probably due to scleroderma. Postoperatively, the patient made an excellent recovery with a full range of pain-free movements in all digits of the right hand. At review 2 yr later, there was no evidence of recurrence. Symptoms in the left hand were not sufficient to warrant surgery.

Carpal tunnel triggering is rare and has not been reported in association with linear scleroderma. There is also no literature on the surgical management of tenosynovitis in this condition. We present a patient with a symmetrical linear scleroderma suffering with bilateral synovitis and triggering of the flexor tendons of the fingers at the entrance to the carpal tunnel. Having been resistant to medical therapy, this was successfully treated with flexor synovectomy and release of selected A1 pulleys.

The surgical treatment of the synovitis in patients suffering with scleroderma causes some concern in view of the risk of postoperative fibrosis resulting in worsening contractures and deformities.

This case demonstrates that good results may be achieved with surgical management and that prolonged medical treatment may be avoided.

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Systemic nocardiosis mimicking an ocular relapse of giant-cell arteritis

We describe a patient treated for temporal arteritis (TA) who experienced recurrent ocular events from different aetiologies, clearly showing that a high degree of suspicion against infection is permanently required in such patients. In addition it seems to be the first reported case of ocular and systemic nocardiosis in that context.

A 83-yr-old man was diagnosed with TA in September 2000, on account of new headaches, tender superficial temporal arteries, polymyalgia rheumatica, a C-reactive protein (CRP) level of 39 mg/l and granulomatous giant-cell arteritis on temporal artery biopsy. Prednisone 40 mg/day (0.5 mg per kg of body weight) led to prompt clinical improvement and CRP normalization. After 3 weeks, however, the left visual acuity suddenly decreased to 6/20 due to choroidal ischaemia attributed to TA (Fig. 1A,B), and progressively returned to 20/20 after four pulses of methylprednisolone 250 mg/day followed by oral prednisone 80 mg/day. At the beginning of May 2001, on prednisone 25 mg/day with the CRP level being 34 mg/l, a painless visual loss of the right eye occurred, leading to a vision of ‘counting fingers at 50 centimeters’. Retinal angiography showed a delayed retinal artery filling time of 35 s and marked localized subretinal oedema without choroidal defect or papillary oedema (Fig. 1C,D). The patient had no signs or symptoms of infection and a negative work-up including chest radiography, echocardiography and microbiological analysis of urine and blood, and we concluded there was TA progression involving the right central retinal artery. A 3-day course of 120 mg/day pulsed methylprednisolone was started on 14 May. During the following days, the CRP level increased rapidly to 240 mg/l and no visual improvement occurred. At the end of May, a pustular lesion was noticed on the patient’s abdomen, and the patient became dyspnoeic with multiple nodular and reticular shadows appearing on chest X-ray. He died on 4 June from septic shock and multi-organ failure. Autopsy showed multiple abscesses varying in size from 1–2 mm to 4 cm in the heart, lungs, pancreas, peritoneum, kidneys, thyroid and in the whole brain (cerebral hemispheres and brain stem, oculomotor nerves and choroid plexus, associated with a purulent meningitis). An accumulation of 1 μm-wide filamentous gram-positive bacilli, Grocott-positive and Ziehl-positive, randomly oriented and

FIG. 2. Intra-operative photograph demonstrating the palmar tenosynovectomy.
branching at right angles was found in the abscesses and meninges, suggesting *Nocardia asteroides* infection. The walls of several cerebellar and coronary arteries were totally destroyed by invasive nocardial infection. Arterial segments taken from the internal carotids and first centimetres of the ophthalmic arteries were normal. A fibrotic lesion on the right vertebral artery, with disappearance of the internal elastic lamina, suggested a sequela of cured TA. Unfortunately, the eyes and retro-orbital content could not be examined due to lack of replacement ocular prostheses at the time of autopsy. We concluded that death occurred from fulminant systemic nocardiosis in an immunocompromised host, that was first manifested by an ocular event mimicking a TA relapse, but *a posteriori* related to a nocardial choroidal abscess.

