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MRI of idiopathic orbital inflammatory syndrome using fat saturation and Gd-DTPA

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Abstract Idiopathic orbital inflammatory syndrome encompasses a group of inflammatory conditions for which no systemic or local cause can be found, and is commonly referred to as orbital pseudotumour. On conventional MRI sequences subtle areas of inflammation or enhancing tissue can easily be masked by the high signal intensity of orbital fat and involvement of the fat itself may not be appreciated. We describe the MRI features of three patients with idiopathic orbital inflammation using frequency-selective fat saturation and Gd-DTPA.

Key words Idiopathic orbital inflammatory syndrome · Orbital pseudotumour · Fat saturation · Gadolinium · Magnetic resonance imaging

Introduction

The commonest cause of an intraorbital mass lesion in the adult is idiopathic orbital pseudotumour [1], which is part of the spectrum of idiopathic orbital inflammation (IOI). Diagnosis of IOI is usually clinical, with a classical triad of pain, ocular paresis and proptosis and rapid improvement with steroid therapy. We describe the value of MRI using fat saturation and Gd-DTPA.

Patients and methods

We studied three patients with presumed IOI, diagnosed by clinical course and response to therapy. In all three, alternative causes of orbital inflammation were excluded clinically and by laboratory tests. MRI was performed using a 1.5 T magnet with spin echo (SE) techniques. All images were obtained using a head coil; slice thickness was 3 mm with a 0.5-mm gap and a 256 × 224 matrix was used. T1-weighted SE (400/14) images were obtained in sagittal and axial planes, with T2-weighted fast SE (FSE) (4000/100) images in the axial plane. T1-weighted SE (400/11) images with fat saturation before and after intravenous Gd-DTPA (0.1 mmol/kg body weight) were obtained in the coronal plane.

Case reports

Case 1

A 28-year-old white woman presented with a 1-week history of increasing pain in the right supraorbital region. As the pain became more severe the eyelid started to swell and redness of the sclera developed. The patient had no systemic symptoms and was on no medication other than an oral contraceptive pill. She had normal visual acuity, right upper lid oedema, 4 mm of right ptosis, 3 mm of right proptosis and diffuse inferior scleral injection; consistent with posterior scleritis there was no other clinical abnormality. Routine laboratory investigations were normal. Axial T1- and T2-weighted SE images were normal. Coronal T1-weighted fat-saturated images with Gd-DTPA showed enhancement of Tenon's capsule and the surrounding orbital fat (Fig. 1) confirming the diagnosis of periscleritis. She was commenced on prednisolone 20 mg and made a complete recovery over the next week.

Case 2

A 25-year-old white man presented with a 3-week history of progressive right orbital pain, ptosis and diplopia. He was otherwise well and on no medication. His visual acuity was 6/12 right and 6/4 left; he had reduced colour perception in the right eye, a complete right ptosis, 5 mm of right proptosis and severely restricted ocular movements on that side, consistent with IOI. Axial T1- and T2-weighted SE images showed proptosis with a slight increase in retrobulbar fat. Coronal T1-weighted images with fat saturation

