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Malignant Evolution of Sphenoid Sinusitis

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ABSTRACT

We learnt from the diagnosis and treatment process of a adult male patient who successively developed severe headache, diplopia, ocular edema, cerebral infarction, epistaxis shock and Balt balloon occlusion that sphenoid sinusitis had fatal harm, because the internal carotid artery stenosis caused by vasculitis later resulted in cerebral infarction, and pseudoaneurysm rupture caused by vasculitis almost led to death of the patient if there was no prompt rescue and interventional surgery, Sphenoid sinusitis is a type of sinusitis that is a common disease. We're used to common diseases, so we often ignore them. This case had severe conditions and rapid progress and was given effective anti-infection and decisive endovascular intervention surgery. We carried on profound reflection on its diagnosis and treatment process, and we need to attach great importance to the role of sinusitis, especially sphenoid sinusitis, in the early stage of headache.

Keywords: Sphenoid Sinusitis; Cavernous Sinus Syndrome; Meningitis; Cerebral Infarction; Inflammatory Aneurysm; Epistaxis; Balt Balloon Occlusion

Summary of Admission Medical Record

A adult male patient suddenly developed persistent severe stuffy and distending pain in the left temporal region and retrobulbar region without predisposing causes, which was obvious at night and affected rest, without nausea, vomiting and fever. One week after the course of disease, the patient received a physical examination in a local hospital. The body temperature was normal. The Erythrocyte Sedimentation Rate (ESR) was up to 123 mm/h, and the hypersensitive C-Reactive Protein (hCRP) level was up to 90.35 μg/L. Brain CT showed left sphenoid sinusitis. The results of brain MRI, MRA and MRV showed "no obvious abnormalities". Nearly one week after the symptomatic treatment with dexamethasone, mannitol and analgesics, the condition became worse, and the right temporal region and retrobulbar region also developed stuffy pain, with diplopia and anorexia. Then he was transferred to our hospital due to superficial temporal arteritis. He had no history of similar headache, sinusitis, hypertension, trauma or drug allergy. The physical examination on admission showed body temperature of 36.3°C, painful expression, limited outreach of the left eye, and no other signs.

Disease Evolution and Diagnosis and Treatment Process After Admission

The blood routine examination on the day of admission (14th day of disease course) showed white blood cell (WBC) of 10.73×109/L, neutrophil granulocyte (N) of 9.57×109/L, neutrophil percentage (N%) of 89.2%, ESR of 140/h, and hCRP of 33.43/L. Brain CT showed left sphenoid sinusitis (Figures 1a & 1b), and no obvious changes in cavernous sinus (Figure 2a). Glaucoma was excluded by ophthalmologic consultation. The results of subsequent head and neck CTA (Figure 3a) and color Doppler ultrasound of the superficial temporal artery were normal; lumbar puncture pressure was 140 mmH2O, and the results of Cerebrospinal Fluid (CSF) routine, biochemical, microbiological and cytological tests were normal; brain MRI+ enhanced scan + MRV showed "a little inflammation of sinus", i.e. left sphenoid sinusitis (Figure 4a), and orbital MRI showed slight thickening of bilateral superior ophthalmic veins (Figure 5a). High-flow oxygen inhalation, tramadol, oxycodone and acetaminophen (oral) and mannitol (intravenous injection) were successively given for the treatment of superficial temporal arteritis and cluster headache, but the analgesic

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effect was not obvious, and methylprednisolone (intravenous drip) could temporarily relieve the pain. On the 19th day of the course, the headache remained severe, and morphine was even used for analgesia, but there was still no fever. Ptosis of the left eye, eyeball fixation, pupil diameter dilation to 5 mm, loss of light reflex, chemosis, left frontal hypohidrosis and right chemosis, limitation of eye movement, pupil diameter of 2 mm, and dullness of light reflex occurred successively.

