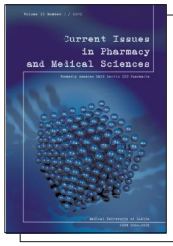

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Neurological complications of temporal arteritis – a case report

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ABSTRACT

The pathophysiology of giant cell arteritis (temporal arteritis) is not fully understood. The disease frequently presents itself with nonspecific complaints like headache, low-grade fever of the unknown origin. The most severe complications of giant cell arteritis include blindness and cerebrovascular incidents. The diagnosis is based on the occurrence of certain factors including age above 50 years and positive results of a temporal artery biopsy. The administration of corticosteroids remains the only proven way of treatment for giant cell arteritis so far. This report presents a case of a patient with temporal arteritis who presented such neurological complications as loss of vision and brain ischemia. A 74 year-old female was admitted to the hospital in Sanok complaining about weakness lasting for about two weeks. She also reported low-grade fever, headache, tenderness, and inflammation along the course of the temporal arteries and bilateral blurred vision (stronger in the right eye). She also complained about jaw and tongue claudication and taste loss. Based on the literature, if giant cell arteritis is suspected, the treatment with glucocorticoids should be initiated promptly to prevent neurological complications.

Keywords: giant cell arteritis, temporal arteritis, treatment, prognosis, neurological complications, visual loss, stroke

INTRODUCTION

Giant cell arteritis (GCA, temporal arteritis) is a systemic inflammatory vasculitis that affects medium and large-sized arteries (involves mainly the temporal artery, vertebral artery, ophthalmic artery, posterior ciliary arteries). The pathophysiology of GCA is not fully understood. After dendritic cell activation in the adventitia, CD4T cells are recruited in the arterial wall and polarized into Th1 and Th17 cells that produce IFN-γ and IL-17. These cytokines activate macrophages, giant cells and smooth muscle cells inducing vascular remodeling leading to ischemic manifestations of GCA. Most commonly, GCA occurs in patients above 50 years of age, with the incidence increasing with age and peaking in the 7-th decade. Women are twice more likely to have temporal arteritis than men [7,9].

The hallmark syndrome of GCA is headache often unilateral in area of superficial temporal artery. Temporal artery usually presents abnormalities like tender, nodular, swollen with decreased or absent pulse. Patients with GCA can experience visual symptoms such as transient visual blurring, diplopia, permanent unilateral visual loss caused-by central retinal artery occlusion. In addition, typical symptoms include the pain in the jaw or the tongue after eating or chewing, odynophagia. Systemic manifestations are malaise, low grade fever, anorexia, weight loss, myalgia. Around 30% of patients experience neurological problems – peripheral neuropathies, transient ischemic attacks, stroke, and neuropsychiatric syndromes [6].

We report a case of a 74 year-old woman with temporal arteritis who presented neurological complications like brain ischemia and visual loss.

CASE REPORT

A 74 year – old female was admitted to the Medical Department of the Hospital in Sanok with main complaints about weakness that lasted for two weeks, low-grade fever, headache, tenderness and inflammation along the course of the temporal arteries and bilateral blurred vision (stronger in the right eye). She also complained about jaw and tongue claudication and tastelessness. She had a history of: a duodenal ulcer and a partial resection of the left lung (tumor). She was treated for hypertension – indapamide, perindopril arginine. Because of the headache and blurred vision, she was relocated to the Neurological Department.

Blood tests showed: ESR-110 mm/h, CRP-82.4 mg/l, RBC-3.78 mln/ul,WBC-14 th/ul, HGB-11.3 g/dl, HCT-33.7%, PLT-255 th/ul, blood sugar-94 mg/dl, Na-144 mmol/l, K-4.12 mmol/l, urea-37 mg/dl, cholesterol-106 mg/dl, HDL-44 mg/dl, LDL-51 mg/dl, TG-53 mg/dl. Because of the visual morbidity, the patient was examined by ophthalmologist. She was diagnosed with the central retinal artery occlusion. Based on the American College of Rheumatology, diagnostic criteria and clinical manifestations the GCA were diagnosed. Medical treatment started with the administration of the methylprednisolone 1g IV for 3 days as an initial dose. Adjuvant drugs were: metoprolol, potassium

