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3-T MRI reveals cranial and thoracic inflammatory changes in giant cell arteritis

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Abstract Giant cell arteritis (GCA) is a diagnostic challenge. The correct diagnosis is needed for immediate initiation of corticosteroid treatment since blindness is a dreaded complication. Typically, the superficial cranial arteries are affected by this granulomatous vasculitis of large- and medium-sized arteries. However, GCA is not limited to the cranial arteries. Involvement of various arteries such as the cervical and thoracic arteries can also occur. Here, we report a case of histologically proven GCA with cranial and extracranial involvement. We illustrate the usefulness of a comprehensive vascular high-resolution magnetic resonance imaging examination that combines assessment of mural inflammatory changes of the small temporal and occipital arteries with the evaluation of extracranial vasculature to assist in the difficult non-invasive diagnosis and to determine the extent of this inflammatory disease.

Keywords Giant cell arteritis · MRA · MRI · Mural inflammation · Temporal artery

Introduction

Giant cell arteritis (GCA) is a granulomatous vasculitis of large- and medium-sized arteries, and it usually involves the superficial cranial arteries [1, 2]. The clinical diagnosis of GCA is often challenging. Criteria for classification of GCA were proposed by the American College of Rheumatology (ACR) [3]. Characteristic findings of temporal

arteritis can be visualized by colour duplex ultrasonography, with a dark halo being the most specific sign. Its clinical value in the diagnosis of GCA, however, has been debated [4–7].

Next to the superficial cranial arteries, involvement of other vascular structures such as the vertebral arteries, the aorta and its branches, the coronary arteries, the mesenteric arteries and the lower leg arteries can also occur [8–14]. Because multiple regions can be affected it would be desirable to characterize the complete vascular involvement pattern of the individual patient. We have recently developed a novel protocol for integrated head-thoracic vascular magnetic resonance imaging (MRI) at 3 T for the comprehensive assessment of inflammatory changes in the head, neck and thoracic aorta. The combination of first-pass imaging of a single-dose contrast agent (MR angiography; MRA) with post-contrast high-resolution head imaging allowed for the simultaneous analysis of the small superficial cranial arteries and the thoracic aorta and supra-aortic branches, including a large section of the subclavian arteries [15]. In the present article we report our findings from a patient with histologically proven GCA to illustrate the usefulness of such a comprehensive assessment of inflammatory disease (Fig. 1).

