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Computed Tomography of Orbital Myositis

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The computed tomographic (CT) scans of 11 consecutive patients with orbital myositis were reviewed to better characterize the CT appearance of this condition. The findings in this series differed from those of previous reports in several ways. Multiple muscle involvement predominated. Bilateral involvement was more frequent than previously reported. Enlargement of the tendon as well as the muscle was a frequent finding, but a normal tendinous insertion did not preclude the diagnosis of orbital myositis. Although the CT appearance of orbital myositis is often helpful, the findings are not pathognomonic; correlation with history, clinical findings, and therapeutic response must be considered in making the diagnosis.

Orbital myositis is a recognized subgroup of the nonspecific orbital inflammatory syndrome or orbital pseudotumor [1–3]. Early-generation computed tomography (CT) characterized orbital pseudotumor as a diffuse process; however, with improved resolution, specific target organs in the orbit have been identified [3]. For example, when the inflammatory process is localized to the lacrimal gland, sclera, nerve sheath, or extraocular muscle, a diagnosis of dacryoadenitis, periscleritis, perineuritis, or orbital myositis can be made.

Previous reports characterized orbital myositis as a predominantly unilateral inflammatory process causing irregular enlargement of a single isolated extraocular muscle and its tendinous insertion [3]. It was suggested that this appearance on CT could serve to distinguish orbital myositis from dysthyroid orbitopathy and other orbital conditions causing enlarged extraocular muscles. The present study was undertaken to better characterize the CT appearance of orbital myositis with high-resolution axial and coronal scans.

Materials and Methods

We reviewed the CT scans of 11 consecutive patients with a diagnosis of orbital myositis based on their history, clinical course, and response to steroids. Patients with diffuse orbital pseudotumor were excluded. Eight of the 11 studies were performed on a GE 8800 scanner. Three studies were performed on an AS & E 0500 scanner. All patients had axial and direct coronal scans with 5-mm sections. Contrast material was used in nine of the 11 studies. No patients had a history of thyroid dysfunction or signs or symptoms of dysthyroid orbitopathy. Laboratory evaluation (done in six patients) was negative for thyroid abnormalities.

Results

Twenty-nine muscles in 11 patients were identified as enlarged on CT: the medial rectus muscle(s) in eight patients, the lateral rectus in five, the superior rectus/levator palpebrae superioris complex in four, and the inferior rectus and superior oblique in two each. Five of 11 cases had bilateral involvement. Of the six with unilateral involvement, five had a single isolated enlarged extraocular muscle or muscle complex.

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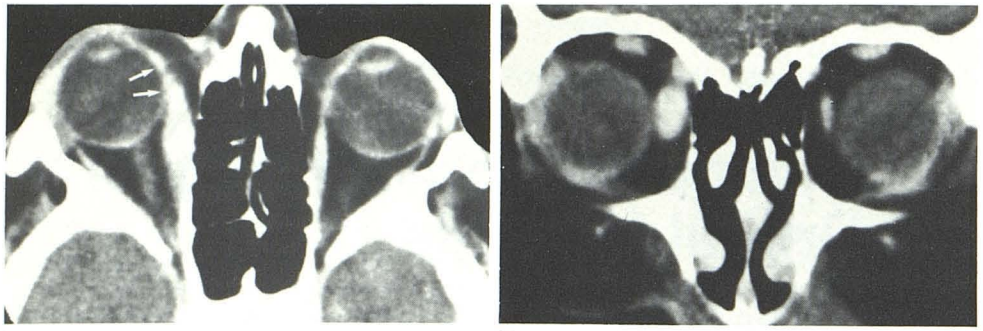


Fig. 1.—36-year-old woman with 1-year history of pain, swelling, redness, and decreased abduction of right eye. She responded to steroids initially, but had three recurrences. Contrast-enhanced axial (**A**) and coronal (**B**) CT scans. Right medial rectus muscle is enlarged and noticeably enhanced. Thickening and enhancement of muscle tendon to its insertion into globe wall (*arrows*). Coronal scan shows prominent enhancement and swelling of superior oblique as well as medial rectus muscle.

