

ing of the biology of phenotypic expression in HD. Since we can use the HD gene test for the differential diagnosis of hyperkinetic movement disorders, we will probably find many more clinical manifestations and atypical expressions of HD, as described in this family with an asymptomatic carrier of the HD gene.

References

- Alexander GE, De Long MS, Strick PL (1986) Parallel organisation of functionally segregated circuits linking basal ganglia and cortex. *Annu Rev Neurosci* 9: 357–381
- Andrew SE, Goldberg YP, Kremer B, et al (1993) The relationship between trinucleotide (CAG) repeat length and clinical features of Huntington's disease. *Nat Genet* 4: 398–403
- Boel, M., P. Casar (1984) Paroxysmal kinesigenic choreoathetosis. *Neuropediatrics* 15: 215–217
- Claes S, van Zand K, Legius E, Dom R, Malfroid M, Baro F, Godderis J, Cassiman JJ (1995) Correlations between triplet repeat expansion and clinical features in Huntington's Disease. *Arch Neurol* 52: 749–753
- Demirkiran M, Jankovic J (1995) Paroxysmal dyskinesias: clinical features and classification. *Ann Neurol* 38: 571–579
- Goldberg YP, Andrew SE, Clarke LA, Hayden MR (1993) A PCR method for accurate assessment of trinucleotide repeat expansion in Huntington's disease. *Hum Mol Genet* 2: 635–636
- Goodenough DJ, Faviello RG, Annis BL, et al (1978) Familial and acquired paroxysmal dyskinesias: a proposed classification with delineation of clinical features. *Arch Neurol* 35: 827–831
- Gusella JF, Wexler NS, Conneally PM, et al (1983) A polymorphic DNA marker genetically linked to Huntington's disease. *Nature* 306: 234–238
- Jankovic J, Ashizawa T (1995) Tourettism associated with Huntington's disease. *Mov Disord* 10: 103–105
- Kinast M, Erenberg G, Rothner AD (1980) Paroxysmal choreoathetosis: report of five cases and review of the literature. *Pediatrics* 65: 74–77
- Kokmen E, Smith GE, Petersen RC, Tangalos EG, Ivnik RJ (1991) The short test of mental status: correlations with standardized psychometric testing. *Arch Neurol* 48: 725–728
- Kurlan R, Behr J, Medved L, Shoulson I (1987) Familial paroxysmal dystonic choreoathetosis: a family study. *Mov Disord* 2: 187–192
- Lance JW (1977) Familial paroxysmal dystonic choreoathetosis and its differentiation from related syndromes. *Ann Neurol* 2: 285–293
- Loong SC, Ong YY (1973) Paroxysmal kinesigenic choreoathetosis: Report of a case relieved by L-dopa. *J Neurol Neurosurg Psychiatry* 36: 921–924
- Martin JB, Gusella JF (1986) Huntington's disease. Pathogenesis and management (review). *N Engl J Med* 315: 1267–1276
- Parent A, Hazrati LN (1995) Functional anatomy of the basal ganglia. I. The cortico-basal ganglia-thalamo-cortical loop. *Brain Res Rev* 20: 91–127
- Penney JB, Young AB (1993) Huntington's disease. In: Jankovic J, Tolosa E (eds) *Parkinson's disease and movement disorders*. Williams and Wilkins, Baltimore, pp 205–216
- Snell RG, MacMillan JC, Cheadle JP, et al (1993) Relationship between trinucleotide repeat expansion and phenotypic variation in Huntington's disease. *Nat Genet* 4: 393–397
- Stevens H (1966) Paroxysmal choreoathetosis: a form of reflex epilepsy. *Arch Neurol* 14: 415–420
- The Huntington's Disease Collaborative Research Group (1993) A novel gene containing a trinucleotide repeat that is expanded and unstable on Huntington's disease chromosomes. *Cell* 72: 971–983
- Turjanski N, Weeks R, Dolan R, Harding AE, Brooks DJ (1995) Striatal d1 and d2 receptor binding in patients with Huntington's disease and other choreas. *Brain* 118: 689–696
- Vogel CM, Drury I, Terry LC, Young AB (1991) Myoclonus in adult Huntington's disease. *Ann Neurol* 29: 213–215
- Warner J, Barron L, Brock D (1993) A new polymerase chain reaction (PCR) assay for the trinucleotide repeat that is unstable and expanded in Huntington's disease chromosomes. *Mol Cell Probes* 7: 235–239
- Whitty CWM, Lishman WA, Fitts-Gibbon JP (1964) Seizures induced by movements. A form of reflex epilepsy. *Lancet* 1: 1403–1406
- Scheidtmann¹ · C. Trenkwalder Max-Planck-Institute of Psychiatry, Clinical Institute, Neurology, Kraepelinstrasse 10, D-80804 Munich, Germany
- Schwarz · T. Gasser Department of Neurology, Ludwig-Maximilians-University, Munich, Germany
- Holinski Department of Pediatric Genetics of the Kinderpoliklinik, Ludwig-Maximilians-University, Munich, Germany

