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Case Report

Takayasu's Arteritis Presenting with Subarachnoid Hemorrhage Associated with Intracranial Aneurysm and Necrotizing Lesion on the Dorsal Surface of the Foot

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Abstract

Takayasu's arteritis (TA) is an inflammatory vasculitis typically involving the aorta and great branches, the pulmonary artery, and the coronary arteries. The vascular inflammation can cause arterial stenosis, occlusion, thrombosis, and aneurysms. Early recognition of the disease is key to reducing morbidity and mortality. Presently described is the case of a 59-year-old female patient who presented complaining of headache, nausea, vomiting, and uneasiness. Imaging studies revealed a subarachnoid hemorrhage caused by an intracranial aneurysm. The patient underwent embolization therapy, but 1 month after being discharged from the hospital, she was re-admitted due to a necrotizing lesion on the dorsal surface of the foot. The results of magnetic resonance angiography imaging were consistent with TA. TA can be recognized with a careful and structured clinical examination.

Keywords: Necrotizing lesion, subarachnoid hemorrhage, Takayasu's arteritis

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Takayasu's arteritis is a rare, systemic, inflammatory vasculitis of the large blood vessels, usually the aorta and its branches. The inflammation can lead to arterial stenosis, occlusion, thrombosis, and aneurysms. Early diagnosis of the disease is of great importance to reduce mortality and morbidity. It must be kept in mind, particularly in female patients presenting with neurological findings or hypertension with concurrent elevation in acute phase reactants.

The present case had a subarachnoid hemorrhage (SAH) caused by the rupture of aneurysms in the left internal carotid artery and the posterior communicating artery, as well as a necrotizing skin lesion on the dorsal surface of the foot caused by segmental stenosis of the superficial femoral artery.

Case Report

A 59-year-old female patient was admitted to the emergency room due to headache, nausea, vomiting, and uneasiness. The patient's past medical history was remarkable for hypertension, and the patient had been using antihypertensive medications for the last 30 years. The initial assessment in the emergency room provided a blood pressure (BP) reading of 180/90 mmHg, and a neurological examination revealed 3/5 muscle strength in the 4 extremities. The findings on cranial computed tomography (CT) were consistent with widespread SAH (Fig. 1). Cranial magnetic resonance (MR) angiography revealed a 32x31 mm lesion in the left middle cerebral artery (MCA) suspicious for thrombosed aneurysm, severe stenosis and vasospasm in

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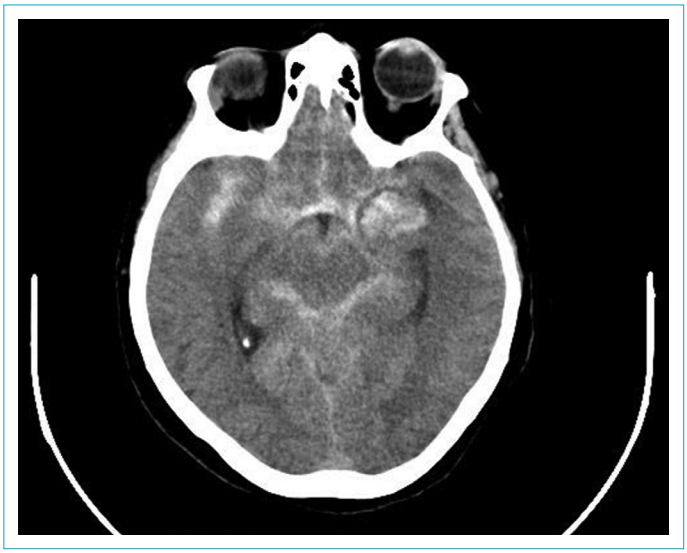

Figure 1. Subarachnoid hemorrhage.

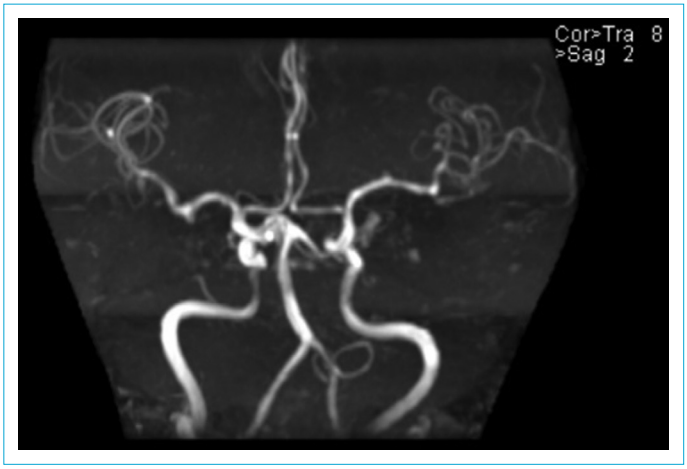

Figure 2. Thrombosed aneurysm and occlusion at the level of the middle cerebral artery.