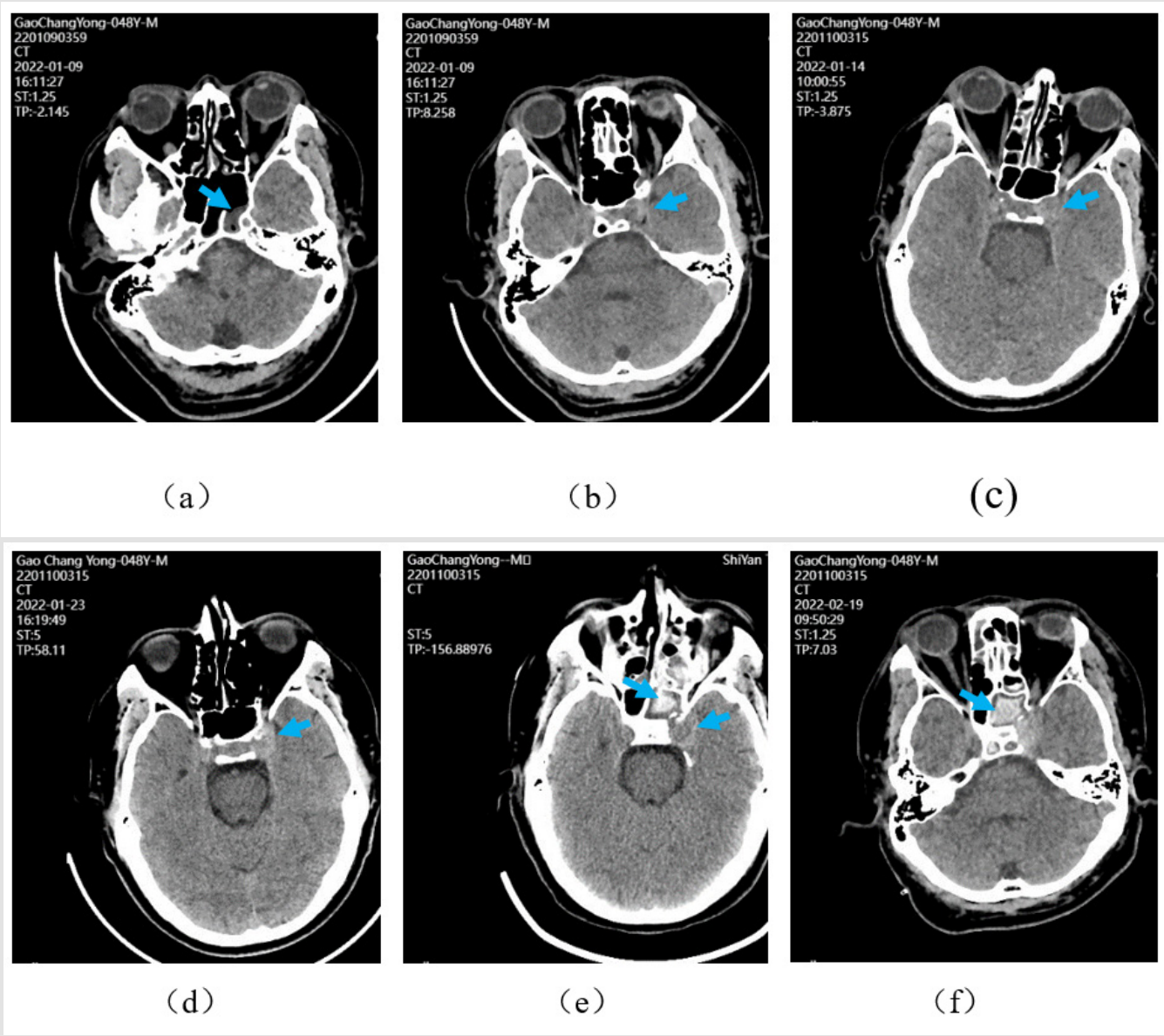

Figure 1: CT of sphenoid sinus and sious cavernosus.

