 60 years old male from Najaf, went to the emergency department with three days history of drowsiness and anorexia after COVID-19 related fever and myalgia. He had significant risk factors. He had 2 weeks fever following a contact with COVID-19 patient. Examination revealed drowsiness, dehydration, the patient was not aware for time and place, eye reflexes was symmetrical. The face was normal with exaggerated jaw jerk CT of the chest showed evidence of COVID-19 infection confirmed by positive PCR. Brain CT showed only brain atrophy without signs of localised ischemia. Further blood investigations confirm activity of infection. During this the patient developed tonic clonic convulsion required anticonvulsant therapy; he remained semiconscious, with difficulty to swallow and dysarthria, Irritability, drowsiness and disorientation. He received anticoagulant, Steroids, mannitol. The condition improved and the patient discharged in better condition with two weeks anticoagulation.

Discussion: this is a story for a rare complication belong to COVID-19 infection, treated as a stroke, as mention in literatures, many recorded cases of Neurocovid that required treatment accordingly.

Conclusion: neurological symptoms of COVID-19are extreme and might be nonspecific but some time life threatening, these might overlap severe infection. The improvement in the neurological sequel overlap the COVID-19 improvement both clinical aspect and investigation.

Keywords: pseudo bulbar palsy, COVID-19, neurological complication

INTRODUCTION
The global world still dealing with the new SARS Cov2 virus infection, and the resultant COVID-19 outcomes, even over half a million deaths have been reported [1] minors and the sexes are not immune to infection [2]. Even countries with high levels of medical care, declare the fact, they cannot control the deadly epidemic [3]; Iraq is facing cases that contrast with a sharp increase in new cases and a huge increase in the number of deaths; this is due to the deterioration of the health system and lack of public awareness [4]. The typical COVID-19 related syndrome mainly respiratory in form of pneumonia with variable degrees of lung injury, that sometimes end with respiratory failure and death in high risk patients like those older than 65 years (approximately 50%) [5], these respiratory symptoms become obvious in most patients within 2 to 3 weeks during the illness. Although, COVID-19 has been studied extensively by researchers, there have been limited reports of related neurologic complications [6]. The pathophysiology of the neurological manifestations related to COVID-19 mainly consisting of either an axonal peripheral neuropathy or a myopathy and vacuities. Pathological reports subsequently showed that patients with SARS had widespread vacuities seen in many organs, suggesting that the clinical neuromuscular features might be more than just nonspecific complications of severe illness. A pathological post-mortem specimen discovered a direct viral infiltration of the brainstem in a very limited number of sample, this raises the possibility that some of the crucial pathophysiology related. Community studies reported 36.4% of COVID-19 cases had some nervous system–related clinical finding1. The onset of the neurological insults varies in different patients. Some researches report cases of olfactory neuropathy with onset after 3 weeks from illness [7]. These neurologic manifestations ranged from fairly specific symptoms (e.g., loss of sense of smell or taste, myopathy, stroke and total paralysis) [8]. To more nonspecific symptoms (e.g., headache, depressed level of consciousness, dizziness, or seizure). Cerebrovascular accident are recorded more in hypertensive and diabetic patients. High body mass index is also regarded a risk factor [9]. Neurologic symptoms were more common in COVID-19 patients with more severe disease (30.2%in non-severe patients and 45.5% in severe patients), 20% of patients needs ICU admission had neurological manifestation, and they are liable for higher mortality [10]. Pseudo bulbar palsy is a clinical constellation characterized by dysarthria, dysphagia, facial and tongue weakness, emotional liability had been recorded (especially in brain tumours of the posterior fossa), particularly when Pons and midbrain are involved [11]. Also a bilateral cortical dysfunction might give clinical features of Pseudobulbar palsy due to an upper motor lesion caused by bilateral disturbance of the corticobulbar tracts figure (1). These tracts extraspura nuclear control over brainstem motor nuclei and are involved in the muscular movement of the head and neck [12]. These nuclei control mastication, deglutition, and speech [13]. Pseudo bulbar palsy prevalence increases, particularly after 50 years of age. Incidence is higher in males compared to females among all age groups [14].
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Figure 1: Diagram (A), (B)

Figure 1: Diagram (A) of the tracts in the internal capsule, motor tract (red), the sensory tract (blue), the optic radiation (occipito-thalamic) is shown in violet, Geniculate portion of motor tract, thalamo frontal tract. Diagram (B) Pathways from the Brain to the Spinal Cord, The motor tract, Anterior nerve roots, Anterior and Lateral cerebrospinal Fasciculus, Decussating of pyramids, geniculate fibbers, internal capsule, and Motor area of cortex. The diagnosis of PBP requires a multidisciplinary team including psychiatrist, internist in addition to the neurologist. Investigation for confirmation and discover the underlying pathology including complete blood count, metabolic profile and serology. In addition to more sophisticated tools like Electro-encephalogram, CSF analysis, imaging studies like Brain computed tomography (CT) and MRI, Diffusion-weighted MRI/T2-weighted RI, Motor-and sensory-evoked potential tests [15]. No specific treatment applied for PBP, but sometimes antidepressant and psychotherapy for the emotional liability in addition for the treatment of underlying illness. Rehabilitation is important line in the treatment [16].

