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CASE REPORT: A 74-YEAR-OLD COVID-19 PATIENT PRESENTING WITH HYPERGLYCAEMIA, STROKE AND GUILLAIN BARRE SYNDROME

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ABSTRACT

Covid-19 infection is an airborne infection caused by SARS-CoV-2, a positive stranded RNA virus. The disease which started in Wuhan a commercial city in China rapidly spread around the globe causing a pandemic. It presents mainly with pneumonitis, but may have other systemic affectations.

We highlight this case of a seventy-four-year old man with no background medical condition who developed severe form of the infection complicated by hyperglycaemia, stroke and post infectious polyneuropathy. He achieved full clinical recovery with the limited available resources.

KEYWORDS: Covid-19, Infection, Hyperglycaemia, Stroke, Polyneuropathy.

INTRODUCTION

In December2019, a novel coronavirus started as an emerging pathogen for humans and resulted in a pandemic. SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2), the virus causing coronavirus 2019 (COVID-19), is a positive-stranded RNA virus, similar to other coronaviruses[1].

The disease, being mainly airborne and spread by coughing and sneezing, has continued to spread rapidly around the globe. The total number of cases at this time is likely much greater than official diagnoses, as many may not know they are infected[2]. Mild cases may be asymptomatic or experience vague symptoms which patients do not consider severe enough to warrant testing, but these cases are still capable of transferring the virus to others, for whom the disease may be severe or even deadly. The illness caused by SARS-CoV-2 may have wide range of presentations ranging from asymptomatic illness to respiratory distress syndrome, septic shock, multi-organ dysfunction and death[2,3].

In the face of this pandemic where knowledge, facilities for diagnosis and management is limited it is pertinent that the care giver must take adequate precaution in order to stay safe and remain alive so as to continue to carry out his duty.

CASE SUMMARY

We present Mr. AO, a seventy-four-year old clergy who was referred to us from a mission hospital with history of fever, cough and weakness of one-week duration and shortness of breath of two days prior to presentation. Fever was high grade and persistent and only relieved mildly by antipyretics and tepid sponging. The cough was initially dry, but later became productive of brownish sputum. He had no history of haemoptysis and no contact with patient with chronic cough. He had associated breathlessness but no paroxysmal nocturnal dyspnoea or orthopnoea. He had progressive weakness to the extent that he hardly fed himself.

He had never smoked cigarettes and did not take alcohol. He had served as a priest of the Catholic Church and a lecturer in the university. He had no previous medical condition and had no family history of diabetes, hypertension or asthma.

Clinical examination revealed an elderly man who was febrile (temp. 37.7°C), with mild central cyanosis, mildly dehydrated, not pale, anicteric and no leg oedema.

Random blood glucose (RBS) on admission was 164mg/dL.

Chest findings included a respiratory rate of 30 cycles per minute, with oxygen saturation (SpO_2) of 76% in room air. He had dull percussion notes and coarse crackles in all lung zones. He had bronchial breath sound and no wheezes on auscultation.

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Cardiovascular examination revealed a pulse rate of 98 beats per minute and regular. The blood pressure was 110/70 mmHg with first and second heart sound and no heart murmurs. The jugular venous pulsation was not elevated.

He had mild distended abdomen with normal liver size and span and non-tender splenomegaly of 4cm below the left costal margin. There was no ascites.

The central nervous and musculoskeletal systems were normal.

A working diagnosis of severe bronchopneumonia (atypical) with differential diagnoses of SARS-CoV-2 infection and severe sepsis with focus in the lungs was made.

Investigations were requested for including FBC, ESR, MP, Real-Time PCR for SAR-CoV-2, S/EUCr, Chest X-ray, blood culture, Urinalysis and urine culture.

Drug treatment included:

IVF Normal Saline 1L 8 hourly x 48 hours.

IV meropenem 1g 12 hourly x 96 hours.

Tab Azithromycin 500mg daily x 5 days.

IV Dexamethazone 8mg stat, the 6mg 12 hourly x 48hours, then change to PO 4mg 12 hourly.

Tab Ivermectin 12mg stat.

IM paracetamol 600mg PRN.

Commence intra-nasal oxygen 8 Litres per minute over 4 hours, then review.

On day 2 of admission, the patient was still breathless but the SpO_2 had improved to 88% in room air and the body temperature was 38°C. The fasting blood glucose was 194mg/dl.

He was given a stat dose of IM paracetamol 600mg and tepid sponged.

Tabs metformin 500mg bd and tabs glimepiride 2mg daily were prescribed.

Subcutaneous dose of enoxaparin 40mg was also commenced.

On day 3 the FBG rose to 285mg/dl, with pulse rate of 90/min, Bp of 130/90mmHg and SpO_2 of 91% in room air. Tab Metformin was increased to 1g bd and oral anticoagulant epixaban at 5mg bd was introduced.

By day 5, the patient felt stronger and fever had stopped. The oxygen saturation in room air was 92% and FBG was 175mg/dl. Glimepiride tablet was increased to 4mg and other treatment schedules continued.

