

A Case of Complete Loss of Vision After Acute Ischemia of the Visual Centers of the Occipital Lobes of the Brain due to a Novel Coronavirus Disease

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Abstract

Research and description of the features of a new coronavirus infection is an urgent task at the present time. The presented clinical case belongs to the rare section, and therefore is quite interesting from the position of a general practitioner. The incidence of acute ischemic stroke against the background of a Coronavirus disease 2019 (COVID-19) ranges from 0.4% to 8.1%, and cases of damage to the visual centers of the occipital lobes of the brain in the literature are limited to isolated cases.

Keywords: COVID-19; Cytokine Storm; Secondary Vasculitis; Cerebral Infarction; Ischemic Stroke; Visual Center

Introduction

In 2019, a novel coronavirus associated with a series of acute, atypical cases of pneumonia and respiratory symptoms was first detected in Wuhan, in the Chinese province of Hubei. Since then, the virus now known as SARS-CoV-2 (severe acute respiratory syndrome coronavirus) has spread to more than 200 countries and is still considered a major global pandemic [1]. Clinical manifestations of COVID-19 that are not related to the bronchopulmonary system, including ophthalmic and neuro-ophthalmic ones, have also been reported [2].

Approximately one third of patients hospitalized for severe COVID-19 develop macrovascular thrombotic complications, including venous thromboembolism, myocardial injury/infarction, and stroke [3]. Ischemic stroke, especially in young patients, has been one of the most severe neurological complications of COVID-19, leading to disability and death. According to reviews reporting on clinical and autopsy indicators of ischemic stroke, the incidence of its development in the case of coronavirus infection ranges from 0.4% to 8.1% [4].

There is also evidence that the prevalence of stroke in patients with COVID-19 was about 5%, with a mean age of 71.6 years. These patients had various risk factors for stroke, such as hypertension, diabetes, and coronary heart disease. On average, a stroke developed on the 12th day after infection with SARS-CoV-2.

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The mechanisms of stroke are hypercoagulability, vasculitis secondary to intracranial cytokine storm, and thromboembolic complications due to atrial fibrillation [6]. The state of hypercoagulability and subsequent thromboembolism are the most common mechanisms for the development of stroke [7].

Involvement of blood flow in the posterior pole of the eye and occipital lobes, visual field defects, and visual snow syndrome have been described in patients against the background of coronavirus infection [8].

Cases of stroke in patients with COVID-19 leading to blindness

A number of clinical cases similar to this one are described in the literature [9]. The case of a 60-year-old man with no previous risk factors for a cerebrovascular incident is described, his event was preceded by days of fatigue, cough and malaise. He had one episode of loss of consciousness and was admitted to the hospital with bilateral vision loss, altered mental status, and later tested positive for COVID-19 infection. A CT scan was performed, which showed bilateral hypodense opacities in the occipital lobes consistent with cerebral infarcts. Thus, cortical blindness secondary to occipital lobe stroke was diagnosed. The patient was then discussed for thrombolytic drugs, but due to late presentation, this therapy was not prescribed. The patient was administered broad-spectrum antibiotics, low molecular weight heparin, enoxaparin, dexamethasone [9].

M. Atum., *et al.* [10] provide data that COVID-19 can lead to bilateral ischemic stroke, which manifests itself only in the form of loss of vision. In confirmation, a clinical case is given, which describes the development of sudden bilateral loss of vision after bilateral ischemic stroke in the occipital lobes of the brain in a patient without a history of traditional risk factors for stroke. Magnetic resonance imaging (MRI) of the patient revealed acute ischemia on both sides in the posterior occipital lobes and on both sides in the cerebellar hemispheres. Treatment included antiedematous and anticoagulant therapy [10].

A case of quadrantanopia due to MRI-confirmed stroke secondary to COVID-19 infection has also been described in the literature. As a secondary prevention of stroke, acetylsalicylic acid was prescribed. The patient was regularly observed by an ophthalmologist and a neurologist. Visual acuity and neurological condition were stable. After 6 months visual acuity in both eyes was 0.9, quadrantanopia still persisted [11].

Other mechanisms of neuroophthalmic lesions

Vasculitis: Patients with severe COVID-19 infection have developed a hyperinflammatory state secondary to cytokine storm. These patients developed stroke as a result of vasculitis [12].

The literature describes a single case of neuro-ophthalmic complications - binocular diplopia on the background of vasculitis. The neurological examination of the patient was consistent with a bilateral trochlear nerve lesion. According to MR angiography, vasculitis was diagnosed that affected the vertebrobasilar system and nuclei of the fourth cranial nerve. Diplopia resolved after a 5-day course of intravenous methylprednisolone [13].