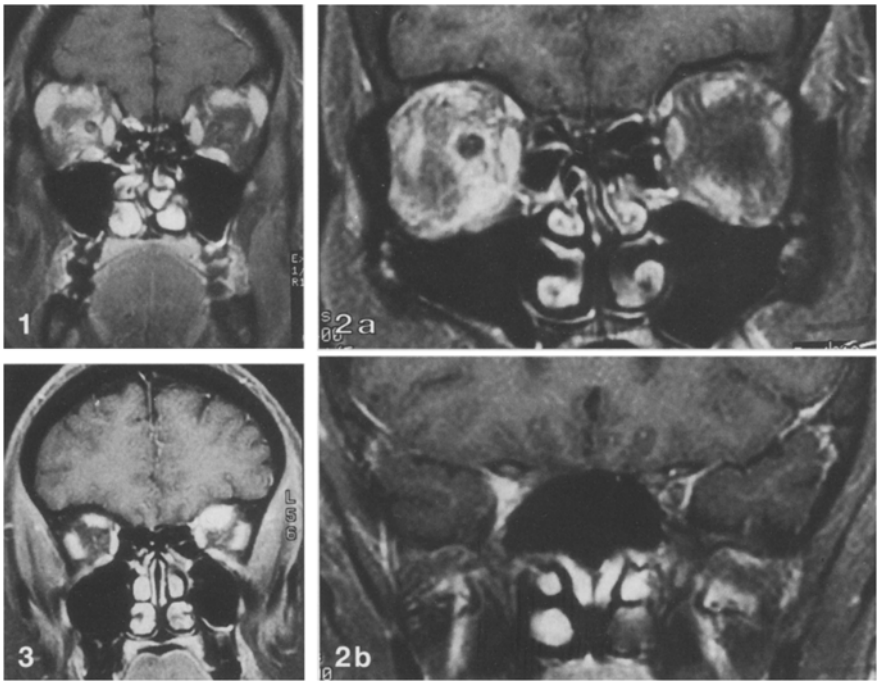

Fig. 1 Contrast-enhanced coronal SE (400/11) images with fat saturation. Enhancement of Tenon's capsule and the surrounding orbital fat

Fig. 2a, b As Fig. 1. **a** The right extraocular muscles are enlarged and show increased enhancement compared with normal enhancement of the muscles on the left. Enhancing tissue is seen throughout the retrobulbar fat. **b** Enhancement of the optic nerve sheath complex and the surrounding orbital fat at the orbital apex

Fig. 3 As Fig. 1. The left superior rectus muscle is enlarged, with enhancement of the contiguous orbital fat

and Gd-DTPA demonstrated increased contrast enhancement and slight enlargement of all extraocular muscles on the right, with enhancement throughout the retrobulbar fat (Fig. 2). At the orbital apex there was intense enhancement of the optic nerve sheath complex and the surrounding fat (Fig. 2). The patient was treated with 1 g intravenous methylprednisolone, followed by prednisolone 120 mg daily. There was a dramatic improvement in colour vision and the pain settled. However, although the visual acuity and ocular movements improved they had no returned to normal after 2 months on high-dose oral steroids. At this point it was decided to administer a course of radiotherapy to the posterior which orbit, led to resolution of the condition clinically and on repeat MRI.

Case 3

A 32-year-old white woman presented with a 2-week history of increasing discomfort around the left eye, pain on eye movement and swelling of the right upper lid for 1 day. She was otherwise well and was on no medication. A similar episode had affected the right eye 4 years previously, when a diagnosis of myositic pseudotumour was made on orbital CT. She had normal vision, an oedematous left upper lid, 3 mm of ptosis, reduced levator palpebrae superioris function, limited depression of the left eye, and diplopia on downward gaze; examination was otherwise normal. All laboratory investigations were normal apart from a sedimentation rate of 20 mm/h. Coronal T1-weighted MRI with fat saturation and Gd-DTPA showed marked enlargement and increased contrast enhancement of the left superior rectus/levator palpebrae superioris complex including its tendinous insertion, extending into the adjacent fat (Fig. 3). The patient was given prednisolone 30 mg daily and made a rapid and complete recovery.

Discussion

Idiopathic orbital inflammatory syndrome (IOIS) is an inflammatory condition of unknown cause characterised by a non-neoplastic, often predominantly lymphocytic tissue infiltrate [2–4]. The spectrum of orbital involvement varies from localised periscleritis or perineuritis to diffuse orbital inflammation [5], often resulting in an inflammatory mass, the "pseudotumour". There is usually dramatic improvement with steroid therapy and the final diagnosis is often based on steroid response, although patients with unusual features may require radiological investigation. Biopsy may be required in patients who do not respond to steroid therapy before irradiation or immunosuppressive therapy, although it is sometimes claimed that biopsy may aggravate the condition [4]. The CT appearances of the IOIS have been classified by a number of workers [6–8]. Nugent et al. [8] described five anatomical patterns based on CT images: anterior, posterior, diffuse, lacrimal and myositic pseudotumour, with lacrimal and myositic pseudotumour resolving most rapidly with steroid therapy. Intracranial extension has been described [9]. CT appearances are often nonspecific and IOI may appear identical to other entities including infiltrative malignancies such as lymphoma or metastasis [10, 11].