On day 6, the patient had a sudden relapse with onset of breathlessness, right sided weakness, slurred speech and urinary incontinence.

The FBG rose to 195mg/dl. Clinical review revealed a conscious but drowsy patient with right facial nerve palsy, with grade 3 power in the right upper and lower limbs. The SpO_2 dropped to 88% in room air. Intranasal oxygen was commenced at 6L per minute.

Clinical assessment of a left hemispheric CVA with right hemiparesis and right facial nerve palsy, to rule out a transient ischaemic attack of possible embolic in origin was made.

Interventions done included to stop all oral hypoglycemic drugs and replace with subcutaneous soluble insulin 8IU 8hourly. Oral anticoagulant apixaban was also stopped and replaced with tablet dabigatran 110mg 12hourly. Fluid management was enhanced with one litre of normal saline 8 hourly for 48 hours. Tablet Dexamethasone was reduced to 2mg bd and subsequently tailed off. Urine culture result was reviewed and IV Ciprofloxacin commenced based on sensitivity result.

On day 7 the patient had remarkable clinical improvement within 24hours, power in right upper and lower limbs were optimal. The FBG had dropped to 179mg/dl and the SpO_2 had improved to 93% in room air. He had temperature spikes and felt weak. The plan was to continue soluble insulin and commence physiotherapy and incentive spirometry.

By day 11, the patient had sustained improvement. He had remained afebrile for 72hours and his oxygen saturation has improved to 96% in room air. Other vital signs were stable. Soluble insulin was stopped and tablets Metformin/sitagliptin combination at dose of 500/50mg was introduced.

On day 13, the patient still had sustained improvement but with FBG of 167mg/dl and SpO_2 of 96 – 97% in room air. Also, he had developed weakness in both lower limbs and was unable to ambulate. Power in the lower limbs was 2/5 while both upper limbs were 5/5. An assessment of Guillain Barré syndrome was made.

The plan was to intensify physiotherapy and Tablets Magnesium 200mg daily commenced. A course of intravenous immunoglobulin 1g/kg/day for two days was given.

Between days 18 – 20, the FBG was on the downward trend from 95 to 65mg/dl. He now ambulated with support. All diabetic regimens were stopped.

By day 25, the patient had no new complaints. He had started ambulating without support and his oxygen saturation was 98% in room air. He was discharged the same day and was given an appointment for two weeks.

By 8 weeks post-discharge, the patient had fully recovered, power was optimal in both lower limbs and could ambulate without support. He has been seen at three months and six months and he is still very healthy.

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INVESTIGATIONS				
FULL BLOOD COUNT	02/07/20	08/07/20	17/07/20	NORMAL
HB (g/dL)	12.3	13.6	13.3	(13 – 17)
PCV (%)	37	41	40	(37 – 50)
TOTAL WBC (c,mm)	12.2	15.5	18.4	(2 – 7 x 10^9/L)
NEUTROPHILS (%)	66	78	70	(40 – 75)
LYMPHOCYTES (%)	25	20	22	(20 – 45)
MONOCYTES (%)	06	07	05	(2 – 10)
EOSINOPHILS (%)	03	03	03	(1 – 10)
ESR WG (mm/hr)	72	66	52	(0 – 7)

SE/U/Cr	02/07/20	08/07/20	17/07/20	NORMAL
UREA	10	13	13	(7 – 23) mg/dl
Na^+	136	141	139	(136 – 139) mmol/L
K^+	4.4	5.8	4.6	(3.5 – 5.5) mmol/L
Cl^-	98	112	110	(95 – 110) mmol/L
Creatinine	0.8	0.8	0.9	(0.6 – 1.4) mg/dL

URINE ANALYSIS
02/07/20
Macroscopy - amber and cloudy SG 1.010, protein +, pH 5.0
Microscopy - Epithelial cells +, WBC 6-8/ hpF, Uric acid ++, Debris +, Bacteria +
Urine Culture - yielded moderate growth of *E. coli* sensitive to ciprofloxacin 3+, perflacin 3+, Erythromycin +, streptomycin 2+, Rocephin 3+. Resistant to septrin, zinnacef.
17/07/20
Repeat Urine Culture yielded no growth.
ABDOMINO/PELVIC ULTRASOUND showed normal study.

DISCUSSION

In December2019, a novel coronavirus started as an emerging pathogen for humans and resulted in a pandemic. A novel member of the human RNA coronavirus which is an enveloped betacoronavirus, it has been termed the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2)[2,3]. The illness caused by SARS-CoV-2 – referred to as human virus disease 2019 (COVID-19) – ranges from asymptomatic illness to acute respiratory distress syndrome, septic shock, multi-organ dysfunction and death. The most common symptoms include fever, fatigue, dry cough, dyspnoea and diarrhoea[3]. Neurological manifestations have also been reported. Profound coagulopathies may manifest with ischemic or haemorrhagic stroke. Rarely, SARS-CoV-2 virus encephalitis or pictures of acute disseminated encephalomyclitis or acute necrotizing encephalopathy have been reported[4,5].