CASE PRESENTATION

R A was 60 years old male went to the emergency department for three days history of drowsiness associated with anorexia and feeding reluctance he had no previous history of diabetes, hypertension, smoking or previous neurological disease. He had 2 weeks fever following a contact with COVID-19 patient in one family member when he advised for self-isolation before he deteriorated. On
examination the patient was drowsy, evidence of dehydration and poor feeding. Vital signs were stable including temperature, blood pressure and pulse rate. His investigation revealed little high blood sugar, normal renal, liver functions and normal electrolytes. Upon neurological examination the patient was awake but not aware for time and place, eye reflexes was normal and symmetrical although gaze was difficult to assess. The face was normal without mouth deviation or blinking abnormality apart from exaggerated jaw jerk. Other facial and buccal sensory function was intact. Chest showed bilateral peripheral ground glass opacities and areas of atelectasis suggestive for COVID-19 infection. Brain CT showed only brain atrophy without signs of localised ischemia, figure (1). PCR comes positive for SARS-2 infection. Shortly the patient developed tonic-clonic convulsion required treatment with Epanuthin infusion, although fit was terminated, however he remained semiconscious. He had also difficulty to swallow and dysarthria. Irritability, drowsiness and disorientation also not disappeared, his ECG was normal only sinus tachycardia, figure (2).

Blood oxygen dropped to 88% and required support. Blood investigation, showed the classical blood film findings in COVID-19 patients, table (1) with high readings of D-Dimer and Serum Ferritin that seen in active infection.

Table 1: first day blood investigations, showed lymphopenia, thrombocytopenia and high ESR with raised both D-Dimer and serum ferritin which is classical in COVID-19 infection (abnormal readings highlighted red)

<table>
<thead>
<tr>
<th>Blood test</th>
<th>Result</th>
<th>Normal value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin (g/dl)</td>
<td>14.3</td>
<td>11.5-17.6</td>
</tr>
<tr>
<td>WBC count (10e3/uL)</td>
<td>5.34</td>
<td>4.00-11.00</td>
</tr>
<tr>
<td>lymphocyte count (10e3/uL)</td>
<td>0.63</td>
<td>1.5-3.5</td>
</tr>
<tr>
<td>Lymphocyte ( % )</td>
<td>11.90</td>
<td>20-45</td>
</tr>
<tr>
<td>Platelet count (10e3/uL)</td>
<td>86.9</td>
<td>150-400</td>
</tr>
<tr>
<td>ESR (mm/h)</td>
<td>85</td>
<td>NV&lt;20</td>
</tr>
<tr>
<td>D-Dimer (ng/ml)</td>
<td>669</td>
<td>30-350</td>
</tr>
<tr>
<td>Serum ferritin (ng/ml)</td>
<td>611</td>
<td>30-350</td>
</tr>
</tbody>
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The estimated Glasgow Coma scale was around 9-10, figure (3).

Enoxaparin 4000IU BID was given, in addition to Dexamethasone 8mg and antibiotics. Mannitol infusion was advised by a committee member although no evidence of papilledema. During the following days body temperature, figure (4) and oxygen level figure (5) fluctuated but towards improvement, even consciousness also improved with mild residual disorientation and swallowing difficulty.
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The patient condition continued in improvement both clinically in form of consciousness, orientation, swallowing, also his GCS improved with time figure (3). Improvement also seen in the laboratory result as the D-Dimer and ESR started to decline figures (6) and (7) respectively. The lymphocytes total count and differential percentage, that carries an important prognostic value also improved during hospitalization figures (8) and (9), respectively.

**Figure 3:** Glasgow Coma Scale (GCS) estimation of consciousness degree during days of hospitalization

**Figure 4:** Body temperature Progress during hospital days

**Figure 5:** pulse oxygen readings progress during hospital days
At the seventh day of admission, MRI was performed but it added nothing, only the same finding of brain atrophy, without any evidence of localized abnormality. The patient discharged in good condition.

**DISCUSSION**

The offered case is a rare presentation of COVID-19 patient, although brain CT scan and MRI didn’t show exact pathological site, however the constellation of signs and symptoms of Pseudo bulbar palsy, points towards focal and or diffuse neurological complaint overlapping his infection. Also his successful treatment of stroke, including antplatelet and other blood thinners like LMWH, warfare in addition to steroids and Manito reflected his improvement of his neurological complaint. As described in the literatures, the range of neurological symptoms in COVID-19 cases is extreme, extending from simple, non-specific headache with myalgia to more specific and life threatening illness like encephalopathy or stroke. Although, neurological sequel in COVID-19 usually comes late in the chronologic time, however, it overlaps a severe, persistent and active infection proved by investigation as seen in the case study above. Improvements after therapy in the COVID-19 related symptoms goes in parallel with improvement in neurological symptoms, this is—also—have been seen in the scenario above. So differential diagnosis range from focal to diffuse brain insult such as stroke, vacuities and hypoxic ischemic encephalopathy.

**CONCLUSION**

1. The spectrum of neurological symptoms in COVID-19 cases are extreme, extending from simple and non-specific headache with myalgia to more specific and life threatening illness like encephalopathy or stroke.
2. Although, neurological sequel in COVID-19 usually comes late in the chronologic time, however, it overlaps a severe, persistent and active infection.
4. Vasculitis might cause stroke in COVID-19 patients, this explained speed of recovery in neuron stunning like phenomena.
5. Presence of severe dysphagia and the resultant aspiration pneumonia might worsen the respiratory symptoms of COVID-19.

RECOMMENDATION
1. The presence of focal neuropathy must be taken into account in any of the COVID-19 patients with disturbance of consciousness.
2. Anticoagulant is advised for pseudo bulbar palsy patient in post discharge for period of time such as two week as in this patient. Taking in consideration low platelets count and impaired liver function that might negatively affect critically ill patient, in which bleeding will catastrophic.

REFERENCE