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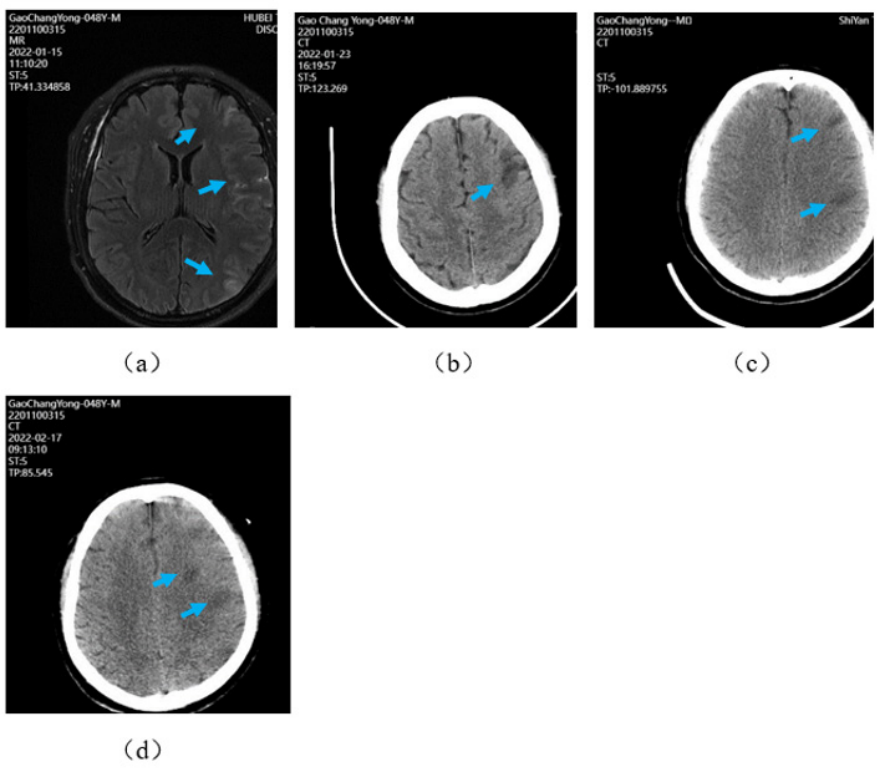

Figure 2: CT and MRI of brain.

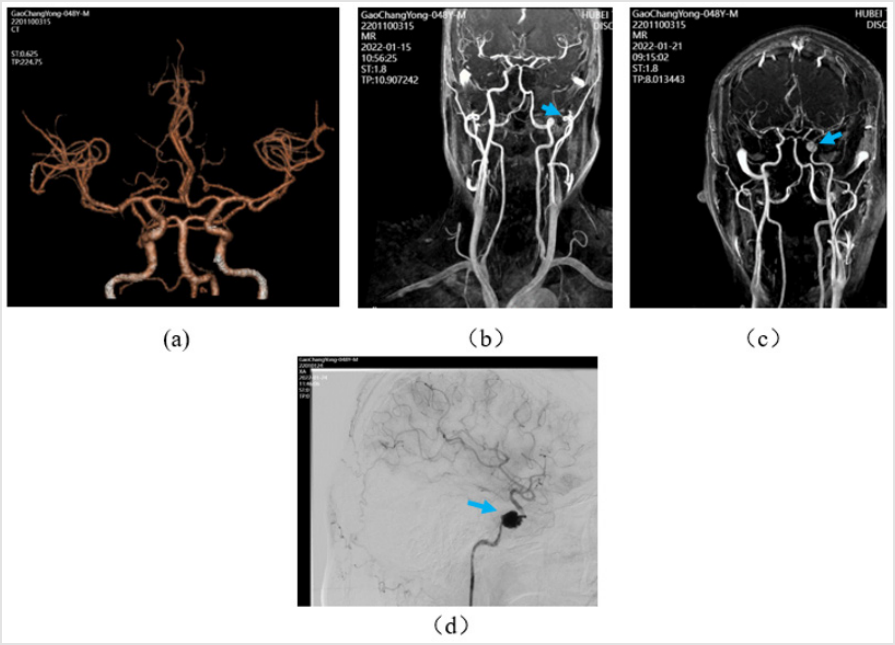

Figure 3: CTA, MRA and DSA of blood vessel of brain.

