Intra-articular rheumatoid nodules and triggering of the knee joint

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Abstract

Rheumatoid nodules are a common extra-articular manifestation in rheumatoid arthritis. Intra-articular localisation of these nodules is rare and may produce clinical symptoms. Seven patients with walking problems due to an intra-articular rheumatoid nodule, which became entrapped on the ridge of the tibial plateau of the knee joint resulting in a phenomenon referred to as trigger knee, are described. After excision of the nodules all symptoms completely disappeared.

Rheumatoid nodules are characteristic extra-articular manifestations of rheumatoid arthritis. They are mainly found in the presence of rheumatoid factor¹ and may be indicative of more severe disease.² These nodules occur in 20-35% of patients with rheumatoid arthritis and they are commonly found subcutaneously over pressure points—for example, at the elbows, the buttocks, and the scalp.¹ They can also develop in several internal organs, such as the lungs, kidneys, and heart.³⁻⁴ Intra-articular rheumatoid nodules, however, are rare.⁵⁻⁹ In this retrospective study we considered seven patients with walking problems associated with intra-articular rheumatoid nodules of the knee joint.

Patients and methods

From 1972 to 1989 six patients who visited our outpatient rheumatology clinic had seropositive rheumatoid arthritis and symptoms typical of intra-articular rheumatoid nodules of the knee joint. We also performed a computer search for patients who had had an operation for triggering of the knee joint caused by rheumatoid nodules and another patient was identified. We retrospectively studied the clinical features associated with the presence of intra-articular rheumatoid nodules in these seven patients.

Clinical data were collected from medical files. Preoperative radiographs of the affected knees were compared with those of the unaffected knees using the score system described by Larsen *et al.*¹⁰ Operative data and post-operative course were reviewed.

The diagnosis of rheumatoid nodule was based on history and physical examination and was confirmed by histological examination of the excised lumps. Central fibrinoid necrosis surrounded by palisading histiocytic cells was considered diagnostic of rheumatoid nodules (figs 1 and 2).

Figure 1 Rheumatoid nodule showing central fibrinoid necrosis. The boxed area is shown in detail in fig 2.

Figure 2 Detail of fig 1 showing typical palisades of histiocytic cells surrounding the necrotic centre.

Results

Six women and one man were identified with symptoms during walking caused by intra-articular rheumatoid nodules. All patients had seropositive rheumatoid arthritis. The mean disease duration was 11.9 years (range 3-33 years, SD=10.7). The mean age of the patients was 53 years (range 39-69 years).

All patients had a history of recurrent pain accompanied by clicking, locking, and giving way of the affected knee joint. In three patients this triggering of the knee joint occasionally caused stumbling and falling. It was noted in one patient that in the presence of a gross joint effusion the triggering and pain disappeared. It recurred soon after removal of the synovial fluid, or after the administration of an intra-articular injection of corticosteroids.

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On physical examination a moderate synovitis with slight effusion was found in four patients and a firm lump was palpable in all seven. The lump appeared to be attached to the capsule. In six patients the lump was situated on the lateral side and in one patient on the medial side of the patella. On flexing the knee, the lump slipped over the underlying tibial plateau and hid behind the patella. Sometimes it was caught on the tibial plateau and caused triggering of the knee joint. This could be perceived as a palpable 'click'.

Anteroposterior and lateral radiographs did not show abnormalities that could account for the presence of intra-articular rheumatoid nodules. Only slight narrowing of the joint space was seen in five patients (Larsen score 0–1). Erosions were absent. In two patients the radiographs were normal. Affected and unaffected knees could not be distinguished radiologically.

All the lumps were excised within two years after the first symptoms. Their dimensions varied from $1 \times 1 \times 1$ cm to $3 \times 2.5 \times 1.5$ cm. The localisation appeared to be intracapsular—that is, within the fibrous capsule of the knee joint.

In six of the seven patients the diagnosis of rheumatoid nodule was confirmed by histological findings. In one patient insufficient material was obtained for histological examination. The synovium showed a mild inflammatory reaction in all patients.

The postoperative course was uneventful in all but one patient. In this patient haemarthrosis developed within two weeks during anticoagulant prophylaxis. In all patients triggering of the knee disappeared after removal of the rheumatoid nodules. Two patients had a symptomatic recurrence of the rheumatoid nodule within one and two years. Two to seven years after excision the others were still asymptomatic.