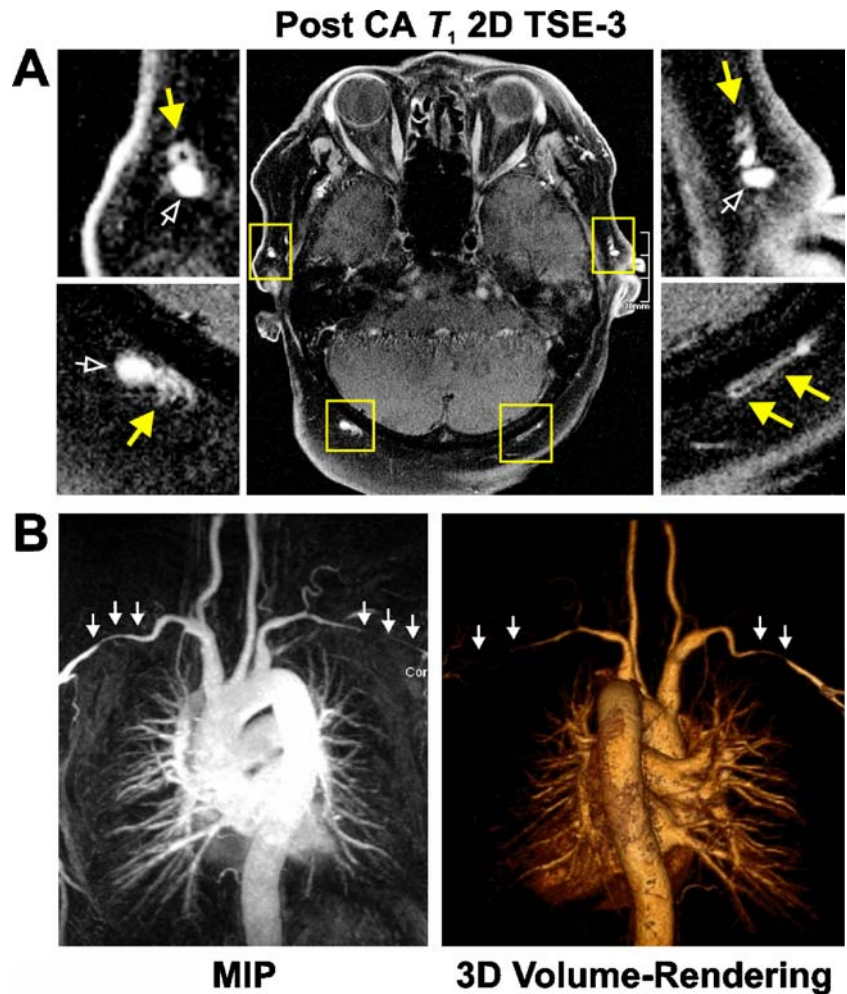
Case report

A 67-year-old female patient with histologically proven GCA presented with progressive, generalized myalgias and arthralgias 8 months after initial diagnosis of GCA. Pertinent findings on physical examination were murmurs over both subclavian arteries with normal peripheral pulses. A difference in blood pressure of both upper extremities was found with 140/90 mm Hg on the right side and 115/90 mm Hg on the left side. C-reactive protein was elevated to 8.3 mg/dl (normal, <0.5 mg/dl), and erythrocyte sedimentation rate was 61 mm in the first hour according to Westergreen (normal <20 mm). These findings were interpreted as relapse of GCA with claudications of both upper extremities probably due to too-fast tapering of the steroid medication. Initially, cortico-

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Fig. 1 A 67-year-old female patient with histologically proven GCA and associated inflammatory signal changes in the superficial cranial arteries (*top*) and stenoses in the supra-aortic vessels (*bottom*). **a** High-resolution T1-weighted head images. Mural inflammatory signal enhancement in superficial cranial arteries can clearly be identified and is shown for the magnified occipital and temporal arteries. Signal enhancement due to the accumulation of contrast agent and circumferential luminal thickening can be appreciated for all four arteries (*solid white arrows*). Note the flow void in the arterial system due to higher velocity (outflow effects) if compared to venous signal (*open white arrows*). **b** Contrast-enhanced MRA. Maximum intensity projection (MIP, *left*) and 3-D volume-rendering (*right*) based on a CE-MRA of the thoracic aorta. Multisegmental stenoses in the left and right subclavian arteries are clearly visible (*solid white arrows*)



steroid retreatment was immediately started with a high dose of steroids (1 mg/kg body weight). According to clinical and serological assessment, steroids were gradually tapered to a daily dose of 5 mg/day at the time of her second presentation. The patient was referred to MRI for assessment of inflammatory involvement of her superficial cranial arteries and for assessment of possible inflammatory stenoses of the supra-aortic arteries. The MRI examination was performed on a 3-T system (TRIO, Siemens Medical Solutions, Erlangen, Germany). To permit a comprehensive examination of the thorax and head, the total exam was subdivided into two parts.

For the first part of the exam, thoracic images were acquired using an eight-channel phased array body coil. Contrast-enhanced (CE)-MRA was performed using intravenous administration of gadolinium contrast agent (gadobenate dimeglumine, Gd-BOPTA chelate, Multihance, ALTANA Pharma, Konstanz, Germany, single dose=0.1 mmol/kg body weight, injection rate=3 ml/s) after a 2-ml test bolus to estimate the contrast agent arrival time in the aortic arch. Background signal was suppressed by means of subtraction of a full-resolution pre-contrast data set. Data were acquired in coronal orientation to permit the assessment of large parts of the subclavian arteries. Parallel imaging was used to accelerate data acquisition and consisted of a generalized

autocalibrating partially parallel acquisition (GRAPPA) reconstruction with an acceleration factor of 2 with 32 reference lines [16]. The resulting 3-D data volume included the entire thoracic aorta and large portions of the supra-aortic arteries, with a spatial resolution of $0.8 \times 1.2 \times 1.25\text{--}5\text{ mm}^3$.

For the second part of the exam, the patient was repositioned in the scanner, and the coil was exchanged by an eight-channel phased array head coil. Arterial wall imaging with high in-plane submillimetre spatial resolution ($0.2 \times 0.3 \times 3\text{ mm}^3$) was performed approximately 10–15 min after the injection of the contrast agent using a T1-weighted turbo spin echo sequence with an echo train length of 3 (TSE-3).

Discussion

A comprehensive assessment of the inflammatory involvement pattern of the superficial cranial, cervical and thoracic arteries was achieved with the use of the presented integrated MRI approach. Morphological analysis of the superficial occipital and temporal arteries revealed thickened and inflamed vessel walls, indicating a relapse of GCA (Fig. 1a, yellow arrows). In addition, inflammatory stenotic changes in both subclavian arteries with predomi-

nance of the left side could be identified using the contrast-enhanced MRA (Fig. 1b, white arrows). The vascular geometry of the aorta and cervical arteries was inconspicuous. These findings lead to an immediate increase in her steroid medication dose to 1 mg/kg body weight, which improved her symptoms and reduced the laboratory scores of acute inflammatory response.