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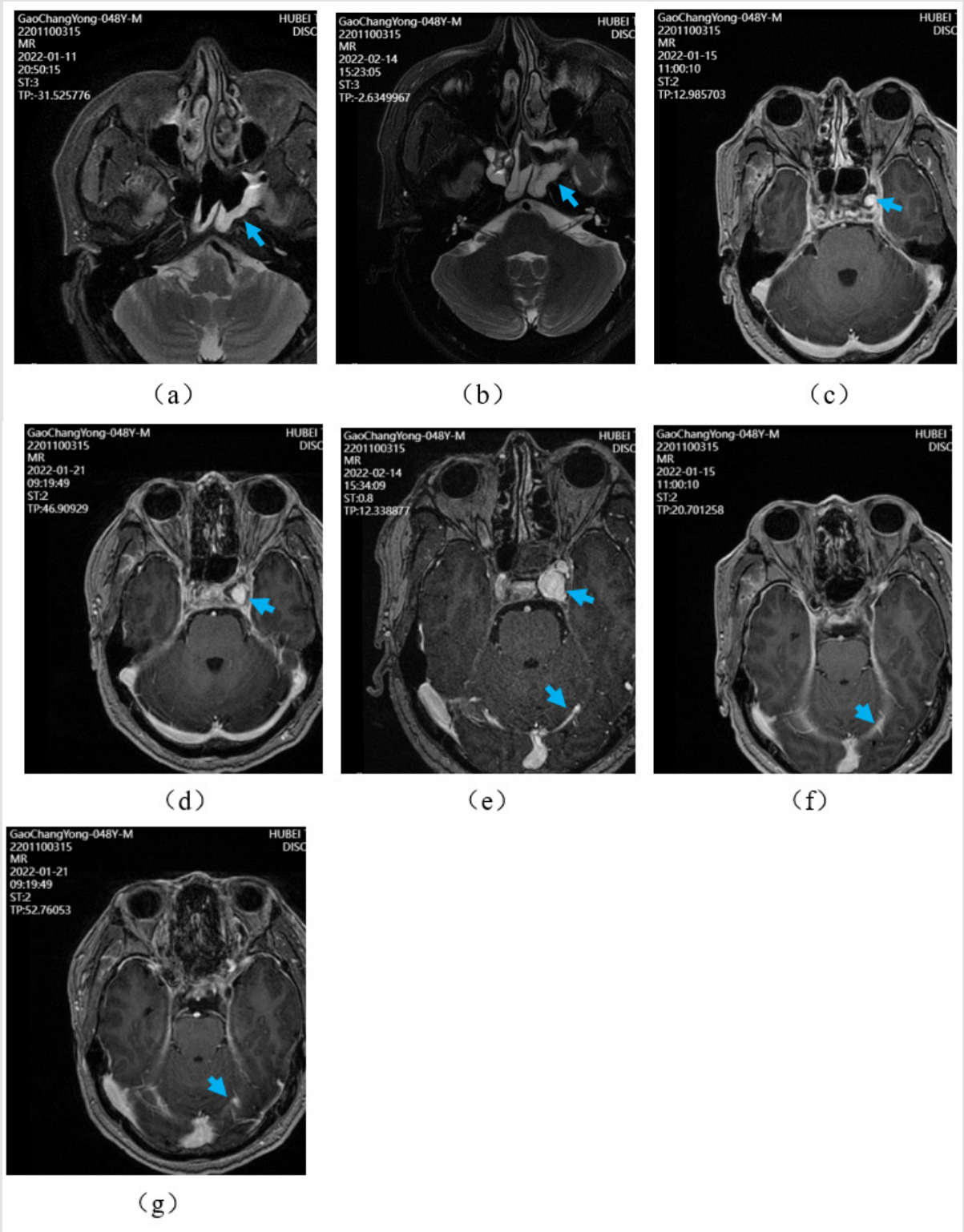

Figure 4: MRI of sphenoid sinus, sious cavernosus and endocranium.