Encephalitis: B. Safarpour Lima., *et al.* [14] describe a case of post-COVID-19 encephalitis that manifested predominantly as bilateral visual loss. This case should increase the wariness of specialists about the neurological complications of COVID-19 [14].

In addition, the relationship between diseases of the posterior pole of the eye, such as central retinal vein occlusion, isolated inflammatory optic neuritis, acute bilateral demyelinating optic neuritis and SARS-CoV-2, is currently being studied. Thus, the issue of clinical manifestations of eye lesions against the background of coronavirus infection remains open [15].

Treatment

Recently developed treatments for ischemic stroke, such as intravenous thrombolysis and mechanical thrombolysis, can significantly improve the outcome of this disease. However, the effects of these treatments are highly time dependent. After the end of the therapeutic time window (4.5 hours), according to the recommendations, it is possible to use antiplatelet agents, antihypertensive drugs and statins [16]. However, the optimal treatment to prevent ischemic events in the neuro-ophthalmic complications of COVID-19 remains unclear [11].

Description of the clinical case

Complaints: Patient M., 63 years old, at admission complained of complete loss of vision in both eyes, severe weakness, unproductive cough, decreased appetite, memory loss, episodes of disorientation, decreased mood, sleep disturbance, moderate pain in the lumbar spine during movements.

Anamnesis of the disease: In November 2021, the patient noted the appearance of edema of the left leg, and therefore, on November 4, the ambulance team was hospitalized in the city hospital, where deep vein thrombosis of the left leg was detected. According to CT scan, massive pulmonary embolism (PE) was diagnosed. During the examination, a positive PCR test for SARS-CoV-2 dated 11/08/2021 was detected, in connection with which the patient was transferred to the ICU. Anticoagulant, corticosteroid, antibiotic therapy was prescribed. COVID-19 was complicated by the development of a hyperinflammatory syndrome (cytokine storm with an increase in CRP up to 496 mg/dL, ferritin over 2000 µg/L), progressive lung damage (CO-RADS 3), and severe respiratory failure. Biological therapy with tofacitinib was carried out. Against the background of the therapy, fever persisted up to 38°C, the level of CRP was 93 mg/l (01/02/2022), ferritin - 1295 µg/l (12/31/2021), procalcitonin from 12/28/2021 - negative.

For further treatment, he was transferred to a specialized infectious diseases hospital. According to CT scan with intravenous contrast dated 01/02/2022 - PE of segmental branches of the lower lobar arteries, the presence of blood clots in the lumen of the thoracic aortic arch. CT features of bilateral polysegmental pneumonia, CO-RADS 3, bilateral small pleural effusion. On admission, the level of CRP was 138 mg/l. Anticoagulant therapy was carried out (rivaroxaban 15 mg x 2 times a day from 11/01/2022), biological therapy (Levilimab 324 mg subcutaneously on 01/02/2022). During therapy, the temperature returned to normal, the signs of a cytokine storm decreased (CRP from 01/13/2022 - 3.8 mg/l). At the control CT scan dated 01/13/2022, there is a positive trend (CO-RADS 2). Discharged from the hospital on 01/14/2022 for outpatient treatment.

From 01/15/2022 to 01/31/2022 he was at home. All this time, normothermia persisted, however, the patient noted the progression of visual impairment, the increase in weakness, the appearance of episodes of disorientation in time, the appearance of an unproductive cough.

For examination and treatment, he was hospitalized in the therapeutic department of City Clinical Hospital Named by S. P. Botkin.

Anamnesis of life: Heredity is not burdened. Chronic diseases previously denied. Until November 2021, he did not take drugs. In blood tests from 2018, for the first time, pronounced asymptomatic changes were detected: leukocytes $18 \times 10^9/L$, platelets $552 \times 10^9/L$, hemoglobin level 184 g/L. Due to the identified changes, the patient was not examined. Past operations: removal of the spleen in 1990 due to trauma. Laparotomy for adhesive intestinal obstruction. Current medications: rivaroxaban 15 mg x 2 times a day. Bad habits: Smoked for over 30 years, 3 packs a day.

Status at admission: Height: 175 cm, weight: 68 kg; BMI: 22.2 kg/m². Temperature: 36.6°C; General condition of moderate severity. Consciousness is clear. Skin color: pale. Respiratory health: frequency of respiratory movements: 18/min; Breathing rhythm: regular. Respiration: normal. Breathing is carried out in all departments. slightly weakened in the lower sections. No wheezing.

The state of the cardiovascular system.

Hemodynamics: stable. BP 100/60 mm Hg, Heart rate: 87 /min; moderate systolic murmur at the apex.

Condition of the gastrointestinal tract, genitourinary system: no deviations.

The state of the organ of vision.

Pupils: wide. Anisocoria: no. Symmetry of the pupils: symmetrical. Meningeal syndrome: negative. Sclera: injected.