The presented MR examination has been performed using a high field strength 3-T MRI scanner. When investigating small structures, like the superficial cranial arteries, submillimetre spatial resolution with the highest achievable signal and the lowest image noise is desirable. Theoretically, 3-T MRI renders twice the signal of 1.5-T scanners [17, 18]. Therefore, the higher field strength is favorable for this purpose. However, we have shown that high-resolution MRI of the superficial temporal arteries is also feasible with the more widely distributed 1.5-T MRI scanner [19, 20].

The presented MRI examination is easy to perform within less than 40 min. Compared to colour-coded duplex ultrasonography equally high spatial resolution was achieved. Note that, although reading of the high-resolution MR images may need a fair amount of training, MR examinations are, in general, considerably less observer-dependant than colour-coded duplex ultrasonography. For future protocols, the comprehensive but still-limited vascular coverage may be extended by the application of whole-body MRA to permit the assessment of whole-body vascular involvement patterns in patients with GCA.

This case demonstrates that high-resolution post-contrast MRI, combined with an angiogram of the thoracic aorta and the supra-aortic arteries, can provide important additional information in the diagnosis and follow-up of GCA. The recurrence of GCA was identified as mural inflammation in multiple segments of the cranial arteries and as stenoses in the subclavian arteries.

Take home message

High-resolution MRI assists in the non-invasive diagnosis of GCA and reveals the cranial, cervical and upper thoracic vascular involvement pattern within a single comprehensive MR examination.

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References

- Horton B, Magath T, Brown G (1932) An undescribed form of arteritis of the temporal vessels. *Proc Staff Mtg Mayo Clin* 7:700–701
- Salvarani C, Cantini F, Boiardi L, Hunder GG (2002) Polymyalgia rheumatica and giant cell arteritis. *N Engl J Med* 347(4):261–271
- Hunder GG, Bloch DA, Michel BA, Stevens MB, Arend WP, Calabrese LH et al (1990) The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. *Arthritis Rheum* 33:1122–1128
- 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 (2003) Color-coded sonography in suspected temporal arteritis—experiences after 83 cases. *Rheumatol Int* 24(6):340–346
- Salvarani C, Silingardi M, Ghirarduzzi A et al (2002) Is duplex ultrasonography useful for the diagnosis of giant-cell arteritis? *Ann Intern Med* 137(4):232–238
- 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
- Ronthal M, Gonzalez RG, Smith RN, Frosch MP (2003) Case records of the Massachusetts General Hospital. Weekly clinicopathological exercises. Case 21-2003. A 72-year-old man with repetitive strokes in the posterior circulation. *N Engl J Med* 349(2):170–180
- Wilkinson IM, Russell RW (1972) Arteries of the head and neck in giant cell arteritis. A pathological study to show the pattern of arterial involvement. *Arch Neurol* 27(5):378–391
- de Gennes C, Le Thi Huong D, Wechsler B et al (1989) Temporal arteritis revealed by upper limb gangrene. *J Rheumatol* 16(1):130–132
- Lie JT, Failoni DD, Davis DC Jr (1986) Temporal arteritis with giant cell aortitis, coronary arteritis, and myocardial infarction. *Arch Pathol Lab Med* 110(9):857–860
- Liozon F, Weinbreck P, Vidal E et al (1986) Arterial stenoses of the arms in Horton's temporal arteritis. Apropos of 3 cases. A review of the literature. *Ann Med Interne (Paris)* 137(4):307–312
- Bley TA, Warnatz K, Wieben O et al (2005) High-resolution MRI in giant cell arteritis with multiple inflammatory stenoses in both calves. *Rheumatology (Oxford)* 44(7):954–955
- Helfgott SM, Bauer MR (1987) Pedal gangrene caused by giant cell arteritis. *Arthritis Rheum* 30(9):1078–1079
- Bley TA, Wieben O, Uhl M, Miehle N, Hennig J, Langer M, Markl M (2005) Integrated head-thoracic vascular MRI at 3 T: assessment of cranial, cervical and thoracic involvement of giant cell arteritis. *MAGMA* 18(4):193–200
- Griswold MA, Jakob PM, Heidemann RM, Nittka M, Jellus V, Wang J, Kiefer B, Haase A (2002) Generalized autocalibrating partially parallel acquisitions (GRAPPA). *Magn Reson Med* 47(6):1202–1210
- Norris DG (2003) High field human imaging. *J Magn Reson Imaging* 18(5):519–529
- Trattning S, Ba-Ssalamah A, Noebauer-Huhmann IM et al (2003) MR contrast agent at high field MRI (3 Tesla). *Top Magn Reson Imaging* 14(5):365–375
- 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, Wieben O, Leupold J, Uhl M (2005) MRI findings in temporal arteritis. *Circulation* 111:e260