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Case Report

Meningoencephalitis and Spontaneous Acute Subdural Haematoma Complicating COVID-19: Report of an Unusual Case and Review of Literature

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Abstract

Neurological complications of COVID-19 are not uncommon and can sometimes be serious and even fatal. We report a case of COVID -19 complicated by meningoencephalitis and spontaneous subdural hematoma requiring emergency craniotomy. Patient had a stormy course in intensive care and eventually died. As far as the authors are aware only handful of COVID-19 patients complicated with subdural hematoma have been previously reported in the literature.

Keywords: COVID-19; Pneumonitis, Intensive Care; Subdural hematoma; Meningoencephalitis; Craniotomy

Introduction

The novel coronavirus SARS CoV2 was first identified in Wuhan, China in December 2019. Since then COVID-19 has become a global pandemic causing considerable morbidity and mortality especially in elderly and persons with underlying health problems such as diabetes. The spectrum of the clinical picture includes asymptomatic infection, mild respiratory symptoms, severe pneumonia, multisystem involvement and coagulopathy leading to intensive care admission. Reports of serious cerebrovascular and neurological complications associated with COVID-19 are still emerging.

Case Presentation

A 66-year-old male with well controlled type 2 Diabetes, presented to primary care with fever, sore throat and cough. He had no exposure to COVID-19. His other co-morbidities included hypertension, dyslipidemia, and palpitations for which he was on Metoprolol. Clinically he was febrile but hemodynamically stable. SARS COV2 RT-PCR test was reported positive the following day. By then he developed dyspnea and was referred to secondary care emergency services where he was found to be febrile, tachypneic and desaturating on air. Chest x-ray showed bilateral diffuse patchy opacities suggestive of COVID Pneumonitis. The following day he deteriorated further and was shifted to intensive care.

Timeline of Significant Events in Intensive Care Unit (ICU)

Day 1: Admission to ICU, respiratory support with Non-Invasive Ventilation (NIV). Chest x-ray worsened and commenced on Remdesivir, cefuroxime and dexamethasone.

Day 4: Intubated due to restlessness and tachypnoea. ECG showed ST elevation, Troponin rose to 79 from 10. Bedside echo showed no wall motion abnormality, good LV contractility and no pericardial effusion. Cardiologist opined this to be likely myopericarditis.

Day 5: Developed SVT which settled with carotid massage.

Day 10: AF with rapid ventricular response and hemodynamic instability. Reverted to sinus rhythm after three DC shocks and Amiodarone.

Day 11: Non enhanced CT scan brain done for suspected encephalitis, reported normal. MRI brain indicates COVID-19 encephalopathy (Figure 1).

Day 12: Hypokalemia induced VT. Treated with DC shock and started on Amiodarone and Colchicine.

Day 21: Unconscious despite stopping sedation, EEG (Table 1) CSF analysis (Table 2) indicated COVID related encephalopathy.

Day 24: Metabolic acidosis corrected by dialysis. AF with hypotension, treated with two DC shocks and Amiodarone, reverted to sinus rhythm.

Day 29: Developed unequal pupils and low GCS. CT head showed Right acute subdural hematoma (Figure 2).

Day 30: Emergency right craniotomy and evacuation of SDH.

Day 36: Developed tachypnea. CTPA showed no evidence of pulmonary embolism.

Day 62: Oliguria with volume overload. Commenced dialysis with vasopressor support.

Day 66: Rising inflammatory markers and worsening chest x-ray.

Day 72: Hypotension and bradycardia followed by PEA. Patient died despite resuscitation.

Table 1: EEG findings.

Day 31	Diffuse high amplitude, low frequency delta activity suggesting encephalopathy. No epileptiform discharge
Day 46	Generalized delta waves with low amplitude. No epileptiform discharges.

Table 2: CSF analysis.

