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Case Report

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Palpitations in the Pandemic

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Abstract

The current pandemic caused by COVID, a primary respiratory pathogen, has resulted in a number of neurological manifestations following the infection. Reports of COVID positive patients with meningoencephalitis, Guillain Barre Syndrome and cerebrovascular accidents have surfaced over the past few months. Our patient, who developed respiratory involvement following infection with COVID, developed dysautonomia in the recovery phase, manifested as Postural Orthostatic Tachycardia Syndrome (POTS). The abnormal sympathetic skin response from lower limbs and an abnormal 30:15 ratio of RR interval in standing position shows the probable neuropathic aetiology of his POTS. Our case highlights the fact that this virus has multiple ways of affecting the nervous system and that a complaint of palpitations in a patient with COVID infection may be a neurological manifestation. In the scenario of the COVID pandemic and we need to be vigilant about dysautonomia as a probable complication of this infection.

Keywords

COVID, Dysautonomia, POTS

Key Message

The current COVID pandemic has many neurological manifestations, one of which is dysautonomia, which can present as postural orthostatic tachycardia syndrome. As practicing neurologists, being unaware of this possibility would lead to diagnostic delimma.

Introduction

Neurological manifestations of the current COVID 19 infection are myriad, with predilection of the virus to elicit symptoms in multiple areas of the neuraxis. Autonomic nervous system can be affected in COVID 19 infection due to immune mediated response or direct infection [1]. Central or peripheral autonomic nervous system can be affected [2]. We report the case of a 32-year-old neurology resident, who was hospitalised with COVID 19 infection and developed respiratory involvement, following which he developed dysautonomia and postural orthostatic tachycardia syndrome.

Case History

A 32-year-old male, neurology resident, presented initially with low grade fever, sore throat and cough; COVID 19 RTPCR testing turned out to be positive and he was so admitted in the COVID isolation area from the second symptomatic day. On the day following admission, the sore throat and fever subsided, but he had increasing cough and was started on oral dexamethasone and antiviral Favipiravir along with supportive medications. He developed anosmia by fifth day of admission. By the sixth day he developed dyspnoea with tachypnoea but saturation was maintained at 95 percent on room air. A CT chest revealed scattered patches of predominant subpleural and peripheral consolidation in bilateral lower lobes and left upper lobe. He was switched over to remdesvir; anticoagulation and steroids were continued.

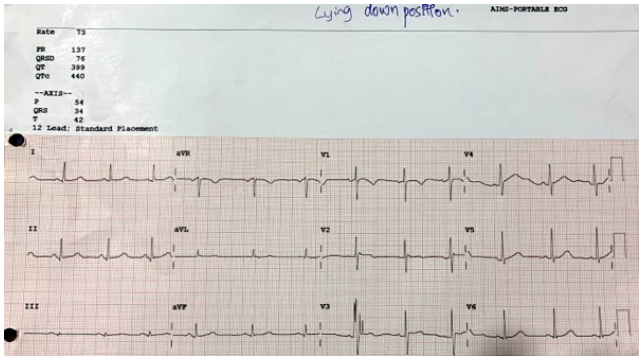


Figure 1: EEG in supine state: Heart rate 73 per minute.

By eighth day, he felt episodic palpitations, unrelated to posture, without any fall in blood pressure. An EKG confirmed sinus tachycardia. A screening ECHO did not show any evidence of myocarditis. By day nine, he was found to have a tachycardia that was triggered by upright posture, associated with dizziness, giddiness and fatigue when upright for longer periods and that reverted on being supine. At this point of time, there were no other symptoms of dysautonomia, including blurred vision, sudomotor symptoms, or gastrointestinal symptoms. Clinical examination revealed no evidence of dehydration and the heart rate was found to fluctuate between 70-80 beats per minute in supine position and 110-130 beats per minute on standing position. ECG on day eleven showed supine heart rate 73 per minute, 116/minute at two minutes and 115 at fifth minute of standing. (Figure 1 and 2) He was hydrated and it was ensured that there were no medications

