ABSTRACT
In December 2019, in China appeared a new infectious disease — coronavirus disease-2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Immediately it has spread worldwide. The disease manifests itself in different ways. It may be asymptomatic. It can also cause various, non-specific symptoms such as cough, fever, sore throat, rhinitis, malaise, headache, muscle pain, diarrhea, loss of smell and taste, or rash. Sometimes, the infection leads to severe pneumonia, which may cause respiratory failure and death. But there are also less frequent manifestations of the disease. For example, increasing numbers of studies reported neurological complications, such as cerebrovascular events, seizures, meningoencephalitis, encephalopathies, acute myelitis, acute facial nerve palsy, or Guillain-Barré syndrome. In our knowledge, up to now, only a few cases of Horner’s Syndrome due to COVID-19 were described. Thus, in this article, we present the case of a patient with COVID-19 pneumonia complicated by Horner’s Syndrome.

Keywords: Horner’s syndrome, COVID-19

Case presentation

A 38-year-old, right-handed male patient was referred to the Emergency Department due to ptosis, general weakness, fever, and mild headache. Co-workers of the patient noticed ptosis and urged him to go to the hospital as those “may be the symptoms of a stroke.” He denied asymmetric muscle weakness, sensory loss, or speech abnormalities. He did not complain of dyspnea, cough, or any other upper-respiratory tract symptoms. Findings from the neurologic exam, at admission, included: left ptosis, slight left pupil constriction in the dim light, and left endophthalmas. There were no other cranial nerves abnormalities, no motor deficits were present, muscle tone was normal, reflexes were symmetric, meningeal signs were negative. The ptosis did not change during the day, no apokamnosis was present. Based on the findings from the neurologic examination, left-sided Horner’s syndrome was stated as an initial diagnosis. Head computed tomography (CT) and ANGIO-CT of the cervical and intracranial arteries did not reveal abnormalities. No artery dissection was present. Due to lung inflammation noticed at the periphery of the ANGIO-CT scan, a full chest CT examination was performed and revealed ground-glass changes suggesting COVID-19 inflammation (Fig. 1). The real-time reverse transcription polymerase chain reaction (RT-PCR) test was done. Then the patient did not agree to further examination and hospitalization, and he was sent home to self-isolate before obtaining the SARS-CoV-2 test result.

The next day, the patient changed his mind and returned to the Emergency Department. He already had dyspnea and felt very weak. In addition, he reported a loss of smell and taste. Polymerase chain reaction for SARS-CoV-2 was positive, and the patient was diagnosed with COVID-19. He presented a SpO2 93% on room air.

The patient was admitted to the COVID-19 ward. He received low-flow oxygen therapy with a flow velocity of 5 L/min through the nasal cannula. Dexamethasone 6 mg was administered by intravenous drip infusion every 24 h. The patient was also treated with a unit of convalescent plasma. Enoxaparin 40 mg was added by
subcutaneous injection every 24 h. The treatment resulted in symptoms relief. After 10 days of hospitalization, the patient, in good condition, was discharged.

A control neurological examination showed a significant reduction of all symptoms of Horner syndrome. Magnetic resonance imaging (MRI) of the head with gadolinium enhancement showed no pathologies. Ultrasonography examination of the neck showed enlarged lymph nodes, possibly of reactive etiology.

**Discussion**

The potential pathophysiology, in this case, is debatable. We excluded the most common causes of Horner’s syndrome: brain stem lesions (MRI), carotid artery dissection (ANGIO-CT), mass lesions in the cervical region (USG, CT) having found no pathology.

Potential causes of Horner’s syndrome include a mass in the lung’s apex destroying the sympathetic stem — the so-called Pancoast’s syndrome — most commonly observed in patients with pulmonary adenocarcinoma. A question arises if other abnormalities in the apex of the lung, such as pneumonia, may produce similar symptoms. According to the literature, the prevalence of Horner’s syndrome in pneumonia is very rare. However, a few cases exist. Knyazer et al. [1] described a 7-month-old girl with complicated pneumonia and Horner’s syndrome. In our knowledge, up to now, only a few of cases of Horner’s Syndrome due to COVID-19 were described [2, 3]. The cause of this syndrome in the course of the disease is unclear. In our case, the ground-glass opacities reached the superior portion of the lungs. However, another possible explanation of the case may be the reactive enlargement of the cervical lymph nodes. SARS-CoV-2 is also known as a neurotropic virus, and many neurological manifestations have been described [4, 5]. One cannot exclude a possibility of direct virus action against the sympathetic tract that resolved during treatment.

Further studies are needed to determine the possible underlying mechanisms and more cases might confirm our observations.

**Conclusions**

Horner’s syndrome can be one of the symptoms of COVID-19. The pathomechanism of this syndrome is debatable and involves the inflammatory process reaching the upper part of the lungs. Another explanation could be a reactive enlargement of the cervical lymph nodes or a direct virus effect on the nervous system.

**References**