Parameter	Results	Normal Range
Protein	0.26	0.15-0.45
Ig G	59	0-34
Glucose	10.32	2.22-3.89
WBC	2	<5
RBC	3	<2
Microbiology	Clear Fluid, No bacterial growth, Virology negative	

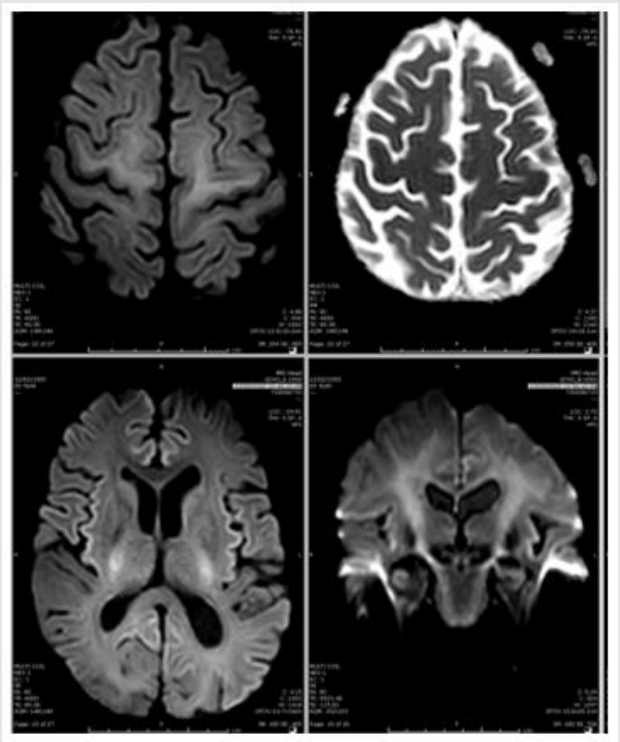

Figure 1: Non enhanced MRI brain Diffuse weighted images (show perirolandic and bilateral corticospinal hyperintensity without diffusion restriction indicating COVID 19 encephalopathy. But there was no susceptibility on SWI (susceptibility weighted imaging) indicating any hemorrhage.

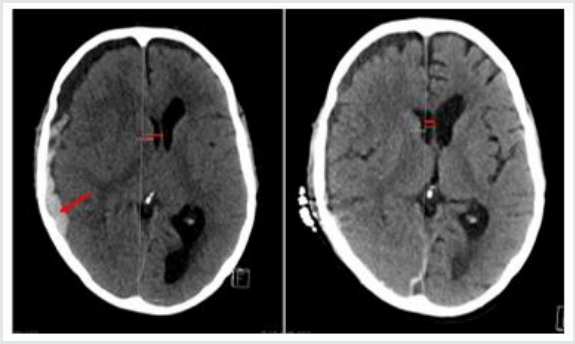

Figure 2: CT head on the left shows right acute subdural hematoma (red pointed arrow) measuring 18 mm in maximal thickness causing 13 mm leftward midline shift (red straight line). Post craniotomy CT Head on the right shows interval re expansion of the right lateral ventricle, left sided midline shift reduced to 5.5 mm (redstraight line)

Discussion

With the rising number of COVID-19 cases throughout the world, neurological involvement is being increasingly recognized as a complication associated with it. A recent systematic review of 225 case studies, showed neurological manifestations ranging from mild symptoms like anosmia, ageusia and headache to serious complications such as stroke, altered sensorium, seizures and encephalopathy [1]. The mechanisms through which SARS-CoV-2 affects the Central Nervous System (CNS) are not yet fully understood. Possible routes for viral spread are hematogenous spread, direct spread to the brain crossing the blood brain barrier and spread through neuronal pathways [2]. The target receptor for the coronaviruses is the angiotensin-converting enzyme-2 (ACE 2) receptor which is also found in the glial cells of the brain and spinal cord tissues. The other mechanisms of brain injury include cytokine-mediated systemic inflammation (cytokine storm) and hypoxic brain injury.