This observation dramatically illustrates the physician’s dilemma between the fear of ocular involvement and the risk of corticosteroid complications in the treatment of temporal arteritis. Ocular manifestations occurred twice, 3 weeks and 6 months after start of corticosteroids, which theoretically reduces the ocular risk of TA to around 1% after 1 week of treatment [1]. The first event, isolated choroidal ischaemia, is an unusual finding in TA because choroidal ischaemia is generally associated in this disease with anterior ischaemic optic neuropathy or central retinal artery occlusion [2]. On the other hand, it may constitute a reversible cause of visual loss in TA [3], as in our patient. The rate of primary corticoresistance in TA is quite high: 13.5% in a prospective trial of 164 patients, and only apparent clinically (normal CRP) in half the cases [4]. Thus, in the absence of an alternative explanation, primary TA corticoresistance appeared to us to be the most plausible cause of the choroidal ischaemic event. That it was the first ocular manifestation of nocardiosis is indeed very unlikely, due to the 6-month interval before the next event without any antibiotic treatment. The second ocular manifestation was certainly related to a nocardial choroidal abscess, based on a compatible angiographic appearance, and widespread intracranial abscesses together with a lack of any active TA lesion at autopsy.

Nocardiosis occurs in immunocompromised hosts and can involve the central nervous system and the eyes by haematogenous spreading from a lung infection, leading to endogenous endophthalmitis or retinal abscesses [5–7]. To our knowledge, only two cases of nocardial infection without ocular localization have been described in patients treated for TA [8, 9]. In this patient, the sequence of a first TA-related then a second infection-related ocular complication highlights both the difficulty of diagnosing opportunistic infections in rheumatological immunocompromised patients [10], and the fact that suspicion of infection should grow in parallel with the cumulative dose of corticosteroids administered.

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<td>Isolated ocular manifestation in treated temporal arteritis may reveal infection.</td>
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FIG. 1. The fundus of the left eye appears normal (A), but fluorescein angiography reveals ischaemic choroidopathy (B). The fundus of the right eye showing a rounded subretinal oedematous lesion (C). On fluorescein angiography, retinal arterial filling was delayed (arm-retina time of more than 30 s), and the subretinal lesion was non-vascularized (D), with late leakage of dye.
An unusual cause of acute rhabdomyolysis

Sir, Last February, a previously healthy 27-year-old male was admitted to our hospital with a 2-day history of afebrile abrupt onset malaise and myalgias involving arms, legs and trunk. Because of simultaneous cephalalgias and rhinorhoea, the primary care physician had suspected influenza and immediately prescribed oseltamivir, a neuraminidase inhibitor. The patient had massive tissue oedema and a rapidly progressive paresis of the proximal and distal muscles of all extremities. His heart rate was 132/minute, his blood pressure 130/90. The ESR was 12 in the first hour. His serum creatine kinase (CK) levels were 125 860 U/l (CK-MB1080 U/l), troponin T was negative, the pro-brain derived natriuretic peptide (proBNP) was 1492 µg/l (normal <125). His leucocyte count was 17800 µl (97% neutrophils), serum CRP was 105 mg/l, creatinine 13 mg/l, ALT 764 U/l and urate 105 mg/l. There was hypocalcaemia (1.6 mmol/l) and hyperphosphataemia (79 mg/l). The patient voided dark brown urine (200 ml within the first 12 h). The urine peroxidase reaction was positive and in the absence of erythrocytes indicative of myoglobin.

The patient denied recent trauma, drug use and a family history of muscle disease. Chest X-ray, ECG and echocardiogram were normal. MRI and 99mTc pyrophosphate scintigraphy revealed a disseminated muscle oedema (Fig. 1A). A muscle biopsy performed at day 2 of admission from an involved region (M. vastus lateralis) showed a regular checkerboard distribution of type I and type II fibres, intact enzyme activities and normal PAS and oil red stains. Single fibre necrosis was observed very rarely, myophagocytosis and inflammatory infiltrations were completely absent. Electron microscopy showed activated satellite cells, an intact myofibrillar lattice and disrupted sarcoplasmic membranes (Fig. 1B).

Serum carnitine and acetyl carnitine, carnitine palmitoyl transferase and ANA were within normal limits [1]. Legionella pneumophila antigen was negative in the urine, as were serum antibodies for HIV, paramyxovirus (adeno-), influenza A, cytomegalovirus, Epstein–Barr and herpes simplex viruses.

Blood drawn at admission yielded a positive ELISA result for influenza B virus IgG (22.9 U; cutoff at 9.0 U), a repeat serum after 15 days showed an almost 8-fold rise (174.0 U). RT-PCR for influenza B virus RNA from the muscle biopsy specimen was negative.

Twelve hours after admission, the patient was unable to lift arms and legs from the bed. The CK had doubled (265300 U/l). Despite instantaneous urine alkalinization, volume substitution (91 within the first 24 h) and treatment with recombinant urate oxidase, there was renal failure requiring haemodialysis. Plasmapheresis was performed on three consecutive days. After 12 days, the myalgias diminished, muscle strength began to improve and the CK had declined (5700 U/l). At the same time...