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chloride, perindopril arginine, indapamide, enoxaparin sodium, metamizole sodium. Within 3 days, ESR had fallen to 90 mm/h. Magnetic Resonance Imaging (MRI) of the brain showed small infarction foci localized in the right frontal lobe white matter. The methylprednisolone IV was replaced with p.o. prednisone 30 mg/daily - but symptoms recurred. This way it was replaced with IV dexamethasone 3x16 mg daily. After this therapy the left temporal artery became painless, the right one remained nodular and painful. The headache almost disappeared. ESR was about 84 mm/h. Dosage of dexamethasone was reduced (16+8+16 mg/day). Ophthalmological examination was repeated. The main diagnosis was ischemic optic neuropathy. General symptoms had resolved and ESR normalized up to 20 mm/h. The patient was discharged from the hospital. Ambulatory treatment was advised – p.o. methylprednisolone 128 mg daily, pantoprazole 20 mg, metoprolol 50 mg 2x1/2, indapamide 1x1, perindopril arginine 5 mg 1x1/2, potassium chloride 1x1. The patient was also advised to stay in touch with the Neurological Out-Patient Clinic. Despite the intensive treatment, the right eye stayed blind.

DISCUSSION

The woman with GCA presented neurological complications like brain ischemia and visual loss. Involvement of the peripheral nervous system is more uncommon than cerebral ischemia and neuroophtalmological complications in patients suffering from GCA [5].

According to Chan et al. [1] patients with blindness and cerebrovascular incidents should be treated with high-dose steroid therapy. Additional antiplatelet therapy is also beneficial.

According to Kermani et al. [3] if GCA is suspected, the treatment with glucocorticoids should be initiated promptly to prevent further vision loss. It cannot be excluded that in the presented case, the steroid therapy was introduced too late and this is why the neurological complications occurred. However, according to Moroianu et al. [4] initial diagnosis of GCA may be difficult because neurological manifestations are intermittent and typical signs of GCA may be absent. Furthermore, the diverse clinical presentations of temporal arteritis may hamper the diagnosis [10]. Despite corticotherapy the progression of neurological symptoms may occur. Chansson et al. [2] reported the case of a patient with GCA who developed neurological deterioration after the onset of the corticotherapy. Similarly, Schmidt et al. [8] presented that the patients with temporal arteritis developed bilateral blindness and cerebral stroke. Moreover, these complications occurred despite the corticosteroid treatment.

CONCLUSIONS

1. GCA causes neurological complications in patients.
2. If GCA is suspected, the treatment with glucocorticoids should be initiated promptly to prevent neurological complications.

REFERENCES

1. Chan M, Lugmani R: Pharmacotherapy of vasculitis. *Expert Opin. Pharmacother.*, 10, 1273, 2009.
2. Chansson N et al.: Bilateral intracerebral angioplasty in a patient with stroke caused by giant cell arteritis. *Rev. Neurol.* (Paris), 166, 328, 2010.
3. Kermani TA, Warrington KJ: Recent advances in diagnostic strategies for giant cell arteri*tis. Curr. Neurol. Neurosci. Rep.*, 12, 138, 2012.
4. Moroianu C et al.: Fatal giant cell arteritis with severe bilateral involvement of the vertebral arteries. *Rev. Neurol.* (Paris), 162, 876, 2006.
5. Pfadenhauer K, Roesler A, Golling A: The involvement of the peripheral nervous system in biopsy proven active giant cell arteritis. *J. Neurol.*, 254, 751, 2007.
6. Rozwodowska M, El-Essa A, Kubica J: Olbrzymiokomórkowe zapalenie tętnic (zapalenie tętnicy skroniowej). Opis 2 przypadków. *Choroby Serca i Naczyń,* 3, 211, 2006.
7. Samson M, Bonnotte B: *Giant-cell arteritis pathogenesis.* Presse Med., 2012 Aug 14. [Epub ahead of print]
8. Schmidt D, Vaith P, Hetzel A: Prevention of serious ophthalmic and cerebral complications in temporal arteritis? *Clin. Exp. Rheumatol.*, 18 (Suppl 20), 61, 2000.
9. Wiszniewska M, Członkowska A: Rola zapalenia naczyń w etiologii udaru mózgu. *Udar mózgu,* 10, 8, 2008.
10. Zamarbide ID, Maxit MJ: Fisher's one and half syndrome with facial palsy as clinical presentation of giant cell temporal arteritis. *Medicina* (*B Aires*), 60, 245, 2000.