Ophthalmic status: Visual acuity: OD - hand movement is eccentric. OS - uncertain light projection. Both eyes: does not determine the field of view. The fundus of the eye - the optic disc is pale pink, the boundaries are clear, the vascular pattern is not changed. IOP 20 \20mm Hg.

Instrumental research

According to the ECG, Holter-ECG without significant deviations.

Echocardiography: Conclusion: Bacterial endocarditis of the mitral and aortic valves. Significant (acute) mitral and moderate (acute) aortic regurgitation. Systolic LV function is moderately reduced (EF 45%).

Ultrasound examination of the veins of the lower extremities. Conclusion: Deep vein thrombosis of the left lower limb, in the stage of partial recanalization. Thrombosis of the SSV of the right lower extremity.

Computed tomography of the chest. Conclusion: Against the background of a minimally expressed right-sided hydrothorax, signs of the presence of discoid atelectasis of both lungs (post-inflammatory changes) are unevenly expressed. More data on the CT picture of the formation of a honeycomb lung and the initial signs of manifestations of bullous transformation of the parenchyma of both lungs.

Fibrous adhesive changes on both sides. Signs of pulmonary hypertension. Atherosclerosis of the aorta/its branches/heart valves. Atherosclerosis of the coronary vessels. Lymphadenopathy of the intrathoracic lymph nodes. Ankylosis of Th4/5 bodies against the background of their anterior wedge-shaped deformity.

Magnetic resonance imaging of the brain, arteriography of intracranial arteries and venography of intracranial veins and sinuses with contrast.

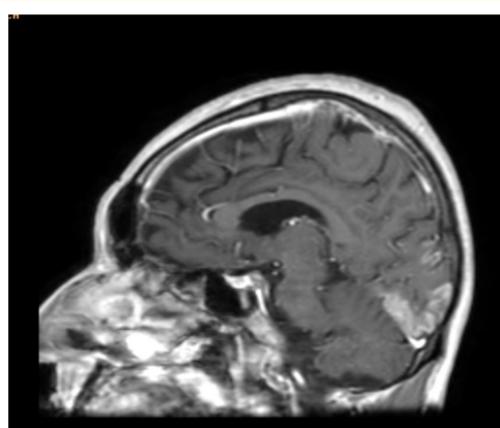


Figure 1: Magnetic resonance imaging of the brain of patient M.

MR signs of ischemic circulatory disorders in the left occipital region, with the formation of cysts in the parietal region of both hemispheres of the brain, acute ischemia in the right occipital region.

MRI of the brain, arteriography of intracranial arteries and venography of intracranial veins and sinuses. Conclusion: signs of saccular aneurysm of the left middle cerebral artery, lack of blood flow (thrombosis?) at the level of the right transverse sinus.

CT angiography of intracranial vessels (arteries and veins) with contrast. Conclusion: Saccular aneurysm of the left middle cerebral artery. Occlusion of the left vertebral artery at the C2 level. Thrombosis of the right internal jugular vein. Atherosclerosis of the brachiocephalic arteries without signs of stenosis.

CT angiography of the thoracic aorta and its branches with contrast. Conclusion: CT signs of multiple focal lesions in the liver, probably a combination of hemangiomas, cysts, and possibly secondary foci. Foci of destruction in the L3 vertebral body and in the pelvic bones (metastases). Changes in the kidneys differentiate between the consequences of kidney infarction and inflammatory changes. Hepatomegaly.

The course of the disease and the treatment

The patient was admitted to the therapy department with increasing asthenia, cachexia, persisting laboratory signs of inflammation (leukocytosis, a significant increase in IL-6 levels, an increase in the level of C3 complement) with persistent normothermia, as well as multiorgan lesions (lesion of the ENT organs, lungs, liver, cardiovascular, nervous system in the form of thrombosis).

Considering the signs of ischemic stroke in the left occipital region revealed by MRI data, the patient was examined by an ophthalmologist, and the neurologist corrected the therapy. Examined by an otorhinolaryngologist: To exclude systemic vasculitis - granulomatosis with polyangiitis (Wegener's disease), a blood test for ANCA antibodies was performed. Data for systemic vasculitis were not obtained.

Given the anamnestic indications of an increase in hemoglobin, leukocytes and platelets in 2018, chronic myeloproliferative disease was excluded. According to the results of a blood test for a genetic mutation, as well as according to the results of a trepanobiopsy, data for a myeloproliferative process were not obtained.

Attention was drawn to the rapid negative ultrasound dynamics of changes in the liver in the form of an increase in the number of hypoechoic and anechoic formations. According to CT scans, multiple foci were revealed in the liver, some of which accumulate a contrast agent in the periphery. When blood cultures for sterility, the growth of microflora was not obtained.

