ISSN 2307-8960 (online)

World Journal of *Clinical Cases*

World J Clin Cases 2024 April 6; 12(10): 1714-1856

Published by Baishideng Publishing Group Inc

World Journal of Clinical Cases

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Thrice Monthly Volume 12 Number 10 April 6, 2024

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ABOUT COVER

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RESPONSIBLE EDITORS FOR THIS ISSUE

NAME OF JOURNAL

World Journal of Clinical Cases

ISSN

ISSN 2307-8960 (online)

LAUNCH DATE

April 16, 2013

FREQUENCY

Thrice Monthly

PUBLICATION DATE

April 6, 2024

World Journal of Clinical Cases

Submit a Manuscript: https://www.f6publishing.com

World J Clin Cases 2024 April 6; 12(10): 1799-1803

DOI: 10.12998/wjcc.v12.i10.1799

ISSN 2307-8960 (online)

CASE REPORT

Subdural effusion associated with COVID-19 encephalopathy: A case report

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Specialty type: Medicine, research and experimental

Provenance and peer review: Unsolicited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's scientific quality classification

Grade A (Excellent): 0
Grade B (Very good): 0
Grade C (Good): 0
Grade D (Fair): D, D
Grade E (Poor): 0

P-Reviewer: Kawahara H, Japan

Received: November 16, 2023
Peer-review started: November 16, 2023
First decision: December 26, 2023
Revised: January 7, 2024
Accepted: March 8, 2024
Article in press: March 8, 2024
Published online: April 6, 2024

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Abstract

BACKGROUND

The precise mechanism by which severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) impacts the central nervous system remains unclear, with manifestations spanning from mild symptoms (*e.g.*, olfactory and gustatory deficits, hallucinations, and headache) to severe complications (*e.g.*, stroke, seizures, encephalitis, and neurally demyelinating lesions). The occurrence of single-pass subdural effusion, as described below, is extremely rare.

CASE SUMMARY

A 56-year-old male patient presented with left-sided limb weakness and slurred speech as predominant clinical symptoms. Through comprehensive imaging and diagnostic assessments, he was diagnosed with cerebral infarction complicated by hemorrhagic transformation affecting the right frontal, temporal, and parietal regions. In addition, an intracranial infection with SARS-CoV-2 was identified during the rehabilitation process; consequently, an idiopathic subdural effusion developed. Remarkably, the subdural effusion underwent absorption within 6 d, with no recurrence observed during the 3-month follow-up.

CONCLUSION

Subdural effusion is a potentially rare intracranial complication associated with SARS-CoV-2 infection.

Key Words: Cerebral infarction; Hemorrhagic transformation; Subdural effusion; COVID-19 encephalopathy; Novel coronavirus infection; Brain fog; Case report

Core Tip: Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) may transmit *via* the retrograde axonal pathway, bloodstream, or direct penetration through the blood-brain barrier, exerting its effects on angiotensin-converting enzyme-2 receptors. This intricate interaction can cause neurological complications, including subdural effusion, which is very rare. Clinical vigilance is advised for cranial imaging in individuals with SARS-CoV-2 infection to enhance diagnostic precision. Considering its unique characteristics, subdural effusion, a seldom reported complication, warrants attention.

Citation: Xue ZY, Xiao ZL, Cheng M, Xiang T, Wu XL, Ai QL, Wu YL, Yang T. Subdural effusion associated with COVID-19 encephalopathy: A case report. *World J Clin Cases* 2024; 12(10): 1799-1803
URL: https://www.wjgnet.com/2307-8960/full/v12/i10/1799.htm
DOI: https://dx.doi.org/10.12998/wjcc.v12.i10.1799

INTRODUCTION

Subdural effusion or subdural hydrocele commonly stems from craniocerebral trauma, postcranial surgery, or cerebral atrophy. Typically presenting unilaterally or bilaterally in the frontotemporal region, symptoms include headache, dizziness, cognitive alterations, and mood changes. Its slow absorption may progress to chronic subdural hematoma, with treatment options ranging from medications to surgical interventions such as bone flap craniotomy, subdural effusion capsule wall stripping, abdominal shunt, and external drainage[1-4]. This report presents a unique case of subdural effusion that formed after severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection but was remarkably resolved within 6 d, showcasing a rare and expeditious resolution.

CASE PRESENTATION

Chief complaints

A 56-year-old male presented with a sudden onset of left-sided limb weakness for over 20 d.