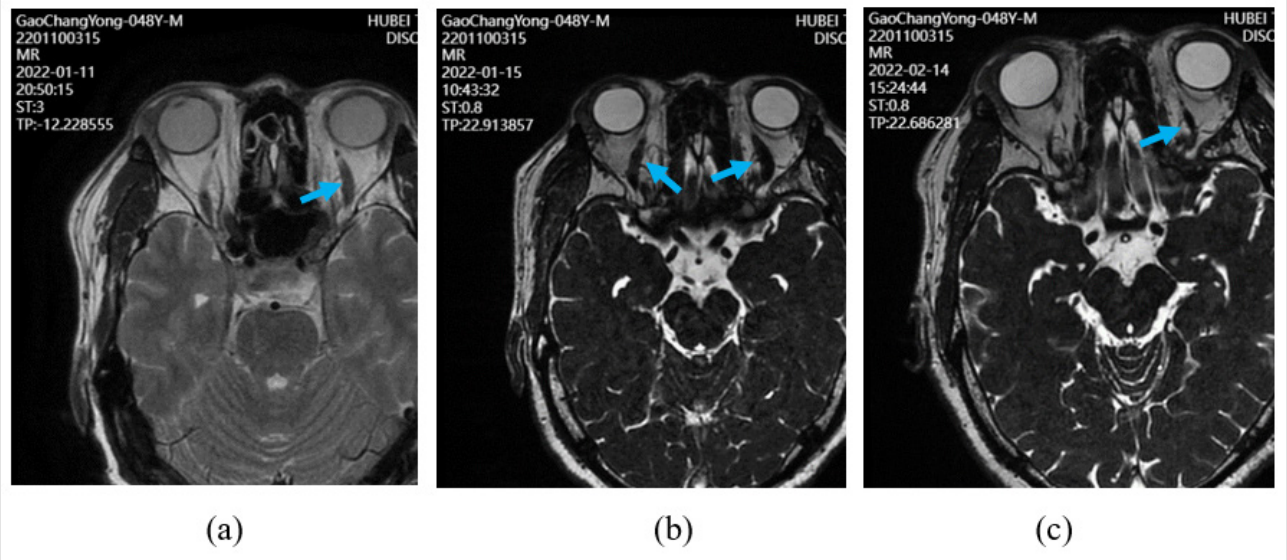

Figure 5: MRI of superior ophthalmic vein and jugular vein.

Brain CT showed left sphenoid sinusitis and slightly increased density of the left cavernous sinus (Figure 1c), which was suspected of cavernous sinusitis and bacterial infection. Ceftriaxone was given for anti-infection. The next day, the headache began to abate, mainly in the left temporal region and the retrobulbar region, without fever and limb numbness and paralysis. The blood routine reexamination showed WBC of 15.77×109/L, N of 14.54×109/L, N% of 92.2%, and ESR of 55 mm/h; skull base MRI plain scan + enhanced scan showed acute cerebral infarction in the left frontal and occipital lobe (Figure 2a), left sphenoid sinusitis, aneurysm or inflammatory nodule (diameter: 0.84 cm) in the cavernous sinus segment of left internal carotid artery (Figure 4c), and extensive inflammatory dural lesions around bilateral cavernous sinus, temporal region and tentorium (Figure 4f); brain MRA revealed severe stenosis or occlusion of left internal carotid artery siphon (Figure 3b), thickening of bilateral superior ophthalmic vein especially on the left side (Figure 5b);the lumbar puncture pressure was 80 mmH2O, CSF was colorless and slightly turbid, and the result of Pandy's test was positive; total cell count and WBC: 858.57×106/L, polymorphonuclear leukocyte percentage: 90%, protein: 0.65 g/L, glucose: 4.68 mmol/L (blood glucose: 11.9 mmol/L), and normal results of acid-fast bacilli, ink and Alcian staining; no microbial gene sequences were found in second-generation sequencing of serum and CSF DNA and RNA; E.N.T. consultation found no fungus in the depth of the nasal cavity.