Surface-coil MRI of IOI may have greater specificity than CT [12]. While lesions were clearly demonstrated by both imaging techniques, the MR signal intensities of lesions relative to fat and muscle were found to distinguish between haemorrhage, metastatic breast carcino-

ma and pseudotumour, although in a retrospective analysis [13] other malignant orbital lesions such as lymphoma and myeloma had signal intensities identical to IOI. On conventional SE images the inflammatory mass gives signal less than that of fat and similar to that of muscle on T1-weighted images and is isointense or gives minimally higher signal than fat on T2-weighted images; subtle lesions can easily be masked by the high signal intensity of orbital fat [12].

Defining the orbital tissue involved by IOI with CT often requires contrast enhancement, but this can be difficult with conventional MRI sequences, as the enhancing lesion has a signal intensity similar to that of orbital fat on T1-weighted images. Fat-suppression techniques such as frequency-selective fat saturation (chem-sat), suppress the high signal from fat but not the high signal from contrast-enhancing lesions following Gd-DTPA, unlike the short tau inversion recovery (STIR) sequence, which eliminates fat signal, but also decreases the signal of the enhancing lesion since it has a similar null point to fat.

Chem-sat combined with Gd-DTPA improves anatomical detail and demonstrates lesions in the optic nerve [14] and its sheath [15] not seen on conventional SE images before and after contrast enhancement. In addition, by suppressing the fat signal, chemical shift artefact is eliminated and lesion contrast is increased by adjusting the dynamic grey scale of the image.

In our study the use of fat saturation and Gd-DTPA clearly demonstrated enhancing tissue involving the sclera, extraocular muscles, orbital fat and optic nerve sheath complex which would otherwise have been obscured by the high signal intensity of the fat. No uveoscleral thickening, a feature well described in orbital pseudotumour [1], was demonstrated in patient 1 on T1- or T2-weighted axial images. The diagnosis of periscleritis was confirmed on the fat-suppressed images, showing enhancement of Tenon's capsule and the surrounding orbital fat. Diffuse orbital involvement was confirmed in patient 2 by the dramatic contrast enhancement of all the orbital fat, the extraocular muscles and the optic nerve sheath complex. Previous CT studies have suggested that the intraorbital abnormalities, particularly within the orbital fat, in pseudotumour may be due to the inflammatory process or to oedema in the soft tissues, due possibly to occlusion of the superior ophthalmic vein or the cavernous sinus [15]. There was no evidence of cavernous sinus or venous occlusion in our patients and the superior ophthalmic vein was of normal size. Well believe therefore that the enhancing tissue within the retrobulbar fat is most likely to represent infiltration by pseudotumour rather than oedema secondary to venous congestion. The diagnosis of myositis in patient 3 was apparent on the conventional T1- and T2-weighted images, but involvement of the surrounding orbital fat was confirmed only on the fat-suppressed images, again reflecting the tendency for IOI to spill over into contiguous areas.

The normal extraocular muscles show intense contrast enhancement but skeletal muscle elsewhere in the body does not show this degree of enhancement on conventional or fat-suppressed images. This has been described by previous workers [14, 15], without postulating a reason. The extraocular and middle ear muscles are the only mammalian muscles to contain slow muscle fibres [17] which contain a higher density of mitochondria, myoglobin and oxidative enzymes, allowing prolonged contractions. These fibres have a higher density of surrounding capillaries [18] which do not possess a blood-tissue barrier. This may explain the intense enhancement of the extraocular muscles, which should not be mistaken as abnormal.

References

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ANNOUNCEMENTS

Society of Magnetic Resonance (3rd Scientific Meeting)
European Society for Magnetic Resonance in Medicine and Biology (12th Annual Meeting)
19–25 August 1995, Nice, France
Information: SMR Office, 2118 Milvia Street, Suite 201, Berkeley, CA 94704, USA. Tel. (510) 841-1899, Fax (510) 841-2340

Erasmus Course on Magnetic Resonance Imaging 1995
Central Nervous System II
9–14 September 1995, Antwerp, Belgium
Information: Prof. De Schepper, Department of Radiology, Univ. Ziekenhuis, Wilrijkstraat 10, B-2650 Edegem, Belgium, Fax 32-3-8252026.

Society of Pediatric Radiology (deutschsprachige Gesellschaft für Pädiatrische Radiologie)
32nd Annual Meeting and 15th Postgraduate Course
21–23 September 1995, Würzburg, Germany
Information: Dr. A. E. Horwitz, Kinderradiologie, Kinderklinik der Universität Würzburg, Josef-Schneider-Strasse 2, D-97080 Würzburg, Germany. Tel. (+49) 931-2013713, Fax (+49) 931-2012242.