History of present illness

On November 25, 2021, the patient experienced a sudden onset of left-sided limb weakness accompanied by slurred speech. After a comprehensive examination, he was diagnosed with cerebral infarction with hemorrhagic transformation affecting the right frontal, temporal, and parietal lobes. He was then treated for his cranial hemorrhage symptoms. Throughout the hospitalization period, he exhibited fever, dyspnea, and generalized muscular aches and pains. The patient's temperature fluctuated at 38.0 °C, accompanied by headache, hallucinations, and incoherent speech.

History of past illness

He had primary hypertension for 4 years, with a maximum blood pressure of 188/106 mmHg. The patient was on long-term oral medication with sacubitril/valsartan sodium tablets (100 mg) once a day.

Personal and family history

The patient denied any personal or family history of related diseases.

Physical examination

The patient was unresponsive, a shallow left nasolabial groove, slurred speech, difficulty swallowing fluids (leading to choking), slight neck resistance, a positive Kernig's sign, and muscle strength graded at 0, 2, and 5 in the left-upper, left-lower, and right limbs, respectively, were observed. Reduced muscle tone in the left upper limb, decreased pharyngeal reflexes, and a positive Babinski's sign on the left side were also observed. The remaining systematic examination did not reveal any positive signs.

Laboratory examinations

The patient's laboratory examination results were as follows: Platelet count, 462 × 10^9/L (reference range: 100-300/L); sodium level, 125 mmol/L (reference range: 135-145 mmol/L); positive throat swab test result for coronavirus disease 2019 (COVID-19); C-reactive protein level, 44.18 mg/L (reference range: 0-10 mg/L); and blood gas analysis, 60 mmHg (reference range: 83-108 mmHg). Furthermore, inflammatory factors were elevated, with interleukin (IL)-2, IL-6, IL-10, and tumor necrosis factor alpha (TNF-α) at 878.54, 62.83, 136.15, and 111.14 U/mL (reference range: 160-625, 0-7, 0-9.5, and 0-8.5 U/mL), respectively. Lumbar puncture revealed a cerebrospinal fluid pressure of 150 mmH_2O (reference range: 80-180 mmH_2O). The cerebrospinal fluid analysis for SARS-CoV-2 showed specific gene Ct values: N gene Ct value of

35.5429 and ORF1ab gene Ct value of 30.3402 (reference range: > 40 for both). Routine biochemistry of cerebrospinal fluid revealed no abnormalities. Cultures and a full set of viral screenings did not detect pathogens such as bacteria, fungi, parasites, *Mycobacterium tuberculosis* complex, Mycoplasma, or Chlamydia. Moreover, cerebrospinal fluid and serum specimens were negative for autoimmune encephalitis antibodies, paraneoplastic syndrome autoantibody profile semiquantitative test, and ganglioside antibody profile. Coagulation function and procalcitonin levels also showed no abnormalities. Electroencephalogram results showed bilateral symmetrical diffuse slow waves.

Imaging examinations

Cranial magnetic resonance imaging (MRI) revealed a typical infarction in the right frontal, temporal, and parietal lobes, along with brain atrophy indications. On magnetic resonance angiography, the right internal carotid artery and middle cerebral artery were largely undetected, and the walls of the left internal carotid artery, left middle cerebral artery M1 segment, and bilateral posterior cerebral arteries exhibited abnormalities (Figure 1A and B). On December 28, 2021, a repeat cranial computed tomography (CT) scan revealed infarction with hemorrhagic transformation in the right frontal, temporal, parietal, and basal ganglia areas, and chest CT showed scattered patchy shadows in the lungs (Figure 1C). On March 15, 2022, cranial MRI revealed new subdural effusion in the right frontal, temporal, and parietal areas (Figure 1D), with subsequent absorption of the subdural effusions on March 21 (Figure 1E).

FINAL DIAGNOSIS

The patient was diagnosed with the following: (1) Cerebral infarction with hemorrhagic transformation on the right frontal, temporal, and parietal lobes (atherosclerotic large artery type); (2) Cerebral atrophy; (3) Primary hypertension, grade 3 (extremely high-risk group); (4) Pneumonia associated with COVID-19; (5) COVID-19 encephalitis; (6) Subdural effusion; and (7) Hyponatremia.