Ceftriaxone was changed to meropenem to intensify anti-infection, and anticoagulation and anti-platelet aggregation therapies were added. On the 26th day of the course, headache was continuously relieved without fever, the right eye returned to normal, facial skin sensation was normal, and the conditions of ptosis of the left eye, exophthalmos, eyeball fixation, mydriasis, loss of light reflex and chemosis were the same as before. Blood routine reexamination: WBC: 5.9×109/L, N: 4.2×109/L, N%: 71.2%, ESR: 52 mm/h, hCRP: 16.73 μg/L; the third time of lumbar puncture pressure: 135 mmH2O, he result of Pandy's test was positive, and the results of CSF routine, biochemical, acid-fast bacilli, ink and Alcian staining were normal. Brain enhanced MRI + MRA + MRV reexamination showed that the small circular enhanced shadow of the cavernous sinus segment of left internal carotid artery was slightly larger than before (1.0×1.3 cm), and tumor body shadow was visible (Figure 4d) and (Figure 3c); The extensive dural thickening and enhancement of bilateral cavernous sinus, temporal region and tentorium were improved than before (Figure 4g). Meropenem was changed to ceftriaxone for subsequent treatment due to the improvement of the condition, especially the rapid recovery of CSF. On the 29th day of the course, the condition was stable without fever, limb numbness and paralysis.

Brain CT reexamination showed left sphenoid sinusitis, high-density shadow of left cavernous sinus (Figure 1d) and cerebral infarction in the left frontal and occipital lobe (Figure 2b); cerebral angiography showed an aneurysm (14.76×11.27 mm) in the cavernous sinus segment of the left internal carotid artery with 70% stenosis of the C5 segment of the parent artery (Figure 3d). The times of subcutaneous injection of low molecular weight heparin were reduced. Anti-infection, anticoagulation and anti-platelet aggregation therapies were continued in neurosurgery consultation, and the patient was transferred to another department for surgery after the condition became stable. In the early morning of the 50th day of the course, stuffy and

throbbing pain in the left temporal and retrobulbar regions disappeared, but the patient suddenly suffered from left nasal hemorrhage of about 100 mL. E.N.T. consultation was determined to give cotton ball packing and dicynone injection to stop bleeding, and the coagulation function was normal. After neurosurgery consultation, the patient was transferred to another department, and the results of CSF pressure, lumbar puncture routine and biochemical tests were normal. WBC: 3.36×109/L, N: 2.12×109/L, N%: 63%, RBC: 3.2×1012/L, Hb: 101 g/L; skull base MRI enhancement showed that the local small circular enhanced shadow of the left cavernous sinus was enlarged (1.9×1.6 cm) (Figure 4e), and the dural thickening was relieved (Figure 4e).

The sphenoid sinusitis involved the right side (Figure 4b), and the thickening of the superior ophthalmic vein was not obvious (Figure 5c). In the early morning of the 52nd day of the course, the patient suddenly suffered from shock due to massive nasal hemorrhage, dilation of right pupil, rapid loss of light reflex, respiratory and cardiac arrest. After cardiopulmonary resuscitation, tracheal intubation, ventilator-assisted respiration and other rescue measures, the autonomous cardiac rhythm recovered rapidly, and 3 mm light reflex was restored in the right pupil, with left exophthalmos, ptosis, chemosis, eyeball fixation, and loss of 5 mm light reflex. Blood routine: WBC: 7.44×109/L, N: 2.48×109/L, N%: 33.4%, RBC: 2.2×1012/L, Hb: 69 g/L; brain CT showed that the left sphenoid sinus and part of ethmoid sinus were filled with high-density shadows, i.e. blood clots, the bone wall between the left cavernous sinus and the left sphenoid sinus was damaged and communicated, there was a circular low-density area in the cavernous sinus (Figure 1e), and the cerebral infarction was larger than before (Figure 2c).

