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CASE REPORT

Infectious Disease

Dizziness can be an early sole clinical manifestation for COVID-19 infection: A case report

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Abstract

SARS-CoV-2 is a novel strain of coronavirus that was first identified in Wuhan, China; it has since spread rapidly throughout the world. Most of the patients with COVID-19 present with respiratory symptoms, including cough, nasal symptoms, fever, and shortness of breath. However, several groups have reported that SARS-CoV-2 can infect the central nervous system via the olfactory bulb followed by spread throughout the brain and peripheral nervous system. This brief report illustrated a 78-year-old man who presented to the emergency department (ED) on March 22, 2020, with chief complaints of dizziness and unsteadiness while walking. He had no symptoms suggestive of COVID-19 on arrival. SARS-CoV-2 nasopharyngeal swab test performed at that time due to his atypical presentation and lymphocytopenia was positive for virus nucleic acids. The neurological symptoms associated with COVID-19 are frequently non-specific and may emerge several days before the respiratory symptoms; as such, identification of patients presenting with these subtle and seemingly unremarkable COVID-19 symptoms will be quite difficult. Added to this, numerous countries still limit testing for SARS-COV-2 to patients presenting with fever or respiratory symptoms. Frontline physicians should be aware of early, non-specific symptoms associated with SARS-CoV-2 infection.

KEYWORDS
case report, COVID-19, dizziness, lymphocytopenia, neurological symptoms, SARS-CoV-2

1 | INTRODUCTION

SARS-CoV-2, a novel single-stranded RNA, was first identified in Wuhan, China, spreading rapidly throughout the world. COVID-19, caused by SARS-CoV-2, has been associated with substantial morbidity and mortality. The virus interacts with the angiotensin-converting-enzyme 2 receptor in alveolar cells, cardiac myocytes, and the vascular endothelium causing different clinical presentations, notably respiratory distress.[1] Before early March 2020, little was known about the aggressive neurotropic and neuro-invasive capabilities of this virus.[2] Several groups have reported that SARS-CoV-2 has the capacity to infect the central nervous system via the olfactory bulb; from there, it can spread to the brain and peripheral nervous system via a synapse-connected route.[3,4] In a mouse model, SARS-CoV-2 preferentially infects neurons in the brainstem wherein is the critical factor that regulates cardiopulmonary function; infection at this site may lead to significant cardiopulmonary dysfunction. Infection of the central nervous system may explain some or all of the cardiopulmonary symptoms

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associated COVID-19 that can be detected when chest radiographs remain relatively clear.[5] In this report, the patient presented with non-specific neurological symptom few days before his respiratory symptoms appeared.

2 | CLINICAL SUMMARY

The patient is a 78-year-old man who presented to the emergency department on March 22, 2020, with chief complaints of sudden onset of dizziness and unsteadiness while walking on the street. He reported having a spinning sensation and tendency to fall when attempting to walk. The patient's wife confirmed that he was not able to walk in a straight line. He reported no additional neurological symptoms, including spurring of speech nor any limb weakness. At presentation, he had neither fever nor any upper respiratory tract symptoms; he denied chest pain, shortness of breath, or gastrointestinal complaints. He had neither travel history nor any known contact with persons visiting from other countries.

His past medical history was notable for Alzheimer's disease treated with both memantine and donepezil. He also had a history of hypertension and coronary artery disease that was treated with a coronary artery bypass graft procedure in 2014. His current medications include aspirin (81 mg/d), atorvastatin (20 mg/d), donepezil (10 mg/d), levothyroxine (88 μg/d), and ramipril (5 mg/d). On arrival, his vital signs included blood pressure at 122/74 mm Hg, heart rate at 109 beats/min, and body temperature at 37.2°C. His respiratory rate was 16 breaths/min with an oxygen saturation of 92% on room air with no signs of respiratory distress.

Physical examination revealed no symptoms suggestive of acute stroke. His pupils were equal and reactive, and there was no slurring of speech or drooling. His cranial and peripheral nerve examinations were both grossly within normal limits. At presentation, he was able to walk independently without symptoms, although he noted that this resulted in fatigue. Past-pointing and heel-to-shin tests were not performed due to issues associated with Alzheimer's disease. Cardiovascular, respiratory, and abdominal examinations were unremarkable.