TREATMENT

The patient received the following: Aspirin enteric-coated tablet (100 mg) orally once a day, rosuvastatin calcium tablet (10 mg) orally at night, butylphthalide soft capsule (0.2 g) orally thrice a day, sacubitril/valsartan sodium tablet (100 mg) orally once a day, and early comprehensive rehabilitation training. He was also prescribed nemavirit (300 mg)/ritonavir (100 mg) tablet orally every 12 h for 5 d, methylprednisolone sodium succinate injection (40 mg) intravenously once a day, and acetylglutamide injection (0.6 g) intravenously once a day.

OUTCOME AND FOLLOW-UP

At 3 months after discharge, we noted that the patient's left-sided limb weakness had improved, and he had clear speech and mild headache that did not disrupt his sleep or daily activities (Figure 1F). The patient's condition has remained stable without any noteworthy concerns.

DISCUSSION

Subdural effusions encompass regressive, stable, progressive, and evolving types, with craniocerebral trauma being the predominant cause. The stable and evolving types are more common among older adults, often progressing slowly and occasionally evolving into chronic subdural hematomas necessitating surgical intervention when conservative approaches fail. Conversely, regressive types are more prevalent in young individuals, showcasing optimal healing outcomes, and progressive types, which are found more commonly in pediatrics, exhibit severe symptoms, neurological deficits, and the highest mortality rate[5]. In the case of our elderly male patient, the subdural effusion spontaneously resolved in only 6 d, aligning with the typical characteristics of regressive subdural effusion.

We analyzed the potential causes of subdural effusion. First, SARS-CoV-2 has a neuroinvasive and neurophilic nature, as seen in the associations of subdural effusion with other viruses, such as enterovirus 71, herpes simplex virus type 1, and Epstein-Barr virus[6]. However, currently, only one case of subdural effusion associated with SARS-CoV-2 has been reported[7]. Involvement in the central nervous system (CNS) by the virus primarily occurs through direct invasion, inflammatory response activation, and autoimmune response induction. Excessive immune activation leads to the intracranial infiltration of inflammatory cells and upregulation of proinflammatory factors such as IL-1α, IL-6, and TNF-α, resulting in a "cytokine inflammatory storm". The virus enters the CNS through the interstitial space between endothelial cells and affects the angiotensin-converting enzyme-2 receptor, causing brain tissue damage and cerebrospinal fluid extravasation[8-12]. Second, blood-brain barrier permeability may be altered. The complex mechanism of cerebral infarction with hemorrhagic transformation involves the release of oxygen-free radicals, inflammatory factors, and cytokines and the degradation of collagen and laminin by brain cell extracellular matrix metalloproteins. This event affects the structural integrity of endothelial cells, basement membranes, and the perivascular pedicle of astrocytes. The