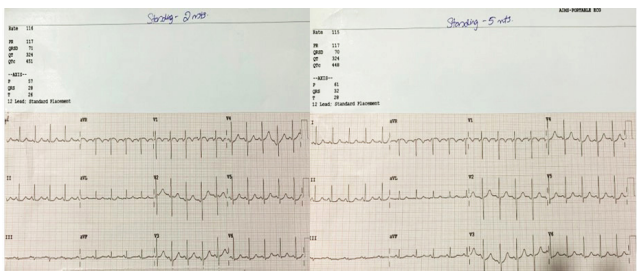


Figure 2: EEG at second and fifth minute of standing: Heart rate 116/min and 115/min respectively.

that could cause tachycardia. A diagnosis of symptomatic POTS was made. He was not started on drugs for POTS. His postural giddiness improved over time but postural tachycardia persisted. On the twelfth day he became COVID negative and a tilt table test was done on the thirteenth day (Figure 3), which again documented the orthostatic tachycardia, with baseline heart rate around 70/minute, that increased to 105 per minute by the eighth minute, and persisted throughout the recording; however, he remained asymptomatic.

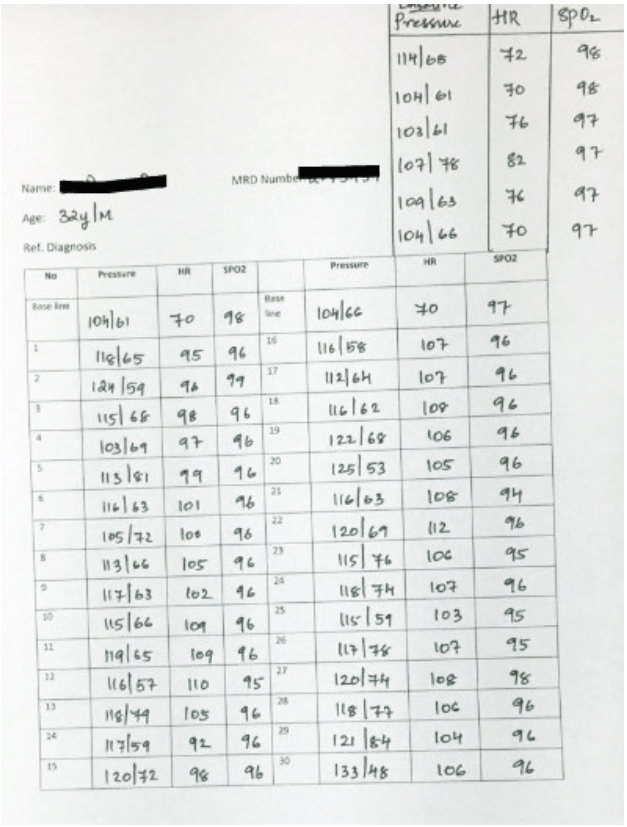


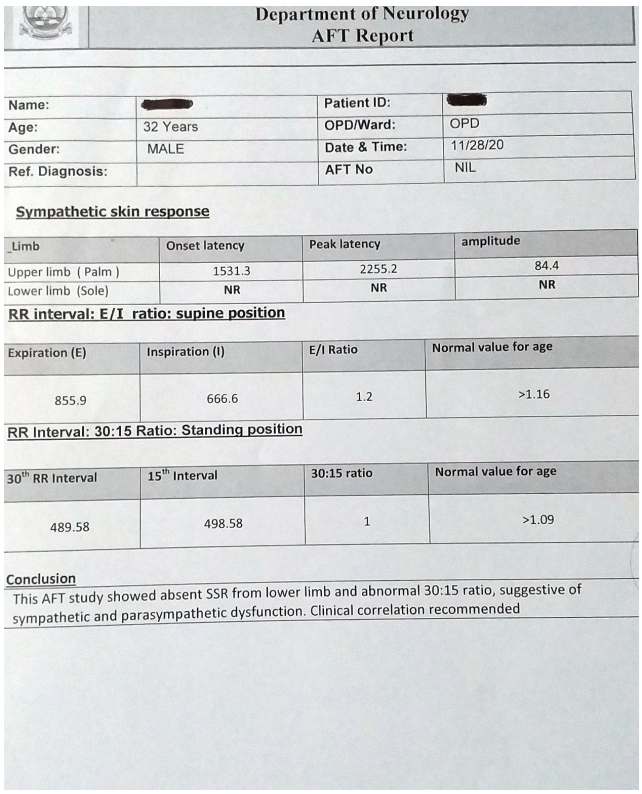
Figure 3: TILT table test- Heart rate increased by 30 beats per minute, from baseline, from sixth minute of upright TILT.