In the afternoon of the same day, bilateral femoral artery cannulation was performed under general anesthesia, and femoral arteriography showed irregular aneurysm in the cavernous sinus segment of left internal carotid artery, and severe stenosis of internal carotid artery distal to aneurysm; balloon occlusion test (BOT) was performed simultaneously, and it was found that the right vertebral artery compensated for blood supply to the distal left internal carotid artery through the left posterior communicating artery of the posterior circulation, and the right internal carotid artery supplied blood to the left anterior middle cerebral artery through the anterior communicating artery; finally, one inflated Balt balloon was placed each in the proximal segment of aneurysm in the cavernous sinus segment of left internal carotid artery, i.e., the horizontal petrous segment and the vertical petrous segment, so that the left internal carotid artery was blocked at the proximal end of aneurysm in the cavernous sinus segment to prevent the rerupture and hemorrhage of pseudoaneurysm.

After postoperative anti-infection, blood transfusion, assisted respiration and other treatments, the ventilator and cannula were withdrawn the next day, and then the consciousness became clear. The physical examination showed aphasia, muscle strength of the right upper limb of grade 0-1 and muscle strength of the right lower limb of grade 4, and the symptoms and signs of the left eye remained unchanged. Brain CT reexamination showed that cerebral infarction in the left frontoparietal lobe was larger than before (Figure 2d), left sphenoid sinus and part of ethmoid sinus were filled with blood clots, left cavernous sinus was communicated with left sphenoid sinus, and high-density shadow was seen in cavernous sinus (Figure 1f), which was the change of aneurysm congestion after internal carotid artery occlusion. After consolidation treatment, the patient's condition was improved and discharged, and the patient was admitted to the rehabilitation department for later rehabilitation treatment. During the diagnosis and treatment for more than one month, the patient developed complications such as leukopenia, liver dysfunction, electrolyte disturbance and hypoproteinemia, which were all corrected after symptomatic and supportive treatment.

Discussion

During the course of disease, the patient's clinical symptoms gradually increased and his condition gradually became worse, including left-sided cavernous sinus syndromes such as severe bilateral temporal headache, diplopia and chemosis, pachymeningitis, cerebral infarction induced by left internal carotid artery stenosis, thrombosis in the jugular foramen of bilateral internal jugular veins, pseudoaneurysm in the cavernous sinus segment of left internal carotid artery, epistaxis and hemorrhagic shock. The condition of disease was controlled and the patient's life was saved following the unremitting efforts of a multidisciplinary team, but the patients paid a heavy price, including economic burden and physical damage such as ophthalmoplegia and cerebral infarction. In the diagnosis and treatment of this case, a lot of experience and lessons were also accumulated.

1. What is the root cause of the patient's intractable headache? Is it sphenoid sinusitis?

The patient presented with persistent and progressively extensive severe headache followed by a series of associated symptoms such as diplopia, chemosis, ocular fixation, epistaxis, and hemorrhagic shock, but sphenoid sinusitis was the root cause. The reasons are as follows:

1) The left lateral rectus muscle paralysis occurred after more than 10 days of headache [1], and then cavernous sinus syndromes including paralysis of the ophthalmic division of trigeminal nerve (especially the left side), oculomotor nerve and trochlear nerve and chemosis occurred successively, followed by pachymeningitis, cerebral infarction induced by left internal carotid artery stenosis and inflammatory pseudoaneurysm. The time course conformed to the process of development from the sphenoid sinus to the cavernous sinus and meninges.

2) Imaging findings also supported the presence of sphenoid sinus lesions followed by cavernous sinus damage. In the 2nd week of the course, brain CT in the local hospital showed left sphenoid

sinusitis. On the day of admission to our hospital, brain CT also showed left sphenoid sinusitis, but the change of cavernous sinus was not obvious. With the prolongation of the course, however, CT and MRI revealed that sphenoid sinusitis always existed, and the left cavernous sinus lesions became increasingly more obvious.

3) The clinical symptoms were consistent with the imaging changes: the left sphenoid sinus lesions were found first in the left head, followed by right headache and cavernous sinus syndrome, and finally the left and right cavernous sinus lesions were found.

4) Although the patient had no clinical fever, ESR and hCRP increased significantly earlier, and then elevation of WBC and neutrophils was found. After anti-bacterial infection, headache was relieved rapidly, and ESR and other indicators recovered rapidly, all of which indicated inflammation and bacterial infection.