the MCA at this localization, and thin collateral arteries in the distal portion (Fig. 2). Digital subtraction angiograms showed aneurysms in the left internal carotid artery and posterior communicating artery. The patient was then admitted to the regular ward of the department of neurosurgery, and 10 hours after admission, she was moved to the intensive care unit (ICU) due to impaired consciousness and a Glasgow Coma Scale score of 5. The patient's general condition was good during the follow-up in the ICU, and she was transferred to an external facility in order for embolization therapy to be performed. After the embolization procedure, a control CT (Fig. 3) was performed. The patient was re-admitted to the ICU of our hospital for further follow-up. On physical examination, BP was 160/90 mmHg, and neuromuscular examination revealed motor aphasia, and right upper (1/5) and right lower (2/5) hemiparesis. The

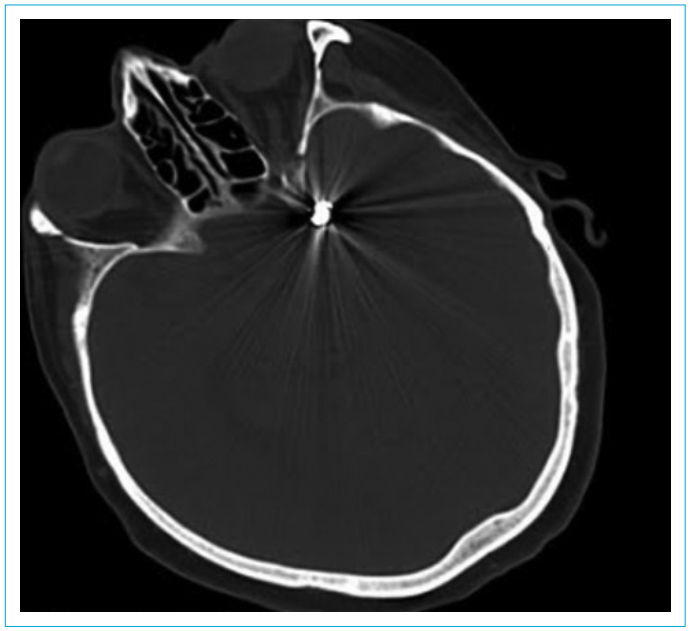

Figure 3. Control computed tomography image after embolization.

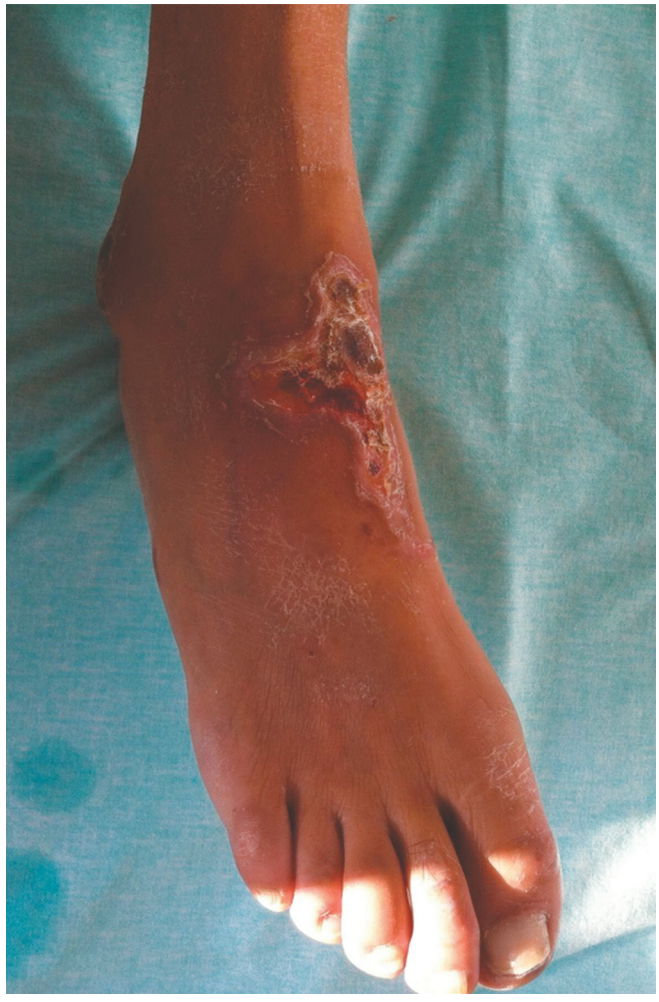

Figure 4. Dorsal aspect of the foot with a superficial necrotizing lesion.