Infections of the Nervous System and AIDS: Images
27–30 September 1995, Bari, Italy
Information: Dr. Cosma Andreula, Cattedra di Neuroradiologia, Universita Policlinico, I-70124 Bari, Italy. Tel. 39-547 85 69, Fax 39-805426994.

Cours de Scanographie Interventionelle
28–29 September 1995, Strasbourg, France
Information: Prof. J. L. Dietemann, Service de Radiologie B, Pavillon Clovis Vincent, Hopital Civil, F-6709 Strasbourg Cedex, France. Tel. 88161195, Fax 88161280.

1st International Neuroradiological Symposium of Pathology of the Nervous System in Infectious Diseases and AIDS
28–30 September 1995, Bari, Italy
Information: Cosma Andreula, Neuroradiologia, Università di Bari, c/o Policlinico, P.zza G. Cesare 11, I-70124 Bari, Italy. Tel. 39-80-547 33 30/522 68 94, Fax 39-80-522 68 94.

IXth Annual Meeting of the European Society of Head and Neck Radiology
11–14 October 1995, Groningen, The Netherlands
Information: Dr. G. H. M. Landman, Catharinahospital, P. O. Box 1350, NL-5602 ZA Eindhoven, The Netherlands. Tel. +31-40 39 85 30, Fax +31-40 39 85 67.

Neuroradiology – A Comprehensive Review
23–27 October 1995, Palm Beach, Florida, USA
Information: Ryals & Associates, P. O. Box 1925, Roswell, GA 30077-1925, USA. Tel. (404) 641-9773, Fax (404) 552-9859.

British Cervical Spine Society Meeting
10–11 November 1995, Southampton, UK
Information: Mr. Fausto Ianotti, Room LF738, Level F, South Academic Block, Southampton General Hospital, Tremona Road, Southampton SO16 6YD, UK. Tel. 01703-796617, Fax 01703-704236.

International Congress on Interventional Neuroradiology and Intravascular Neurosurgery
19–22 November 1995, Kyoto, Japan
(Joint Meeting of the 3rd Scientific Meeting of the World Federation of Interventional and Therapeutic Neuroradiology and the 11th Annual Meeting of the Japanese Society for Intravascular Surgery)
Information: Secretariat, International Congress on Interventional Neuroradiology and Intravascular Neurosurgery, Congress Corporation, Kinki Invention Center Building, 14 Yoshida Kawaharacho, Sakyo-ku, Kyoto 606, Japan. Tel. +81-75-752-0888, Fax +81-75-752-2963.

VIIIth European Pan-Arab Neurosurgical Course
9–11 December 1995, Riyadh, Saudi Arabia
Information: Dr. A. A. Al Khader, Department of Postgraduate and Academic Affairs, Riyadh Armed Forces Hospital, P.O.Box 7897, Riyadh 11159, Saudi Arabia. Fax 0966-1-478-4057.

Course de Tomodensitometrie Cranienne et Rachidienne
11-15 December, Strasbourg, France
Information: Prof. J. L. Dietemann, Service de Radiologie B, Pavillon Clovis Vincent, Hopital Civil, F-67091 Strasbourg Cedex, France. Tel. 88161195, Fax 88161280.

European Society of Magnetic Resonance in Neuropediatrics and Society of Magnetic Resonance Workshop: Quantitative MR in Neuropediatrics
15–16 December 1995, Oxford, UK
Information: Ernest B. Cady, President ESMRN, Department of Medical Physics and Bioengineering, University College London Hospitals, 11–20 Capper Street, London WC1E 6JA, UK. Tel. 44-171-380-9700 ext. 8448, Fax 44-171-380-9577, e-mail ecady@medphys.ucl.ac.uk.

MRI Update 1996:
Brain, Spine, Head, ENT and Pediatrics
18–23 February 1996, Rancho Mirage, California, USA
Information: Marti Carter, CME, Inc., 11011 W. North Avenue, Milwaukee, WI 53226, USA. Tel. (414) 771-9520.

2nd International Neuro-Interventional Procedures Course
3–5 March 1996, Riyadh, Saudi Arabia
Information: Department of Postgraduate and Academic Affairs, Riyadh Armed Forces Hospital, P. O. Box 7897, Riyadh 11159, Saudi Arabia, Fax 0966-1-478-4057.