5) E.N.T. consultation found no fungus in the depth of the nasal cavity. It can be inferred that the patient's symptoms were rooted in sphenoid sinusitis [2,3] and were bacterial sphenoid sinusitis [4] rather than fungal sphenoid sinusitis [5]. The patient had no history of upper respiratory tract infection such as nasal obstruction, runny nose and fever before the onset, and the acute sphenoid sinusitis might be caused by acute attack of chronic sphenoid sinusitis.

2. Sphenoid sinusitis induces "stormy waves".

Simple sphenoid sinusitis is characterized by few symptoms and mild conditions, and damage will be caused to multiple sites in a few cases [1]. Differently, sphenoid sinusitis of this case first caused stubborn severe headache not attributable in the early stage, and involved the cavernous sinus10 days later, causing left abducens nerve injury and diplopia. A few days later, bilateral cavernous sinus syndromes emerged mainly on the left side, and chemosis was caused due to the influence of increased pressure of cavernous sinus inflammation on superior ophthalmic vein reflux. Then imaging and CSF examinations revealed meningitis, mainly pachymeningitis, inflammatory aneurysm in the cavernous sinus segment of the left internal carotid artery, stenosis of the distal segment of the left internal carotid artery, stenotic cerebral infarction, and thrombosis in the jugular foramen of bilateral internal jugular veins. On the 50th day of the course, the inflammatory aneurysm in the cavernous sinus segment of the left internal carotid artery ruptured and bled. A large amount of blood broke through the damaged thin bone plate between the cavernous sinus and the sphenoid sinus and flowed into the sphenoid sinus and ethmoid sinus, causing epistaxis shock, which endangered the patient's life. The above evolution process alert us to the importance of role of sinusitis, especially sphenoid sinusitis, in the early diagnosis and treatment of headache.

3. Pay attention to the diagnosis and differential diagnosis of sphenoid sinusitis in headache disorders.

Sinusitis, especially chronic sinusitis, is common in clinic, and has no symptoms in many people, so it is easily ignored by clinicians and even radiologists. Sphenoid sinusitis is a rare type of sinusitis, especially early isolated sphenoid sinusitis without obvious symptoms or only with non-specific headache or complicated with lateral rectus paralysis [1], which is more prone to misdiagnosis [6]. In this case, the patient had acute onset, stubborn headache, no cold and fever, and less obvious early hemogram changes. Brain CT and MRI revealed left sphenoid sinusitis sometimes. In the case of normal results of the first lumbar puncture, the patient was first diagnosed with giant cell arteritis and painful ophthalmoplegia based on the increase in ESR and hCRP, and hormones were also temporarily effective. On the 19th day of the course, brain CT simultaneously showed left sphenoid sinusitis and slightly increased density of the left cavernous sinus, which suggested possible bacterial infection. After expert consultation, cavernous sinus infection was considered, but sphenoid sinusitis was not determined as its cause. The antibiotic ceftriaxone was given, and the headache continued to be relieved the next day until it finally disappeared.

4. Early diagnosis and treatment is an important measure to prevent and control a series of complications caused by sphenoid sinusitis.

The successful treatment of this patient reflected the spirit of multidisciplinary collaboration. In particular, the Department of Critical Care Medicine worked feverishly on saving the patient from hemorrhagic shock and the Neurosurgery Department blocked the left internal carotid artery to prevent rebleeding, thereby saving the patient's life. If the neurologist raises vigilance and communicates with the radiologist about the diagnosis to offer anti-infection therapy as soon as possible, E.N.T. consultation and sphenoid sinus incision and drainage can be performed early [7], and the proximal artery of the inflammatory aneurysm can be embolized by interventional surgery after neurosurgery consultation, the subsequent complications may not occur or not occur completely. As can be seen from the diagnosis and treatment of this patient, we have to pay enough attention to common sinusitis, especially sphenoid sinusitis. Sphenoid sinusitis is not terrible, but it can cause a series of serious complications and even threaten life. Prompt and effective multidisciplinary collaboration is conducive to the diagnosis and treatment of this disease.

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