patient was discharged from the hospital after treatment, but she was re-admitted to the emergency room 1 month later due to a general feeling of being unwell and a wound on the dorsal surface of the foot. On initial examination in the emergency room, the patient had motor aphasia and right hemiparesis. There was a 4x5 cm necrotizing lesion on the dorsal surface of the right foot and a 1.5x3 cm open wound lateral to this lesion (Fig. 4). The laboratory work-up included the following: C-reactive protein (CRP): 13.5 mg/dL (normal range: 0.0-0.8 mg/dL), erythrocyte sedimentation rate (ESR): 78 mm/hour (normal range: 1-20 mm/hour). The wound culture did not show bacterial growth. Other laboratory parameters showed normal findings. Echocardiography indicated left ventricular hypertrophy. The patient was hospitalized in the department of internal medicine. On physical examination, pulses were non-palpable on the left and right brachial arteries, bilateral femoral arteries, popliteal arteries, dorsalis pedis artery, and tibialis posterior artery. The markers used in the diagnosis of rheumatic diseases were negative. Abdominal MR angiography revealed results consistent with TA: segmental stenosis in the bilateral iliac arteries, total occlusion in a segment of 5 cm in the left external iliac artery, as well as in the common femoral artery supplied by arterial collateral vessels and the superficial femoral artery. The patient was evaluated by a rheumatologist and azathioprine and steroid treatment were administered. There was an improvement in the general condition of the patient. The patient was discharged from the hospital with follow-up to be conducted at the rheumatology clinic.

After hospital discharge, 1 control is recommended every 3 months for the first year. We suggest annual controls after the first year. A neurological examination should be included. When necessary, a CT or CT angiography scan may be performed.

Discussion

TA is a chronic granulomatous vasculitis The vascular inflammation can cause arterial stenosis, occlusion, thrombosis, and aneurysms.[1] Cerebrovascular accidents may be an important complication determining the prognosis in such patients.[1] Patients with TA rarely present with aneurysmal or non-aneurysmal SAH.[1] Although TA has been considered a rare disease mostly affecting young Asian women, it has also been described in other parts of the world.[1,2] Although the American College of Rheumatology defined age below 40 years as a criterion for TA,[3] disease onset is after the age of 40 years in 13.0% to 17.5% of patients with TA.[3, 4]

Patients with TA usually present with constitutional symptoms before the development of various ischemic symptoms. ESR and CRP are usually elevated in the acute phase, but they also remain normal in one-third of patients. The present case had elevated ESR and CRP levels that could not be explained otherwise.

As arterial lesions ensue, more characteristic features of TA may be found, such as limb claudication, decreased or absent peripheral pulses, vascular bruits, hypertension, and reduction or discrepancies in blood pressure due to stenotic or occlusive lesions between arms.[3]

The pathology of TA is characterized by the involvement of all arterial layers with a variable inflammatory infiltrate, including acute exudative inflammation, chronic and granulomatous inflammation situated mainly in the media and adventitia, while hyperplasia and neovascularization are observed in the intimal layer.[3-6, 7]

Inflammatory lesions in TA eventually evolve to diffuse and/or nodular fibrosis in the arterial wall.[8] The vast majority of arterial lesions in TA are stenotic, and aneurysms can be found in up to one-third of TA patients.[3, 7] Maksimowicz and McKinnon concluded that abnormal intracranial hemodynamic forces play a role in the development of an aneurysm.[1]

Most cases of SAH in TA have been reported as related to intracranial aneurysmal rupture. Based on a literature review, the total number of SAHs due to a proven intracranial aneurysmal rupture was 25 aneurysms in 16 patients.[1-8]

The present case had SAH caused by the rupture of aneurysms in the left internal carotid artery and the posterior communicating artery, as well as a necrotizing skin lesion on the dorsal surface of the foot caused by segmental stenosis of the superficial femoral artery.

The assessment of the arterial lesions and the extent of vascular involvement usually determine the severity of clinical manifestations.[3] The ability to perform daily activities is impaired in up to 74% of TA patients, and 23% to 47% of patients are fully disabled.[3, 9] Vascular claudication impaired performance of routine activities in 60% of TA patients, and the likelihood of disability is significantly associated with the number of disease relapses.[10] Factors that contribute to significant morbidity in TA include heart failure and neurological ischemic events, such as stroke and transient ischemic attacks.

Conclusion

TA can be diagnosed with a careful and well-structured physical examination. Early diagnosis of the disease is of great importance in reducing mortality and morbidity. It must be kept in mind in patients presenting with neurological findings or hypertension with concurrent elevation in acute phase reactants.

Disclosures

Informed Consent: Written informed consent was obtained from the patient who participated in this study.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

Authorship contributions: Concept – G.G.O., F.C.C.Y; Design – G.G.O., F.C.C.Y; Supervision –G.G.O., A.E.K; Materials – G.G.O., A.E.K; Data collection &/or processing – G.G.O., A.E.K; Analysis and/or interpretation – G.G.O., A.E.K; Literature search – G.G.O., A.E.K; Writing –G.G.O; Critical review – G.G.O., A.E.K., S.B.

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