Due to the fact that a significant increase in the level of oncomarkers (CA19-9, CA-125, Carcinoembryonic antigen (CEA)) was detected, and the appearance of new multiple formations in the liver, the patient was discussed by an oncologist - an additional examination was recommended according to the cancer search program: gastroscopy, colonoscopy.

Total colonoscopy revealed no significant pathology. According to gastroscopy - erosive antral gastritis, according to bronchoscopy - deformation and narrowing of the lumen of the lobar and segmental bronchi on the right.

At the control ultrasound of the pleural cavity, an increase in the volume of effusion from 250 to 900 ml is noted, as well as the appearance of an air strip in the upper sections. Performed thoracentesis on the right, evacuated 800 ml of serous contents. Cytological examination of the pleural effusion revealed malignant cells with a cytological picture of glandular cancer.

For morphological verification of the diagnosis and determination of therapeutic tactics, a puncture biopsy of the liver under ultrasound guidance was performed, which was complicated by intra-abdominal bleeding, in connection with which the patient was trans-

ferred to the ICU. Anticoagulant therapy was stopped. Echocardiography revealed signs of acute infective endocarditis of the aortic and mitral valve with the formation of acute mitral insufficiency. Conducted antibacterial therapy (cefepime + sulbactam), against which noted the normalization of body temperature. Due to the rapid accumulation of exudate in the right pleural cavity, drainage of the right pleural cavity was performed.

The next follow-up ultrasound of the veins revealed occlusive iliofemoral thrombosis of the left lower limb. Given the floating nature of phlebothrombosis, the impossibility of adequate anticoagulant therapy, the negative dynamics of ultrasound, the threat of repeated pulmonary embolism, the patient, according to vital indications, needs to have cavagraphy with thromboextraction, installation of a cava filter in the IVC.

Then, a thromboextraction operation was performed, and a cava filter was implanted. In the early postoperative period, the phenomena of cardiovascular and respiratory failure, resistant to increasing doses of vasopressors, rapidly increased. Recorded cardiac arrest.

Resuscitation measures in full within 30 minutes without effect. The biological death of the patient was stated.

So, during the examination of the patient, the diagnosis was established:

Diagnosis at admission:

- The main diagnosis: I63.5 Cerebral infarction, in the vertebrobasilar arterial system, early recovery period. An unspecified pathogenetic variant according to TOAST, probably against the background of a COVID-19. Aneurysm of the left MCA.
- Competing diagnosis: J12.9 Bilateral viral polysegmental pneumonia caused by SARS-CoV-2, in the process of resolution. COPD. Chronic bronchitis of a smoker, exacerbation. Pneumosclerosis. Respiratory failure grade 1.
- Complications: M31.1 Postcovid syndrome with hyperinflammatory reaction. Thrombotic microangiopathy.
- Concomitant diseases: G93.4 Dyscirculatory encephalopathy. Asthenic syndrome. Dysomnia. Thrombosis of deep and superficial veins of the left lower limb. PE. Chronic gastritis. Chronic pancreatitis, without exacerbation.

Postmortem diagnosis

- The main diagnosis: I63.5 Cerebral infarction, in the vertebrobasilar arterial system, early recovery period. Unspecified pathogenetic variant according to TOAST, against the background of a COVID-19.
- Competing diagnosis: D47.1 Adenocarcinoma with unspecified primary lesion. Metastatic lesions of the liver, pleura and bones (L3, right iliac spine). Right-sided hydrothorax. Drainage of the right pleural cavity dated February 22, 2022. Liver biopsy dated February 18, 2022.
- Complications of the underlying disease: I80.2 Acute infective endocarditis involving mitral and aortic valves. Acute mitral regurgitation 3 stage. Moderate secondary pulmonary hypertension. Hyperinflammatory syndrome. Subcapsular hematoma of the liver, small hemoperitoneum. Disseminated intravascular coagulation. Thrombosis of the inferior vena cava with flotation. Operation: open thromboextraction from the IVC with implantation of Cava Filter "OPTEASE" dated February 26, 2022. Acute cardiovascular failure. Condition after cardiopulmonary resuscitation dated February 26, 2022.
- Concomitant diseases: G93.4 Thrombosis of deep and superficial veins of the left lower limb. PE from November 2021. Dyscirculatory encephalopathy. Aneurysm of the left middle cerebral artery. Chronic gastroduodenitis. Erosive gastritis COPD. Chronic bronchitis of a smoker, exacerbation. Pneumosclerosis. Respiratory failure grade 1.

Conclusion

This clinical observation is interesting in that severe COVID-19, complicated by a cytokine storm, further contributed to the development of systemic vasculitis, multiple thromboses, cerebral infarction with damage to the visual centers of the occipital lobes of the brain. Pathological systemic disorders led to death, despite therapeutic measures.

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