

Influence of corticosteroid treatment on MRI findings in giant cell arteritis

T. A. Bley · T. Ness · K. Warnatz · A. Frydrychowicz ·
M. Uhl · J. Hennig · M. Langer · M. Markl

Received: 2 August 2006 / Accepted: 15 August 2006 / Published online: 5 October 2006
© Clinical Rheumatology 2006

Abstract Giant cell arteritis (GCA) remains a diagnostic challenge. With the use of a high-resolution MRI protocol, visualization of the superficial cranial arteries is feasible and mural inflammation can be assessed noninvasively. Until today, it is not known how soon inflammatory signals in diagnostic MR imaging vanish after initiation of treatment. Here, we report sequential MR imaging findings during the initial weeks of corticosteroid treatment in a 79-year-old female patient with histologically proven GCA. Mural inflammatory changes decreased within the first 2 weeks and have almost entirely vanished after 2 1/2 months of continued treatment. Moreover, MR angiography revealed sequential stenoses of the subclavian artery, which improved in variable extent with some residuals despite high dose steroid medication. This report underlines the value of high-resolution MRI in diagnosis and follow-up of GCA and illustrates the potential of MRI to detect and monitor intra- and extra-cranial involvement patterns of GCA in high detail.

Keywords Arteries · Corticosteroids · Giant cell arteritis · MR angiography · Vasculitis

T. A. Bley (✉) · A. Frydrychowicz · M. Uhl · J. Hennig ·
M. Langer · M. Markl
Department of Diagnostic Radiology and Medical Physics,
University Hospital Freiburg,
Hugstetter Strasse 55,
79106 Freiburg, Germany
e-mail: thorsten.bley@uniklinik-freiburg.de

T. Ness
Department of Ophthalmology, University Hospital Freiburg,
Freiburg, Germany

K. Warnatz
Department of Rheumatology and Clinical Immunology,
University Hospital Freiburg,
Freiburg, Germany

Introduction

Due to its variable clinical presentation and unspecific symptoms, the noninvasive diagnosis of giant cell arteritis (GCA) is challenging. Although the clinical criteria of the American College of Rheumatology [1] assist in finding the correct diagnosis, concordance with these criteria does not necessarily provide the correct conclusion in the individual patient [2]. Color-coded duplex ultrasonography has proven feasible in the hands of experienced observers [3–5] with its sensitivity being dependent on the pretest likelihood of the disease [6]. Recently, a noninvasive MRI protocol for the assessment of mural inflammatory changes in the superficial temporal arteries has been introduced [7]. In a single MRI examination, the cranial involvement pattern (i.e., location and extent of inflammatory changes of the small superficial cranial arteries) can be assessed in high detail [8]. Furthermore, this protocol can be amended by a MR angiography of the thoracic aorta and the supra-aortic arteries to accomplish a single comprehensive noninvasive imaging study of the thoracic, cervical, and cranial vasculature, which can be performed in less than 40 min [9–11].

Until today, it is not known how soon inflammatory signs in diagnostic MR imaging vanish after initiation of treatment. We report sequential MR imaging findings in a patient with histologically proven GCA during the initial weeks of corticosteroid treatment.

Case report

A 79-year-old female patient presented with an impairment of visual function due to anterior ischemic optic neuropathy (AION). There were no classical symptoms of GCA,

especially headache or jaw claudication, but erythrocyte sedimentation rate (75 mm after the first hour), C-reactive protein (63 mg/l), and fibrinogen (856 mg/dl) were highly elevated. These laboratory values indicated GCA as the cause of AION. Temporal artery biopsy revealed the typical aspect of GCA. After initial high-dose intravenous corticosteroid therapy, laboratory values normalized within 1 week and remained normal over the next 3 months.

High-resolution MR follow-up examinations of the supra-aortic and superficial cranial arteries revealed significant improvement of vasculitic findings during the course of steroid treatment: At initial presentation, three sequential stenoses of the left subclavian artery were found in MR

angiography (arrows in Fig. 1a), demonstrating extra-cranial manifestations of GCA in this patient. At the same time, inflammatory contrast enhancement and thickening of the vessel wall were depicted in enlarged, high-resolution MR images of the left superficial temporal artery (arrows in Fig. 1d). After 2 weeks of corticosteroid treatment, subclavian artery stenoses were still present (arrows in Fig. 1b), whereas mural inflammatory changes of the superficial left temporal artery had decreased (open arrows in Fig. 1e). Further MRI follow-up after 2 1/2 months of continued steroid medication revealed a substantial improvement regarding the proximal and the further distal subclavian stenoses (open arrows in Fig. 1c) while the third