Present address:

¹ Neurologische Klinik Bad Aibling, Kolbermoorer Strasse 72, D-83043 Bad Aibling, Germany

**Margitta T. Kampman
Eva A. Jacobsen**

Pyomyositis and osteomyelitis in a patient with radiating pain in the leg

Received: 20 January 1997

Received in revised form:

25 February 1997

Accepted: 6 March 1997

Sirs: Pyomyositis – or abscess within muscle – occurs commonly in tropical climates. The first case occurring in temperate areas was described in 1971 [5]. Since then, “non-tropical” pyomyositis has been increasingly recognized. From 1971 to 1984, 31 cases were reported in the United States [3], and a total of 98 cases had been reported in North America up to 1991 [2]. The true incidence of pyomyositis in temperate climates is unknown. We would like to draw attention to an uncommon, though relevant differential diagnosis in pa-

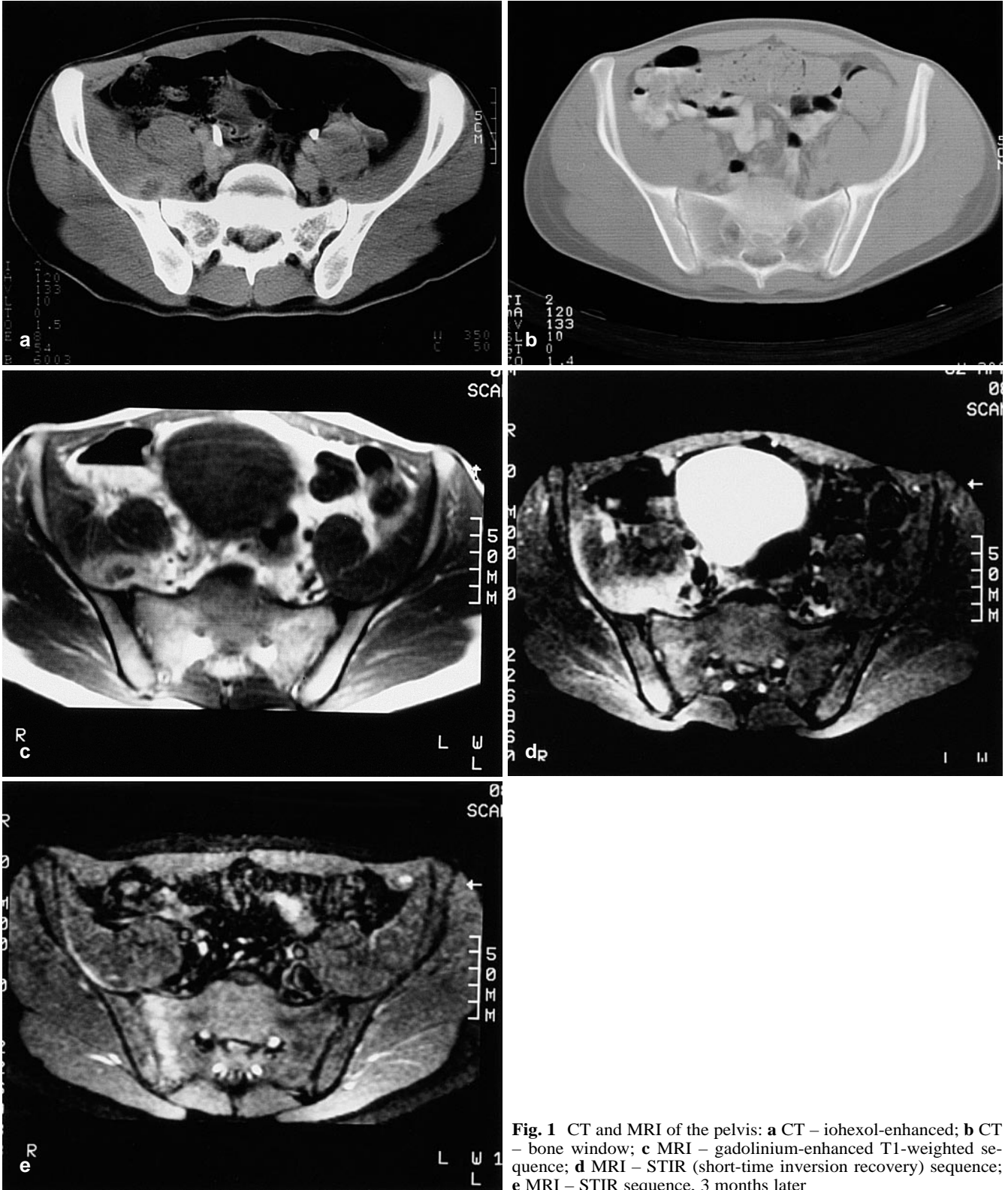


Fig. 1 CT and MRI of the pelvis: **a** CT – iohexol-enhanced; **b** CT – bone window; **c** MRI – gadolinium-enhanced T1-weighted sequence; **d** MRI – STIR (short-time inversion recovery) sequence; **e** MRI – STIR sequence, 3 months later

tients with radiating pain in the leg and fever.

The patient was a 28-year-old accountant who presented to our emergency department with a 7-day history of lumbar pain radiating to his right leg. After the symptoms started, he had not been able to place weight on his right leg, and he had been confined to a low chair for 5 days. He had had fever for 4 days before admission, with the highest measured temperature being 40.2°C. His previous medical history was unremarkable. The patient denied homosexuality, promiscuity, or intravenous drug abuse. There was no history of recent illness or travel. On physical examination he appeared to be well nourished but in severe pain. His body temperature