An autonomic function test done in our neurology lab revealed dysautonomia in the form of absent sympathetic skin response from the lower limb, as well as parasympathetic dysfunction in the form of an abnormal 30:15 ratio of RR interval on standing (Figure 4).

His past history was remarkable for Immune Thrombocytopenic Purpura (ITP) in 2009 for which he took steroids for more than a year, with two relapses over the past 10 years, which required 6-month course of steroids each. The last episode was three years back and he is off steroids since the past 2.5 yrs. He also had a childhood history of bronchial asthma. He has no history suggestive of any cardiovascular involvement in the past, and he was not on any regular medications.

Discussion

The current pandemic of COVID 19, a primary respiratory disease, has been recognised to have neurotropic potential. Various neurological manifestations have been reported till



Department of Neurology
AFT Report

Name:	[redacted]	Patient ID:	[redacted]
Age:	32 Years	OPD/Ward:	OPD
Gender:	MALE	Date & Time:	11/28/20
Ref. Diagnosis:		AFT No	NIL

Sympathetic skin response

Limb	Onset latency	Peak latency	amplitude
Upper limb (Palm)	1531.3	2255.2	84.4
Lower limb (Sole)	NR	NR	NR

RR interval: E/I ratio: supine position

Expiration (E)	Inspiration (I)	E/I Ratio	Normal value for age
855.9	666.6	1.2	>1.16

RR Interval: 30:15 Ratio: Standing position

30th RR Interval	15th Interval	30:15 ratio	Normal value for age
489.58	498.58	1	>1.09

Conclusion

This AFT study showed absent SSR from lower limb and abnormal 30:15 ratio, suggestive of sympathetic and parasympathetic dysfunction. Clinical correlation recommended

Figure 4: Autonomic function test absent sympathetic skin response from lower limbs and abnormal 30: 15 ratio of RR interval on standing (normal > 1.09; patient value is 1).

date that includes meningoencephalitis, stroke, Guillain-Barré syndrome and anosmia [1]. The virus has been isolated from CSF and from brain parenchyma of patients with neurological manifestations [2]. Reports of autonomic nervous system involvement is rare; we report a patient with COVID who developed POTS secondary to autonomic nervous system dysfunction.

Our patient satisfied the diagnostic criteria POTS: an increase in heart rate of 30 beats/min or more when moving from a recumbent to a standing position that lasts more than 30 seconds (or ≥ 40 beats/min in individuals 12-19 year of age) in the absence of orthostatic hypotension [3]. POTS can be primary and secondary based on etiology. Secondary POTS is due to autonomic nervous system involvement in diabetes mellitus, amyloidosis, Sjogrens, or paraneoplastic syndrome [4].The abnormal autonomic function test showed that POTS in our patient was most likely due to autonomic dysfunction. Common symptoms include light-headedness, blurred vision, weakness, cognitive difficulties and fatigue and are often accompanied by palpitations, shortness of breath, syncope, or gastrointestinal symptoms [5] and our patient had few of above-mentioned symptoms on upright posture.

Since our patient had a history of immune mediated disorders in past like ITP and bronchial asthma, probable immune triggered autonomic dysfunction was most likely. In addition, the development of a POTS in the second week of illness, rather than in the initial phase of viremia, also lends strength to this possibility. The supine norepinephrine is often high normal in patients with POTS, while the upright norepinephrine is usually elevated (> 600 pg/ml), a reflection of the exaggerated neural sympathetic tone that is present in these patients while upright. We could not assay serum norepinephrine level due to technical issues. Preferential denervation of sympathetic nerves innervating the lower limbs is postulated as a cause for neuropathic POTS [6] and central hyperadrenergic POTS is thought to be due to excessive sympathetic discharge.

Treatment of POTS include non-pharmacological interventions and drug therapy. Non pharmacological interventions include exercise, excess fluid and salt intake. Various drugs have been tried including fludrocortisone, beta blockers, selective serotonin reuptake inhibitors and pyridostigmine. Drugs precipitating tachycardia should be avoided in such patients. Our patient was not started on any medication for POTS and he gradually improved.

This is probably the first report of POTS reported in a COVID patient from India. There are reports of POTS during COVID infection reported elsewhere [7]. This report adds a new neurological disorder to the expanding list of nervous system complications in patients with COVID.

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