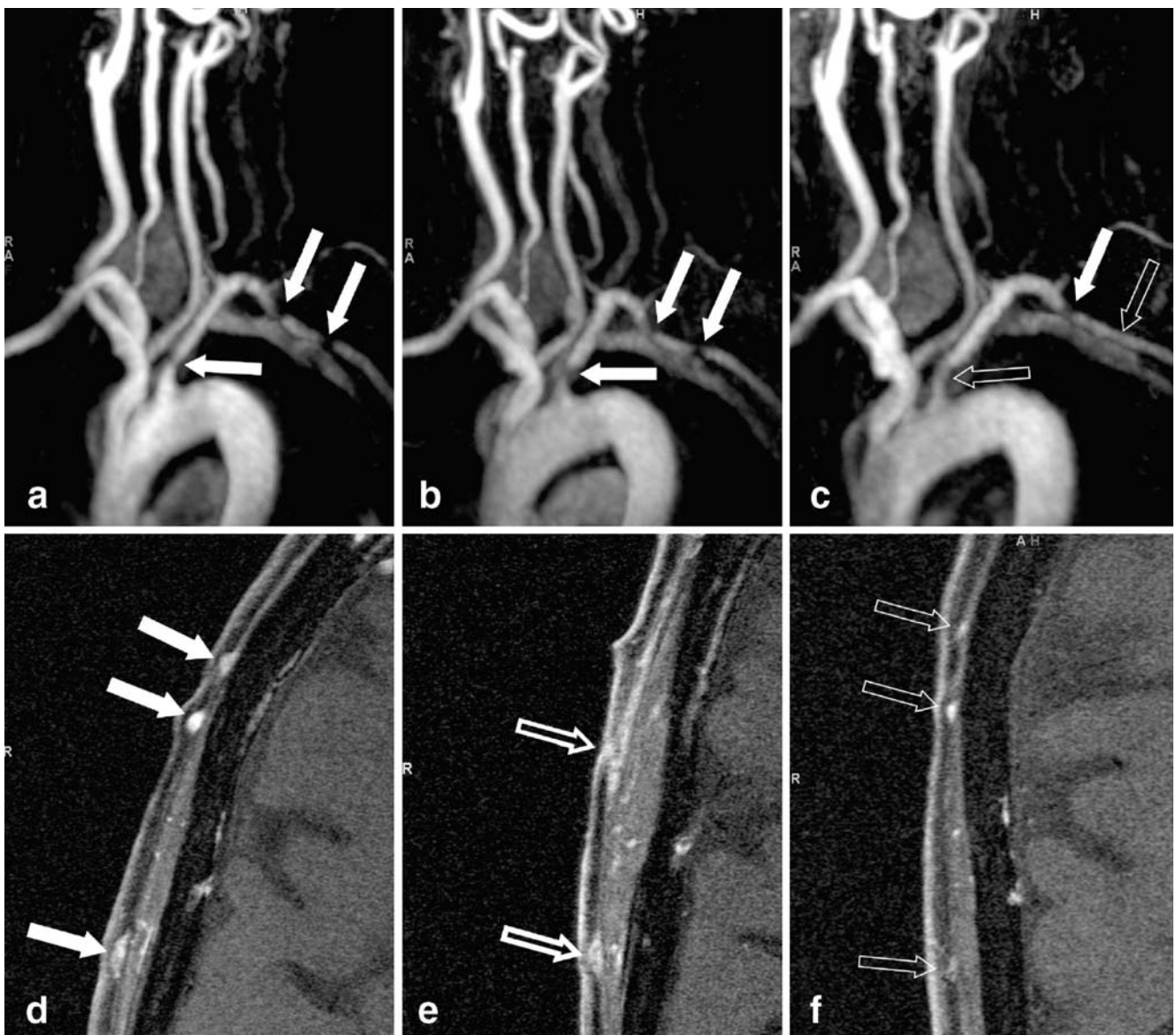


Fig. 1 MR angiography of the aortic arch and the supra-aortic arteries reveals three sequential stenoses of the left subclavian artery (arrows in a) that are still present after 2 weeks of corticosteroid treatment (arrows in b). Follow-up after 2 1/2 months of continued steroid medication revealed a substantial improvement (open arrows in c),

while one stenosis (solid arrow in c) remained. Enlarged high-resolution MR images of the left superficial temporal artery (arrows in d) depict mural inflammatory enhancement that had decreased after 2 weeks of steroid medication (open arrows in e) and had almost entirely vanished after 2 1/2 months (light arrows in f)

stenosis (solid arrow in Fig. 1c) remained. At this time, the mural inflammatory signs at the left superficial temporal artery had almost entirely vanished (light arrows in Fig. 1f).

Discussion

This report reveals the value of high-resolution MRI in diagnosis and follow-up of GCA. At initial presentation, mural inflammatory changes of the superficial cranial arteries could be successfully demonstrated by noninvasive MRI. During the first weeks of steroid treatment, these inflammatory signs decreased substantially as depicted by sequential follow-up MRI examinations. Stenoses of the supra-aortic arteries have improved in variable extent with some residuals despite high dose steroid medication. This illustrates the potential of high-resolution MRI to detect and monitor intra- and extra-cranial involvement patterns of GCA in high spatial and temporal detail.