was 39.0°C. Findings of the physical examination were significant only for a healed laceration less than 1 cm in diameter over the left tibial tuberosity. He had sustained sores on both knees and both elbows when playing soccer on artificial turf 16 days prior to admission. Neurological examination was limited owing to severe pain provoked by any movement, even after administration of morphine. The patient was examined lying in bed in a prone and supine position. Tenderness in the lower back was maximal in the right paravertebral region. Any attempt at straight leg raising provoked excruciating pain. Muscle strength testing proximal to the ankle was virtually impossible. In the right lower extremity the only reliable finding was a diminished patellar tendon reflex. There was no evidence of distal paresis. Sensibility was intact. No other neurological impairment was found.

The leukocyte count was $7.7 \times 10^9/l$ with a left shift (78.2% neutrophils). C-reactive protein (CRP) was increased to 315 mg/l. An HIV antibody test was negative, and there was a normal CD4/CD8 ratio. Chest radiography, lumbar radiculography and CT of the lumbar spine were normal. CT of the pelvis showed an increased volume of the right iliacus

and psoas muscles. Injection of iohexol disclosed a ring enhancement in the iliacus muscle (Fig. 1a). CT with bone window did not reveal any bone involvement (Fig. 1b). Magnetic resonance imaging (MRI) was performed with axial T1-weighted sequences without and with gadolinium, axial and coronal T2-weighted sequences, and axial short time recovery (STIR) sequence (0.5 T Philips Gyroscan). Contrast enhancement was seen in the muscle lesions (Fig. 1c). Oedema in muscles and bone marrow was shown on STIR sequences (Fig. 1d) and was still seen in the bone marrow 3 months later (Fig. 1e).