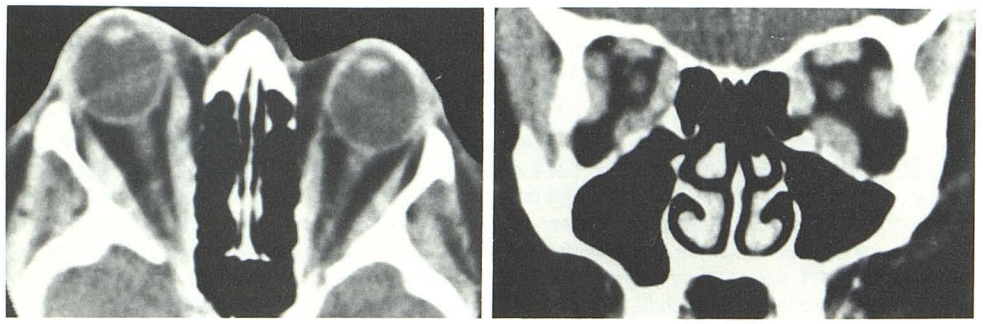


Fig. 2.—33-year-old woman with 2-week history of pain and proptosis of right eye with accompanying diplopia. She responded to steroids initially, but had several recurrences of myositis bilaterally. **A**, Contrast-enhanced axial CT scan at midorbital level. Right-sided proptosis. Medial and lateral rectus muscles are enlarged bilaterally and uniformly enhanced. Each muscle shows normal tapering to thin, nonenhancing muscle tendon (cf. fig. 1). **B**, Slightly oblique coronal scan. Enlargement of all rectus muscle groups except right superior rectus–levator complex.

Enhancement of the enlarged extraocular muscles was seen in all nine patients who had contrast-enhanced scans. Five patients showed thickening and enhancement of the muscle tendon (the region between the anterior muscle and its insertion onto the periphery of the globe) (fig. 1). In six patients the tendons were spared (fig. 2). No patients had scleritis or other involvement of the globe. One patient had asymmetric optic nerves with moderate enlargement on the side with myositis.

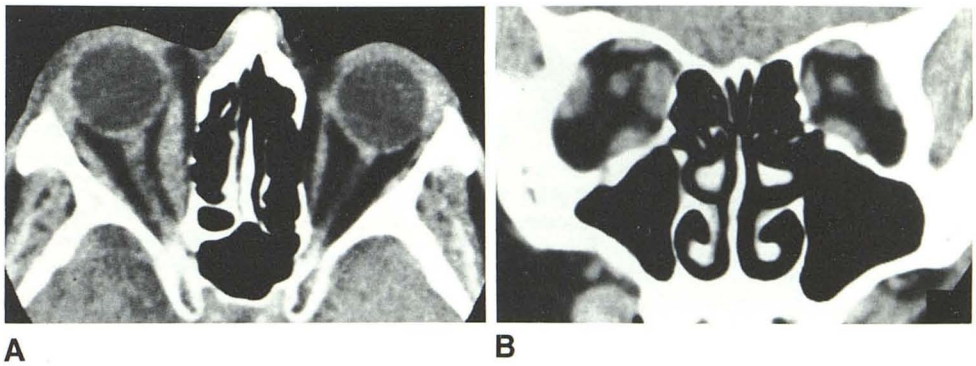
Discussion

Orbital myositis is a subgroup of the nonspecific orbital inflammatory syndrome or orbital pseudotumor. One or more of the extraocular muscles are affected by a diffuse infiltrate of inflammatory cells with well differentiated lymphocytes predominating [2, 4]. Its etiology remains obscure, although immunologic mechanisms have been postulated [1–3, 5]. There may be a higher incidence of orbital myositis in patients with known ocular and systemic autoimmune diseases [1]. Some cases of orbital myositis and orbital pseudotumor have been reported as occurring after upper respiratory infection [5–7]; this, too, may be related to immune-mediated mechanisms [7, 8].

Patients with orbital myositis characteristically present with acute orbital pain, diplopia, proptosis, eyelid swelling, blepharoptosis, and conjunctival injection and chemosis over the involved extraocular muscle. Alternatively, they may have chronic orbital pain or cephalgia. The diplopia is often elicited by extraocular movement in the direction of action of the affected muscle, unlike the restriction of gaze opposite the field of action of the affected muscle that is seen in dysthyroid orbitopathy [1]. With recurrent or chronic myositis the muscle(s) may become fibrotic and restricted in the opposite direction, thus mimicking the motility disturbance of dysthyroid orbitopathy [2].

Patients with orbital myositis are often quite responsive to systemic corticosteroid therapy, which usually provides relief of their symptoms within 48 hr of treatment. They usually receive 1–1.5 mg/kg of oral prednisone per day, the dose being decreased on an individual basis. The response to steroids may be complete with no residual sequelae or recurrences. However, many patients have recurrent signs and symptoms over a long period and need chronic steroid maintenance or radiation to the orbit to curb their inflammatory response [2]. With chronic and recurrent orbital myositis, patients may have residual proptosis or extraocular muscle dysfunction and fibrosis [1, 2].