Temporal artery biopsy, which typically reveals multinucleated giant cells or signs of granulomatous inflammation, is considered the diagnostic gold standard in GCA [12]. Even after 2 weeks of corticosteroid treatment, biopsy may reveal signs of GCA in cases of active disease [13]. In addition, high-resolution MRI can provide information about the effect of steroid treatment on vascular inflammatory changes such that signs of mural inflammation vanish under long-term corticosteroid treatment [14]. Decreased enhancement in large-vessel vasculitis presumably relates to improvement of disease status [15, 16]. However, the exact temporal evolution of segmental vascular changes, as depicted by MRI associated with steroid treatment, has not been investigated so far. As mural hyperenhancement can undergo considerable changes within only a few days of corticosteroid treatment, it is recommended to examine the patients suspected of GCA before or at least as early as possible after initiation of treatment. However, since irreversible visual loss is a dreaded complication of GCA, corticosteroid therapy needs immediate initiation.

Upper extremity artery balloon angioplasty in combination with immunosuppressive therapy represents an efficient method for treatment of stenoses in extra-cranial GCA [17]. In this case, the remaining subclavian artery stenosis was clinically not relevant at the time of the third investigation. Therefore, balloon angioplasty was not performed.

Potentially, MRI may be used to monitor treatment effects and to indicate relapse of disease. Yet, the significance of MRI findings in the time course of steroid treatment needs to be evaluated in larger patient studies.

References

- Hunder GG, Bloch DA, Michel BA et al (1990) The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. *Arthritis Rheum* 33(8):1122–1128
- Bloch DA, Moses LE, Michel BA (1990) Statistical approaches to classification. Methods for developing classification and other criteria rules. *Arthritis Rheum* 33(8):1137–1144
- Schmidt WA, Kraft HE, Vorpahl K, Volker L, Gromnica-Ihle EJ (1997) Color duplex ultrasonography in the diagnosis of temporal arteritis. *N Engl J Med* 337(19):1336–1342
- Reinhard M, Schmidt D, Hetzel A (2004) Color-coded sonography in suspected temporal arteritis—experiences after 83 cases. *Rheumatol Int* 24:340–346
- Pfadenhauer K, Weber H (2003) Giant cell arteritis of the occipital arteries—a prospective color coded duplex sonography study in 78 patients. *J Neurol* 250(7):844
- Karassa FB, Matsagas MI, Schmidt WA, Ioannidis JP (2005) Meta-analysis: test performance of ultrasonography for giant-cell arteritis. *Ann Intern Med* 142(5):359–369
- Bley TA, Wieben O, Uhl M, Thiel J, Schmidt D, Langer M (2005) High-resolution MRI in giant cell arteritis: imaging of the wall of the superficial temporal artery. *AJR Am J Roentgenol* 184(1):283–287
- Bley TA, Weiben O, Uhl M et al (2005) Assessment of the cranial involvement pattern of giant cell arteritis with 3T magnetic resonance imaging. *Arthritis Rheum* 52(8):2470–2477
- Bley TA, Wieben O, Uhl M, Miehle N, Langer M, Hennig J, Markl M (2005) Integrated head-thoracic vascular MRI at 3T: assessment of cranial, cervical and thoracic involvement of giant cell arteritis. *Magn Reson Mater Phys* 18(4):193–200
- Markl M, Uhl M, Wieben O, Ness T, Langer M, Hennig J, Bley TA (2006) High resolution 3T MRI for the assessment of cervical and superficial cranial arteries in giant cell arteritis. *J Magn Reson Imaging* 24(2):423–427
- Bley TA, Uhl M, Venhoff N, Thoden J, Langer M, Markl M (2006) 3-T MRI reveals cranial and thoracic inflammatory changes in giant cell arteritis. *Clin Rheumatol*. DOI 10.1007/s10067-005-0160-7
- Weyand CM, Goronzy JJ (2003) Medium- and large-vessel vasculitis. *N Engl J Med* 349(2):160–169
- Achkar AA, Lie JT, Hunder GG, O'Fallon WM, Gabriel SE (1994) How does previous corticosteroid treatment affect the biopsy findings in giant cell (temporal) arteritis? *Ann Intern Med* 120(12):987–992
- Bley TA, Warnatz K, Wieben O et al (2005) High-resolution MRI in giant cell arteritis with multiple inflammatory stenoses in both calves. *Rheumatol (Oxf Print)* 44(7):954–955
- Choe YH, Kim DK, Koh EM, Do YS, Lee WR (1999) Takayasu arteritis: diagnosis with MR imaging and MR angiography in acute and chronic active stages. *J Magn Reson Imaging* 10(5):751–757
- Matsunaga N, Hayashi K, Sakamoto I, Matsuoka Y, Ogawa Y, Honjo K, Takano K (1998) Takayasu arteritis: MR manifestations and diagnosis of acute and chronic phase. *J Magn Reson Imaging* 8(2):406–414 (Review)
- Both M, Aries PM, Muller-Hulsbeck S, Jahnke T, Schafer PJ, Gross WL, Heller M, Reuter M (2006) Balloon angioplasty of upper extremity arteries in patients with extracranial giant cell arteritis. *Ann Rheum Dis* 65:1124–1130