Discussion

Walking problems are a common clinical symptom in patients with rheumatoid arthritis. Usually they are due to arthritis, instability of the lower limb joints, or loss of muscle strength. Rare causes of walking problems in rheumatoid arthritis include normal pressure hydrocephalus¹¹ and myelopathy.¹²

We report here intra-articular rheumatoid nodules of the knee joint as another cause of walking problems in patients with seropositive rheumatoid arthritis. Entrapment of an intra-articular rheumatoid nodule on the ridge of the tibial plateau has an easily recognisable clinical pattern consisting of clicking, locking, and giving way of the knee joint. This clinical pattern is comparable with trigger finger or trigger wrist.⁸ Thus 'trigger knee' seems to be the most suitable name for this phenomenon. Chamberlain⁵ reported another clinical feature of intra-articular rheumatoid nodules, namely the lack of full extension of the knee, resulting in walking problems. In three of our patients triggering of the knee joint resulted in stumbling

and falling. To our knowledge, such severe walking problems have not yet been reported in association with intra-articular rheumatoid nodules.

The differential diagnosis of triggering of the knee joint also includes intra-articular loose bodies, meniscal tears and cysts, ganglions, and patellar osteophytes.

In our patients the presumed diagnosis of intra-articular rheumatoid nodules of the knee joint was easily made solely by history and physical examination. Ultrasonography is possibly useful in distinguishing intra-articular rheumatoid nodules in several joints.^{13–16} As a result of the localisation of the rheumatoid nodules within the fibrosis capsule, we conclude that arthroscopy will be superfluous in most patients with intra-articular rheumatoid nodules of the knee joint.

In our series of patients anteroposterior and lateral radiographs of the affected knees showed surprisingly little damage and had a similar Larsen score to those of the unaffected knees, as shown by other workers.^{5–7}

In conclusion, intra-articular rheumatoid nodules can cause serious walking problems in patients with seropositive rheumatoid arthritis due to triggering of the knee joint, as a result of entrapment of the nodule on the ridge of the tibial plateau. This phenomenon is probably rare: only seven patients with trigger knee caused by intra-articular rheumatoid nodules were identified during 17 years in our rheumatology outpatient clinic. As most of the intra-articular rheumatoid nodules will probably remain symptomless, it is difficult to predict how often they really occur. In accordance with the other workers,^{5–7} most of the nodules were located on the lateral side of the patella (86% of patients) and only one (14%) medially. The diagnosis can easily be made on the basis of the typical clinical manifestations and confirmed by histological examination. No additional investigations are required. Complete relief of the symptoms can be obtained by excision of the nodule.

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- 1 Harris E D. The clinical features of rheumatoid arthritis. In: Kelley W N, Harris E D, Ruddy S, Sledge C B, eds. *Textbook of rheumatology*. 3rd ed. Philadelphia: Saunders, 1989: 965–6.
- 2 van der Heyde D M F M, van Riel P L C M, van Rijswijk M H, van der Putte L B A. Influence of prognostic features on the outcome in rheumatoid arthritis: a review of the literature. *Semin Arthritis Rheum* 1988; 17: 284–92.
- 3 Schumacher J R. *Primer on the rheumatic diseases*. 9th ed. Atlanta, GA: Arthritis Foundation, 1988: 90–2.
- 4 Krane S M, Simon L S. Rheumatoid arthritis: clinical features and pathogenetic mechanisms. *Med Clin North Am* 1986; 70: 263–84.
- 5 Chamberlain M A. Intra-articular rheumatoid nodules of the knee. *J Bone Joint Surg [Br]* 1971; 53: 507–9.
- 6 Kampner S L, Kuzell W. Intra-articular rheumatoid nodule of the knee joint. *Clin Orthop* 1976; 114: 243–6.
- 7 Huang T L, Fossier C, Ray R D, Ghosh L. Intra-articular rheumatoid nodule of the knee joint associated with recurrent subluxation of the patella. *J Bone Joint Surg [Am]* 1979; 61: 438–40.
- 8 Carvell J E, Mowat A G, Fuller D J. Trigger wrist phenomenon in rheumatoid arthritis. *Hand* 1983; 15: 77–81.
- 9 Ishikawa H, Ueba Y, Hirohata K. An intra-articular rheumatoid nodule of the hip. *J Bone Joint Surg [Am]* 1988; 70: 775–7.