The earliest reports of the CT appearance of orbital myositis characterized it as a typically unilateral process involving a single extraocular muscle [3, 9, 10]. In our series, multiple muscle involvement predominated, frequently with a bilateral distribution. The discrepancy between our results and those previously reported might be explained by our use of high-resolution scanners and routine coronal scans, which provide a more sensitive survey of the muscles. More recent reports by Slavin and Glaser [1] and Bullen and Young [2] confirm that orbital myositis can occur bilaterally. CT proved extremely



A B

Fig. 3.—59-year-old man with 10-day history of right orbital pain and diplopia and decreased abduction. He responded promptly to steroids. Axial (**A**) and coronal (**B**) CT scans. Enlargement of right medial and lateral rectus muscles. Optic nerve/sheath complex is thickened but is not compressed at orbital apex (muscle anulus). Etiology of this thickening is uncertain.

accurate in detecting bilateral disease in our series; in fact, two patients had bilateral involvement noted on CT that was not initially suspected clinically.

Trokel and Jakobiec [10] have stated that the typical CT finding in inflammatory myositis is enlargement of the extraocular muscle(s) extending anteriorly to involve the tendon inserting on the globe. We found this "tendon sign" in only five of 11 patients studied (fig. 1); the other six failed to demonstrate extension of the inflammatory process to the tendon on CT (fig. 2). Three of these six had bilateral involvement that could not be differentiated from dysthyroid orbitopathy solely on the basis of the CT appearance. Thus, whereas the presence of tendinous extension of the inflammatory process may be a good radiologic indicator of orbital myositis, its absence does not rule out myositis.

It has also been stated that the shape of the extraocular muscles in orbital myositis is more irregular than that seen in dysthyroid orbitopathy [3]. In our experience this was more the exception than the rule. When the enlargement of the extraocular muscle extended anteriorly, as in five patients in our series, the pattern was less fusiform than that seen in dysthyroid orbitopathy. However, in the absence of the tendon sign, no definite shape was noted to distinguish it from dysthyroid enlargement of the extraocular muscles.

Enhancement was seen in the involved muscles in all cases studied with contrast material. This finding was probably attributable to vascular congestion and inflammation in the muscles. The enhancement was indistinguishable in degree and character from that seen with dysthyroid orbitopathy or that seen with the enlarged extraocular muscles of a carotid–cavernous sinus fistula or a cavernous sinus–dural arteriovenous malformation.

Only one patient showed abnormalities of the optic nerve (fig. 3). Compression of the optic nerve in orbital myositis has been suggested in one report [2]. Usually, visual loss is unassociated with orbital myositis unless a concomitant diffuse anterior or posterior orbital pseudotumor is present [1, 11]. The aforementioned patient had no clinical evidence of visual loss, pupillary abnormalities, or optic nerve edema on funduscopy. We do not believe the optic nerve asymmetry noted on his CT scan was clinically significant.

The differential diagnosis of enlarged extraocular muscles includes dysthyroid orbitopathy, carotid-cavernous fistula, cavernous sinus–dural arteriovenous malformations, metastatic or infiltrative neoplasia, and the rare involvement seen in acromegaly [10, 12–15]. Any mass lesion or process at the orbital apex that obstructs venous return may also cause enlarged extraocular muscles.

Dysthryoid myopathy is the most common cause of enlarged extraocular muscles. Involvement is almost always bilateral. It is not uncommon for the involvement to be quite asymmetric [10]. The enlargement is fusiform and characteristically tapers near the tendinous insertion to the globe [3, 9, 10].

A strictly unilateral presentation or a presentation with single muscle involvement and tendinous extension may be more characteristic of orbital myositis. However, most cases in our series had bilateral symmetric findings that could not be differentiated from dysthyroid myopathy without clinical correlation. The distribution of involvement in myositis in our series revealed the medial rectus muscle(s) to be most commonly involved, followed by the lateral rectus, superior rectus–levator complex, and inferior rectus muscle(s). This contrasts with the more common involvement of the inferior followed by the medial, superior, and lateral rectus muscle(s) in dysthyroid myopathy [16].

Carotid-cavernous fistulas and cavernous sinus–dural arteriovenous malformations cause enlargement of the extraocular muscles by vascular congestion. These conditions usually are unilateral and show diffuse, uniform enlargement of the extraocular muscles in the orbit. The finding of an enlarged superior ophthalmic vein on high axial CT sections in association with fullness or dilatation of the ipsilateral cavernous sinus is highly suggestive of these conditions and should not lead to confusion with orbital myositis or dysthyroid orbitopathy [10, 15].

Tumor may directly invade or metastasize to the orbit and cause extraocular muscle enlargement [13, 14]. Often, however, the enlarged extraocular muscle is adjacent to an infiltrative orbital mass [10]. Divine and Anderson [14] describe a case with an enlarged extraocular muscle that mimicked orbital myositis. In this case and others [17], focal contrast enhancement, nodularity, and irregular enlargement of the extraocular muscles are more characteristic of a neoplasm and help distinguish this condition from orbital myositis or dysthyroid myopathy.

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