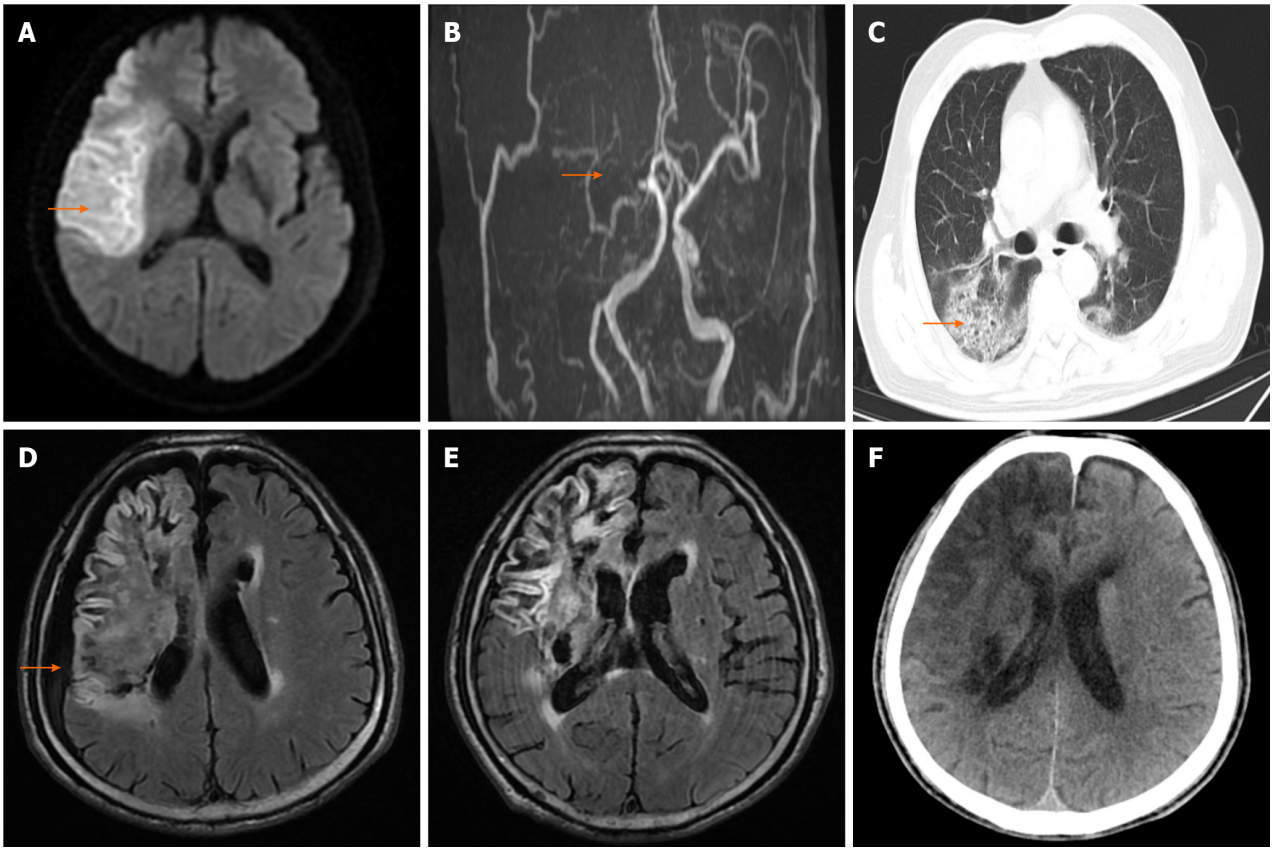

Figure 1 Cranial magnetic resonance imaging. A: Cranial magnetic resonance imaging (MRI) revealed distinct, patchy, nodular long T2 signal shadows in the right frontal, temporal, and parietal lobes; B: Head and neck MRI displayed non-visualization of the right internal carotid and middle cerebral arteries, particularly noting superficial shadows in the right internal carotid siphon, the middle cerebral artery M1 segment, and the right anterior cerebral artery A1 segment (indicated by arrow); C: Chest computed tomography showed scattered patchy shadows in the lungs; D: Cranial MRI displayed new subdural effusion in the right frontal, temporal, and parietal regions; E: Cranial MRI displayed marked absorption of subdural effusion in the right frontal, temporal, and parietal regions at 6 d after its appearance; F: Head computed tomography displayed the scar of cerebral infarction with hemorrhagic transformation in the right frontal, temporal, and parietal lobes at 3 months after discharge.

patient's right cerebral hemispheric parenchyma displayed edema, and MRI revealed fragmented protein deposition in the lesion, further implying blood-brain barrier disruption. Third, brain atrophy could be a potential cause. Cranial CT and MRI revealed ventricular enlargement, cerebral pool enlargement, cerebral sulcus and fissure deepening, and brain tissue volume reduction. Brain atrophy coupled with compensatory dilation of the subdural space was considered to be a factor leading to subdural effusion.

In summary, the mechanisms by which SARS-CoV-2 affects the CNS are insufficiently understood. This virus may use various routes of transmission, ranging from retrograde axonal pathways to bloodstream transmission and direct transmission through the blood-brain barrier. Clinical manifestations may range from mild symptoms (*e.g.*, olfactory and taste deficits) to severe ones (*e.g.*, stroke, seizures, encephalitis, and neurally demyelinating lesions)[13-15]. Subdural effusion has been proposed as a potentially rare feature of SARS-CoV-2 infection. Hence, early evaluation of brain imaging in infected patients is crucial to promptly detect neurological involvement, with timely screening and intervention playing a critical role in reducing subsequent morbidity and mortality.

CONCLUSION

Subdural effusion is a potentially rare intracranial complication associated with SARS-CoV-2 infection.

ACKNOWLEDGEMENTS

We thank Dr. Ming Cheng for his editing assistance.

FOOTNOTES

Author contributions: All authors made a significant contribution to the work reported, whether that was in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; all authors took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Informed consent statement: The study participant provided written informed consent before enrollment.

Conflict-of-interest statement: All the authors report no relevant conflicts of interest for this article.

CARE Checklist (2016) statement: The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016).

Country/Territory of origin: China

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S-Editor: Wang JJ
L-Editor: Webster JR
P-Editor: Zhao S

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