- 10 Larsen A, Dale K, Eek M. Radiographic evaluation of rheumatoid arthritis and related conditions by standard reference films. *Acta Radiol [Diagn]* 1977; **18**: 481–91.
- 11 Rasker J J, Jansen E N H, Haan J, Oostrom J. Normal-pressure hydrocephalus in rheumatic patients. *N Engl J Med* 1985; **312**: 1239–41.
- 12 van der Horst-Bruinsma I E, Markusse H M, Macfarlane J D, Vielvoye C J. Rheumatoid discitis with cord compression at the thoracic level. *Br J Rheumatol* 1990; **29**: 65–8.
- 13 Sattler H, Schmidt K L. Zum Stellenwert der Arthrosonographie in der rheumatologischen Diagnostik: Untersuchungstechnik, Befunde und ihre Interpretation. *Z Rheumatol* 1986; **45**: 1–6.
- 14 De Flaviis L, Scaglione P, Nessi R, Ventura R, Calori G. Ultrasonography of the hand in rheumatoid arthritis. *Acta Radiol* 1988; **29**: 457–60.
- 15 Koski J M. Axillar ultrasound of the glenohumeral joint. *J Rheumatol* 1989; **16**: 664–7.
- 16 Goldenstein C, McCauley R, Troy M, Schaller J G, Szer I S. Ultrasonography in the evaluation of wrist swelling in children. *J Rheumatol* 1989; **16**: 1079–87.

PostScript

MATTERS ARISING

Treatment of resistant giant cell arteritis with etanercept

Tan *et al* recently described a case of "resistant giant cell arteritis" successfully treated with etanercept.¹ Their patient, who had classical symptoms of polymyalgia rheumatica (PMR), developed headaches while receiving low dose steroids. Biopsy of a temporal artery showed no arteritis. Because giant cell arteritis (GCA) was suspected on clinical grounds, high dose steroids were instituted. Six months later, despite continued steroid treatment, a transient ischaemic attack (TIA) involving right arm weakness occurred, which was ascribed to "arteritis (sic)". An insufficiency fracture ensued. As the erythrocyte sedimentation rate and C reactive protein were persistently raised, a diagnosis of GCA resistant to treatment was made, and etanercept was given. The acute phase reactants normalised, and the symptoms referable to PMR resolved completely.

I am not persuaded that the patient in question had GCA.

Firstly, the temporal artery biopsy was negative. Definitive criteria for the entity of so-called biopsy negative GCA are lacking, and, in my opinion, this concept remains a problematic one. Negative temporal artery (TA) biopsies do occur in certain subsets of GCA—for example, upwards of 50% of patients with so-called large artery involvement have such negative biopsies²—but the extent to which TA biopsies are negative in bona fide cases of cranial arteritis in GCA is unclear. Two recent papers have suggested that in fact a unilateral TA biopsy negative for arteritis markedly reduces the probability of the diagnosis of GCA, because the yield of a positive contralateral biopsy is no more than 1–3%.^{3,4}

The issue of what constitutes a flare in GCA (and PMR) is also problematic. It has been my experience over the years that many cases of alleged flares of both conditions involve little more than asymptomatic rises in the acute phase reactants, and that the pursuit of such rises with increased doses of steroids not uncommonly results in sundry untoward complications—notably, steroid induced osteoporosis and associated fractures.

The patient under discussion is a case in point. The acute phase reactants were raised coincident with the occurrence of a TIA, but it is unlikely that this latter episode was caused by GCA. Though GCA is occasionally complicated by stroke, such an event nearly always involves the territory of the vertebral-basilar circulation, and rarely occurs in the distribution of the internal carotid artery. The explanation for this fact may result from the specific exclusion of the intracranial arteries from involvement by GCA, possibly because these arteries lack an internal elastic lamina, which plays a pivotal part in the pathogenesis of GCA. The internal elastic lamina is said to be maintained for a few millimetres after the vertebral arteries pierce

the dura, thus accounting for the strokes referable to the vertebral-basilar circulation.⁵ The patient described by Tan *et al* had left arm weakness, almost surely attributable to ischaemia of the middle cerebral artery, thus effectively ruling out GCA as the cause for the TIA.

I therefore submit that this patient did not have "resistant giant cell arteritis"; rather, he represents a case of the successful treatment by tumour necrosis factor α (TNF α) blockade of symptoms and signs referable to PMR.

One final caveat: although further study may show that TNF α blockade does successfully reduce the levels of cytokines that drive the acute phase response in GCA, thus ameliorating constitutional symptoms and signs, this treatment may not mitigate the disease's most feared consequence—namely, ischaemia leading to visual loss. As demonstrated by elegant work over the past decade by Weyand and Goronzy,⁶ ischaemia in GCA results from an array of other cytokines with pathogenic potential—for example, platelet derived growth factor and vascular endothelial growth factor, which would be unaffected by TNF α blockade.