This report presents the case of a seventy–four-year old man who was previously healthy and had no co-morbidity. He was diagnosed of severe pneumonia and was responding poorly to treatment which necessitated the referral. The timing of admission coincided with the peak of the COVID-19 pandemic (June 2020) and accounted for the reason we considered COVID-19 infection as the likely aetiology for the pneumonia. A few days into admission, his blood glucose started rising necessitating introduction of oral hypoglycaemic agents. It has been documented that acutely ill patients suffering from SARS-CoV-2 infection often present with hyperglycaemia caused among other factors by endogenous stress induced glucocorticoid hypersecretion[7]. In preliminary reports of clinical presentation of patients with novel COVID-19 infection, 51% of them had hyperglycaemia[8]. Transient hyperglycaemia had also been reported in patients with SARS (Severe Acute Respiratory Syndrome) in 2003, caused by the coronavirus which is closely related to the cause of COVID-19, SARS-CoV-2[9]. In addition, the enzyme dipeptidyl peptidase 4 (DPP-4), which physiologically is implicated in the modulation of insulin action plays a role in the glucose metabolism and is responsible for the degradation of incretins such as glucagon-like peptide-1 (GLP-1). Thus, it is suggested that the hyperglycaemia seen in patients with COVID-19 may result via such analogous mechanisms[10,11].

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This needs to be further investigated. As seen above, in the management of this patient, response to oral hypoglycemic agents was poor, but the patient's response to soluble insulin was better supporting the work done by Hamdy et al that insulin therapy should be the treatment of choice for critically ill patients with COVID-19[12]. The main aim of a safe and effective regimen should be to reduce contact frequency, reduce glucose variability, minimize risk of hyperglycaemia and improve glycaemic control[12]. Inpatient hyperglycaemia during the pandemic has been associated with worse outcomes, so it is mandatory to implement effective glycaemic control treatment[12,13]. The American Diabetes Association (ADA) proposes a range of 140 – 180 mg/dl (7.8 – 10.0 mmol/L) as a target level for the majority of non-critically ill patients[14].

Our patient had neurological deficits that presented as right-sided weakness, slurred speech and right facial nerve palsy, and lasted less than twenty-four hours. It has been documented that characteristic cytokine storm incites severe metabolic changes and multiple organ failure[15]. This in turn, manifest with profound coagulopathies with ischaemic or haemorrhagic stroke. Rarely, SARS-CoV-2 virus encephalitis or pictures like acute disseminated or acute necrotizing encephalopathy have been reported [4,5]. Our patient had an ischaemic stoke or, possibly, a transient ischaemic attack which occurred in the second week of presentation. It is also noteworthy that our patient did not have a vascular risk factor. This agrees with the work reported by Ahmadi et al[16] in which more than half of the patients did not have cardiovascular risk factors. He was quickly revived with fluid management using normal saline, antiplatelet drug and anticoagulant therapy. Initially he was started on subcutaneous injection of enoxaparin 40mg daily. Later apixaban was added with subsequent withdrawal of enoxaparin. In spite of the anticoagulation he developed a right sided weakness and seventh facial nerve weakness which was probably a thromboembolic event. We had to stop the apixaban and replace with dabigatran 110mg twice daily. The reason for stopping the apixaban was just due to lack of enough experience in the use of apixaban as many of our patients have been on dabigatran.

Dabigatran is a member of the Direct Thrombin Inhibitors (DTIs), a novel group of oral anticoagulants used in the management of venous thromboembolism (VTE). It is administered orally and requires little or no monitoring[17].

About the time we were contemplating discharging the patient, he developed sudden paraparesis of the lower limbs. Clinical review following neurological consultation suggested Guillain Barré syndrome (GBS). This syndrome is an inflammatory polyneuropathy associated with many viral infections including COVID-19 infection and post-infectious, immune-mediated inflammatory processes have been held responsible[18]. Recently, there have been case reports describing the association between COVID-19 and GBS but a lot remains unknown about the strength of this association and the features of GBS in this setting[18]. The mean age of patients presenting with GBS in COVID-19 was 59 years, with males presenting at a mean age of 65 years. The mean time from COVID-19 symptoms to symptoms of GBS was eleven days[18]. Our patient was 74 years and presented with features of GBS about the 13th day of admission. Electrophysiological assessment and/or cerebrospinal fluid examination are needed to confirm the diagnosis of Guillain Barre syndrome, but given the clinical condition of the patient, available resources and safety of the managing team, we did not do all these. Our patient had a single course of intravenous immunoglobulin with subsequent improvement in symptoms and, finally, had significant clinical improvement after 8 weeks. This is in line with many other documented cases of GBS in COVID-19 infection[18]. Barefoot medicine still has a great role in the management of patients when sophisticated equipment is not available or cannot be employed when the safety of the managing team cannot be guaranteed. We should not be left handicapped but resort to clinical judgments when facilities or other circumstances limit us from pinning down the diagnosis.

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