Clinical picture of COVID-19 related neurological manifestations include polyneuropathy, encephalitis and acute ischemic or hemorrhagic cerebrovascular events involving both peripheral and central nervous system [3]. COVID-19 ischemic strokes are more common than hemorrhagic strokes, and the average time from diagnosis of COVID-19 to an ischemic stroke in one study was about 10 days [4]. A panel of experts from World Stroke Organization reported the risk of ischemic stroke during COVID-19 to be around 5% [5]. Intracranial hemorrhage in COVID-19 disease has been linked to cytokine storm or coagulation abnormalities [6]. COVID-19 is associated with a prothrombotic state and severity of this is linked to the elevated D-dimer levels [7]. Apart from coagulation abnormalities, the other laboratory parameters which are reported to be deranged in COVID-19 patients with neurological involvement include elevated levels of Interleukin-6, procalcitonin, C-reactive protein and blood urea nitrogen. decreased lymphocytes and platelets compared to patients without CNS manifestations [8]. Our patient also had deranged clotting profile, raised CRP, raised procalcitonin high D-dimer and thrombocytopenia.

The diagnosis of COVID-19-related encephalitis can be challenging as the definitive confirmation of viral encephalitis depends mostly on isolation of virus from cerebrospinal fluid (CSF). This is extremely difficult or almost impossible as SARS-CoV-2 virus dissemination is transient and it's CSF titer may be very low [9]. CSF analysis was done in our patient which revealed normal cell count and no organism was isolated. Two case series have been reported which involved analysis of CSF from 12 patients where CSF had no white blood cells and the PCR assay for this virus was negative [10,11]. Apart from encephalitis, neurological complications include hemorrhage and infarction. In a multicenter retrospective observational case series, 18 patients with COVID-19 infection developed intracranial bleed within 11 days. Out of these 6 had Intracerebral Hemorrhage (ICH), 11 had Subarachnoid Hemorrhage (SAH) and 1 had Subdural Hemorrhage (SDH) [12]. ICH and SAH could possibly be linked to arterial hypertension which can be induced by binding of SARS-CoV-2 to ACE2 receptors, and thrombocytopenia [13]. ICH is linked with high mortality rates and hence, patients who are considered as high risk with multiple co-morbidities, previous history of aneurysms and on anti-coagulants, need to be identified, and early interventions can improve their outcome [14].

It has been reported that traumatic SDH can be a rare presenting feature of underlying COVID-19 infection [15]. But there was no history of trauma in our patient and in fact the SDH in this case was spontaneous. Atraumatic SDH can be associated with anticoagulants but our patient was not on them. There have been reports of association of SDH with coagulopathy which could be inherited as well as acquired [16]. COVID related coagulation abnormality is a well-recognized complication especially in patients on intensive care [17] and this could explain the reason for the development of SDH in our patient. Radmanesh et al reported a large retrospective observational case series involving 241 COVID-19 patients who underwent CT or MRI of the brain. The most common radiological findings were nonspecific white matter microangiopathy (55.4%), chronic infarct (19.4%), acute or subacute ischemic infarct (5.4%),

and acute hemorrhage (4.5%). White matter microangiopathy was associated with higher 2-week mortality in this study [18]. In our patient initial imaging showed age related involutional changes with periventricular leukoencephalopathy secondary to microangiopathy. He had a repeat CT scan 16 days later in view of anisocoria and reduced GCS, which showed an acute spontaneous right frontal Subdural hematoma with midline shift. Subsequent imaging later also revealed features suggestive of meningoencephalitis. The treatment of COVID-19-related encephalitis is mainly supportive. A variety of treatments, including high-dose IV steroids, IV immunoglobulin, and immunomodulators (e.g., rituximab), have been tried in various cases, with somewhat limited outcomes [19]. Treatment of SDH is either Craniotomy with evacuation of hematoma or burr hole insertion with insertion of drain [20].

Conclusion

Subdural hematoma as a complication of COVID-19 is extremely rare and is a part of the wide spectrum of neurological conditions seen in such patients. COVID-19 related neurological manifestations are being increasingly recognized but questions remain unanswered about the frequency and severity of neurological symptoms, the underlying etiology and sequence of disease progression. Hence further research is needed to understand the neurological complications of COVID-19. Patients with COVID-19 should be evaluated early for any neurological involvement, and timely workup is crucial to reduce subsequent morbidity and mortality.

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