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References

- 1 Tan AL, Holdsworth J, Pease C, Emery P, McGonagle D. Successful treatment of resistant giant cell arteritis with etanercept. *Ann Rheum Dis* 2003;62:373–4.
- 2 Brack A, Martinez-Taboada V, Stanson A, Goronzy JJ, Weyand CM. Disease pattern in cranial and large-vessel giant cell arteritis. *Arthritis Rheum* 1999;42:311–17.
- 3 Hall JK, Volpe NJ, Galea SL, Grant TL, Syed NA, Balcer LJ. The role of unilateral temporal artery biopsy. *Ophthalmology* 2003;110:543–8.
- 4 Boyev LR, Miller NR, Green WR. Efficacy of unilateral versus bilateral temporal artery biopsies for the diagnosis of giant cell arteritis. *Am J Ophthalmol* 1999;128:211–15.
- 5 Wilkinson IMS, Russell RWR. Arteries of the head and neck in giant cell arteritis: a pathologic study to show the pattern of arterial involvement. *Arch Neurol* 1972;27:378–91.
- 6 Weyand CM, Goronzy JJ. Medium- and large-vessel vasculitis. *N Engl J Med* 2003;349:160–9.

Authors' reply

We thank Dr Dockens for his comments on the difficulty in the diagnosis, treatment, and classification of polymyalgia rheumatica (PMR) and giant cell arteritis (GCA). However, the sole purpose of our case report was to show how etanercept might have a role in the treatment of resistant disease of the PMR/GCA spectrum. From a clinical standpoint we are happy to label our patient as having GCA (in addition to PMR) based on his headaches, reported visual abnormalities, temporal tenderness, and resistance to 15 mg of prednisolone a day. We know that the

disease was resistant because his symptoms and laboratory abnormalities persisted despite continued use of relatively high dose steroids, and there was clinical deterioration mirrored by an increase of the acute phase response.

Of course it is entirely possible that his transient ischaemic attack was related to atheroma, even in the face of very active GCA, but a rare arteritic related event could not be excluded in the clinical circumstances. The adverse effect of high dose steroids on blood pressure and lipid profiles and their association with atheromatous related disease was an additional concern about their continued use. We agree that it would be folly to treat patients on the basis of a raised erythrocyte sedimentation rate (ESR) alone but an extremely high ESR, as in this case, invariably signifies disease flare. Activation of the inflammatory cascade has a pivotal role in the pathogenesis of GCA so it seems logical that anti-tumour necrosis factor treatment could abrogate this regardless of the other cytokine mediators of disease.

Fifteen months after the original case report the clinical diagnosis remains the same and the patient continues to experience flares in disease with tapering of the steroid dose below 5 mg/day.

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Fenofibrate and losartan

The leader by Professor Bardin¹ makes an excellent point. We could benefit from the hypouricaemic action of drugs that are not licensed for this use (for example, losartan and fenofibrate).

Other drugs in common use may also have a uricosuric effect. For example, atorvastatin can reduce serum uric acid concentrations in patients with peripheral arterial disease or hyperlipidaemia.^{2–4} However, the mechanisms involved are not clear cut; we speculate that atorvastatin can increase renal blood flow and decrease serum creatinine levels.^{2–4} Thus, the Medical Research Council/British Heart Foundation Heart Protection Study (HPS) showed that simvastatin decreased the deterioration of the glomerular filtration rate (GFR) over a period of 4.6 years in high risk patients with (n = 5963) or without (n = 14 573) diabetes.⁵ This effect on GFR would almost certainly influence urate excretion. These statin mediated effects are relevant because, as Professor Bardin¹ points out, patients with hyperuricaemia may also be dyslipidaemic.

Closer to the interests of rheumatologists are the non-steroidal anti-inflammatory drugs (NSAIDs). Some NSAIDs may exert a favourable effect on urate excretion. For example, diflunisal has been reported to have a uricosuric effect, although the inhibition of xanthine oxidase activity has also been proposed.^{6,7} Azapropazone (not used as a first line option) has been shown to lower serum urate levels.^{7,8} Indometacin may have uricosuric properties.⁷ Tiaprofenic acid

is another NSAID with hypouricaemic effect.⁹

Aspirin has a bimodal effect on the renal handling of uric acid. High doses (>3 g/day) are uricosuric, while lower doses (1–2 g/day) cause urate retention.¹⁰ At the lowest dose (75 mg/day) aspirin caused a 15% decrease in urate excretion with a slight but significant increase in serum urate levels.¹⁰

The clinical significance of these “additional” uricosuric effects remains to be established. There is also a need to assess the value of using combinations of these drugs (for example, losartan and fenofibrate together with an NSAID with beneficial effects on urate excretion).