Laboratory tests were notable for a normal white blood cell count with lymphocytes at 0.9×10^9/L (normal range $1.0–4.0 \times 10^9$/L). His electrolytes, renal function values, electrocardiogram, non-contrast head computed tomography, and urinalysis were normal. His liver enzymes were also unremarkable except for alanine transferase at 41 U/L (normal <36 U/L). C-reactive protein was also elevated at 44 mg/L (normal <7.5 mg/L). Prothrombin time (PT) and partial thromboplastin time (PTT) were normal. Chest radiograph revealed a subtle right lower lobe infiltrate that raised the suspicion of early-stage pneumonia. At the date of his presentation, SARS-CoV-2 nucleic acid testing was recommended only for patients presenting with fever, respiratory symptoms, and/or a positive travel history. Nevertheless, because of his unusual presentation, including oxygen saturation at 92% on room air, lymphopenia, increase in C-reactive protein, and abnormal alanine transferase, a nasopharyngeal swab test for SARS-CoV-2 was performed. Because his condition was stable, and he exhibited no respiratory distress, he was discharged on levofloxacin for presumptive early-stage pneumonia as per the abnormal radiography finding. Self-isolation was recommended together with instructions to return to the ED if any of his symptoms worsened. At ~30 h later, the result of his SARS-CoV-2 test returned as positive. Three days later, the patient returned to the ED reporting gradual onset of shortness of breath, increase in confusion, and general weakness. There was no deterioration of his neurological symptoms. However, he required 3 L of oxygen to maintain an oxygen saturation of 94%. Chest radiograph revealed mildly increased infiltrates, now detected in both lower lobes of his lungs. He was admitted from the ED to the COVID-19 ward; levofloxacin was discontinued and piperacillin-tazobactam was initiated. He did not receive any anti-viral treatment. During his hospitalization, he never complained of cough or sputum. His condition improved and no oxygen was required 5 days after hospitalization. His conscious status restored back to his baseline dementia. He was kept in the hospital for 2 main reasons. First, he was not able to remember to stay isolated because of his dementia. His daily activity was solely dependent on his frail wife. Second, discharging the patient would put his wife and the community in danger. Of note, his SARS-CoV-2 swab test remained positive for 41 days. He was discharged on May 5, 2020, after 2 negative tests on 2 consecutive days according to the guideline suggested at that time.

TABLE 1 Central and peripheral nervous system disorders associated with COVID-19

Central nervous system	Peripheral nervous system
Acute demyelinating encephalopathy	Gullain-Barré syndrome
Acute ischemia stroke	Mixed axonal neuropathy
Ageusia	Miller-Fisher syndrome
Anosmia	Myopathy
Ataxia	Non-specific paresthesia
Confusion	Polyneuropathy
Cranial nerve palsy	Polyneuritis cranialis
Dizziness or visual disturbance	
Headache	
Intracerebral hemorrhage	
Meningitis	
Necrotizing hemorrhagic encephalopathy	
Seizure—generalized/focal	
Toxic encephalopathy	
Vertigo	
Viral encephalitis	

3 | DISCUSSION

Neurological symptoms caused by COVID-19 are diverse and complex (Table 1). Non-specific symptoms including headache, dizziness, vertigo, and paresthesia have been reported.[6] The most frequent one is

anosmia. Some patients develop respiratory symptoms several days (median, 1–2 days) after the emergence of non-specific neurological symptoms, including headache and dizziness.[7]

SARS-CoV-2 may use a variety of pathophysiological mechanisms to induce central nervous system dysfunction, including direct invasion, hypoxic insult, hypercoagulopathy, and immune-mediated injury.[3] Avulaa et al reported 4 cases of acute stroke associated with COVID-19.[8] According to Li et al, the incidence of this complication is ~5% at a median age of 71.6 years.[5] It is postulated that SARS-CoV-2 may cause vascular endothelial dysfunction via mechanisms that include hypoxic insult and a hypercoagulable state, both of which encourage the formation of thromboemolism.[1,9] These pathophysiological mechanisms lead to acute ischemic stroke, pulmonary emboli, or acute myocardial infarction even in the absence of respiratory symptoms.[1,9–11] Oxyley et al[12] described the cases of 5 comparatively young patients (33–47 years of age) who presented with acute large-vessel stroke (mean NIHSS score = 17) after days of cough or lethargy alone.

COVID-19-associated meningitis and encephalitis have also been reported. In early March, 2020, Moriguchi et al[13] reported a patient presented to his family doctor with a chief complaint of fever. He was sent home with a prescription for laninamivir. Later, he presented to the ED by ambulance with fever, neck stiffness, confusion, and a generalized tonic-clonic seizure that lasted for ~1 min. He was diagnosed with acute meningitis and spinal tap was performed. SARS-CoV-2 nucleic acid was detected in cerebrospinal fluid but not on the nasopharyngeal swab.

Helms et al[14] conducted an observational study that included 64 COVID-19 patients with neurological presentations. The median age in this patient cohort was 63 years and agitation was the most common presentation (69%). Only 16% of the patients experienced fever (body temperature >38.5°C), and no respiratory symptoms were recorded.

Mao et al[6] also studied the neurological presentation of COVID-19. A total of 214 patients were collected for this study from 3 designated special care hospitals. The median age of this patient cohort was 52.7 years and 36.4% had neurological symptoms. Among these patients, elderly patients, and those with underlying medical conditions, most notably, hypertension, experienced more severe infections and fewer typical respiratory symptoms. Focal status epilepticus was also reported by Vollono et al.[15]

Dizziness is a very common symptom reported by patients in a routine clinical practice. According to Guan et al,[5] the most common finding (914/1099 patients, 83%) among patients with COVID-19-associated neurological dysfunction is lymphocytopenia. Of interest, lymphocytopenia has been associated with the more severe central nervous system symptoms, including acute stroke, intracerebral hemorrhage, seizure, and encephalitis.[6,9]

To conclude, emergency physicians should be vigilant, especially when elderly patients present with non-specific symptoms and unexplained lymphocytopenia.

CONFLICTS OF INTEREST

None.

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