Blood for cultures was obtained, and empirical intravenous therapy with cloxacillin, gentamicin and netilmicin was started 4 h after admission. After *Staphylococcus aureus* had been isolated from six blood cultures, antibiotic treatment was changed to dicloxacillin and fusidic acid intravenously for 14 days. Oral treatment with dicloxacillin was continued for 4 weeks.

Christin [2] and Skoutelis [6] have reviewed the literature, listing 98 and 76 cases of pyomyositis, respectively. The thigh is most often affected, but deep pelvic infections involving the psoas, iliacus, piriformis, and obturator internus are not uncommon [2].

S. aureus is the most frequently identified pathogen, causing about 70% of cases in temperate climates. Bacteraemia was reported for 31% of North American patients. Figures from tropical areas have been considerably lower [2]. As normal skeletal muscle has a high intrinsic resistance to bacterial infection, it has been suggested that underlying muscle damage may facilitate the onset of pyomyositis [2]. In reports from non-tropical regions, diabetes mellitus and AIDS are frequently mentioned and probably predispose to development of pyomyositis. In a review of 76 cases, 14 patients were reported to have diabetes, and 9 patients were infected with HIV [6]. The condition is also often diagnosed

in addicted, immunocompromised or burned patients [1].

Our patient had an abscess and muscle swelling causing radiating pain in the leg, probably by compression and/or irritation of the right femoral nerve passing between the iliacus and the major psoas. The abscess was diagnosed by CT, but the accompanying osteomyelitis was only seen on MRI.

We suspect that staphylococcal infection of the patient's burn-like skin abrasions had developed into septicaemia. We have not been able to identify any predisposing factors. At follow-up 5 weeks after admission, the patient had recovered completely from the symptoms related to the abscess, and CRP had normalized. However, it will take months for the oedematous changes on MRI to resolve.

Radiating pain in the leg can arise from mass lesions in deep pelvic muscles. Pyomyositis should be considered in the differential diagnosis in patients with fever. Imaging of the pelvis by CT, and/or MRI will be helpful in establishing the diagnosis [4], while routine laboratory studies may be normal. Early cases can be managed with antibiotics alone, but surgical drainage is often necessary. Though recovery without sequelae is the rule, delayed diagnosis is associated with septic cardiorespiratory complications, leading to death in up to 10% of patients [2].

Increased awareness of pyomyositis will improve management and outcome. We advocate the use of MRI in cases where the abscess is located adjacent to bone. Isotope scans may be performed if MRI is not available. However, MRI is more sensitive and may detect lesions earlier. In addition, MRI is a superior imaging modality for showing the anatomical location. If concomitant osteomyelitis is found, antibiotic treatment has to be adjusted accordingly.

References

1. Banker BQ (1994) Other inflammatory myopathies. In: Engel AG, Franzini-Armstrong C (eds) *Myology – basic and clinical*. McGraw-Hill, New York, pp 1475–1476
2. Christin L, Sarosi GA (1992) Pyomyositis in North America: case reports and review. *Clin Infect Dis* 15: 668–677
3. Gibson RK, Rosenthal SJ, Lukert BP (1984) Pyomyositis: increasing recognition in temperate climates. *Am J Med* 77: 768–772
4. Gordon BA, Martinez S, Collins AJ (1995) Pyomyositis: characteristics at CT and MR imaging. *Radiology* 197: 279–286
5. Levin MJ, Gardner P, Waldvogel FA (1971) “Tropical” pyomyositis. An unusual infection due to *Staphylococcus aureus*. *N Engl J Med* 284: 196–198
6. Skoutelis A, Andonopoulos A, Panagiotopoulos E, Bassaris H (1993) Non-tropical pyomyositis in adults: report of four cases and literature review. *Eur J Clin Microbiol Infect Dis* 12: 769–772

M. T. Kampman (✉)
Department of Neurology,
Tromsø University Hospital,
N-9038 Tromsø, Norway
Tel.: +47 776 267070,
Fax: +47 776 267074,
e-mail: margitta@petrus.fagmed.uit.no

E. A. Jacobsen
Department of Radiology,
Tromsø University Hospital,
N-9038 Tromsø, Norway