The search for NSAIDs that do not exert renal toxicity may well be worthwhile because of their widespread use. Acute attacks of gout are usually treated with high doses of NSAIDs. It could be useful to have NSAIDs with uricosuric properties as well as the analgesic and anti-inflammatory effect.

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References

- Bardin T. Fenofibrate and losartan. *Ann Rheum Dis* 2003;**62**:497–8.
- Elisaf M, Mikhailidis DP. Statins and renal function. *Angiology* 2002;**53**:493–502.
- Youssef F, Gupta P, Seifalian AM, Myint F, Mikhailidis DP, Hamilton G. The effect of short-term treatment with simvastatin on renal function in patients with peripheral arterial disease. *Angiology* 2004;**55**:53–62.
- Athyros VG, Papageorgiou AA, Elisaf M, Mikhailidis DP on behalf of the GREACE Study Collaborative Group. Statins and Renal Function in Patients with Diabetes Mellitus. *Curr Med Res Opin* 2003;**19**:615–19.
- Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol-lowering with simvastatin in 5963 people with diabetes: a randomised placebo-controlled trial. *Lancet* 2003;**361**:2005–16.
- Ferraccioli G, Spisni A, Ambanelli U. Hypouricemic action of diflunisal in gouty patients: in vitro and in vivo studies. *J Rheumatol* 1984;**11**:330–2.
- Tiitinen S, Nissila M, Ruutsalo HM, Isomaki H. Effect of nonsteroidal anti-inflammatory drugs on the renal excretion of uric acid. *Clin Rheumatol* 1983;**2**:233–6.
- Higgins CS, Scott JT. The uricosuric action of azapropazone: dose-response and comparison with probenecid. *Br J Clin Pharmacol* 1984;**18**:439–43.
- Lyfar V, Fernandes L, Thornton E, Cook JG. A study of the hypouricaemic effect of tiaprofenic acid. *Ann Clin Biochem* 1988;**25**:595–600.
- Caspi D, Lubart E, Graff E, Hahot B, Yaron M, Segal R. The effect of mini-dose aspirin on renal function and uric acid handling in elderly patients. *Arthritis Rheum* 2000;**43**:103–8.

NOTIFICATION AND CORRECTION

Corrections printed in the journal also appear on the *Annals* website www.annrheumdis.com and are linked to the original publication

Intra-articular rheumatoid nodules and triggering of the knee joint (Tak-Diamant Z, Hoening van Duyvenbode FJ, Eulderink F, Janssen M. *Ann Rheum Dis* 1992;**51**:533–5.)

The name of the first author of this paper has changed from Tak-Diamant Z to Diamant Z.

Does long term treatment with azathioprine predispose to malignancy and death in patients with systemic lupus erythematosus? (Nero P, Rahman A, Isenberg DA. *Ann Rheum Dis* 2004;**63**:325–6.)

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We regret that the references for this letter were omitted. They are given below.

- Hazleman B. Incidence of neoplasms in patients with rheumatoid arthritis exposed to different treatment regimens. *Am J Med* 1985;**78**(suppl 1A):39–43.
- Moll JMH. Drug therapy (3): “specific” drugs. In: *Management of rheumatic disorders*. 1st ed. London: Chapman and Hall: 1983:169–212.
- Silman A, Petrie J, Hazleman B, Evans S. Lymphoproliferative cancer and other malignancy in patients with rheumatoid arthritis treated with azathioprine: a 20-year follow up study. *Ann Rheum Dis* 1988;**47**:988–92.
- Sutcliffe N, Smith C, Speight PM, Isenberg DA. Mucosa-associated lymphoid tissue lymphomas in two patients with rheumatoid arthritis on second-line agents, and secondary Sjögren syndrome. *Rheumatology (Oxford)* 2000;**39**:185–8.

NOTICE

8th EULAR Postgraduate Course in Rheumatology

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The course will cover clinical aspects of rheumatic disease concentrating on outcome, assessment, and evidence based management and will include the scientific basis of rheumatology. The course is aimed at junior rheumatologists at the end of their training but is open to all rheumatologists.

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