Methods.—The dilatory responses of the brachial artery to post-ischemic increased blood flow (endothelium-dependent flow-associated dilatation (FAD) and to sublingually administered nitroglycerin (endothelium-independent nitroglycerin-induced dilatation (NID)) were measured by ultrasound in 24 patients with newly diagnosed AAV (female/male: 9/15, age: 55 ± 12 years) which were compared to 24 healthy controls (female/male: 9/15, age: 59 ± 7 years). Plasma concentration of vWF was measured by ELISA. Patients were reexamined after 6 and 12 months of immunosuppression (plasmapheresis, corticosteroids and cyclophosphamide followed by azathioprine). All patients reached remission during follow-up.

Results.—NID was impaired in AAV patients as compared to controls (109 ± 6 vs. 123 ± 5%, \( P < 10^{-6} \)) whereas a difference in FAD was insignificant (102 ± 3 vs. 104 ± 4%, \( P = 0.17 \)). Plasma vWF was increased in patients (2.0 [1.0–3.8] kIU/L vs. 1.1 [0.4–1.8], \( P < 10^{-4} \)). One year of immunosuppression did not change NID, FAD or vWF.

Discussion.—AAV affects small vessels but has also been shown to increase large-vessel cardiovascular risk beyond traditional risk factors. Impaired dilatory capacity and elevated vWF have both been associated with vascular damage and increased cardiovascular risk in other patient groups. Hence, the findings of the present study may represent mechanisms in AAV behind increased cardiovascular risk in this disease.

Conclusion.—This study indicates that AAV results in a sustained damage to major vessels which could be a potential link between AVV and increased cardiovascular risk.

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Initial presentation of giant cell arteritis with ischemic optic neuropathy without headache: Report of four cases

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Introduction.—Anterior ischemic optic neuropathy (AION) is the most common ocular presentation of giant cell arteritis (GCA). Although, AION in GCA is usually unilateral at presentation, contralateral disease can occur within days or weeks if treatment is not begun immediately or is stopped while the disease is active. We present four cases of AION associated with GCA.

Methods.—We identified four patients with AION which met the criteria for GCA. Retrospectively, clinical, laboratory and histological data were analyzed.

Results.—Four patients; three females aged 79, 82, and 83 years, and 1 male aged 66 years, were studied. The onset of visual loss was acute and monocular over 1 and 4 days, but in one patient progressed to the contralateral eye in a few days. Ophthalmic examination showed swelling of the optic disc affected in three patients, and was normal in the 4th patient. We observe severe impaired vision, abnormal evoked visual potentials, and elevated ESR and RCP, in all the cases. Methylprednisolone was started immediately, without waiting for the biopsy to be completed, and followed by oral prednisone. GCA was confirmed by temporal artery biopsy in three patients and was negative in one patient, which met the diagnostic criteria for GCA. As the disc oedema subsides, optic atrophy becomes evident in all patients. Despite treatment with steroids visual loss remained unchanged.

Discussion.—Only, a few reports have documented in patients with AION and GCA visual recovery after steroids therapy. Once ischemic infarction of the optic nerve head is established persistent loss of vision remains. The ischemic insult in AION is typically due to occlusive disease of the posterior ciliary arteries, which supply the optic nerve. Steroid therapy should be started as soon as the diagnosis of GCA is suspected.

Conclusion.—In patients with loss of vision and increased levels of acute phase reactants the possibility of AION associated with GCA should be considered, and an immediate steroid therapy, and order a temporal artery biopsy are indicated.

Further reading

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