of a higher von Willebrand factor antigen in patients with diffuse disease suggests endothelial cell damage may be more extensive and active in this group. The extent of vascular injury as shown by von Willebrand factor antigen concentrations may be regarded as a prognostic marker in these disorders.

A D BLANN
Department of Surgery
Research and Teaching Unit
University Hospital of South Manchester
Nell Lane, Didsbury
Manchester M20 8LR, UK

KAREN J ILLINGWORTH
M J V JAYSON
University of Manchester Rheumatic Diseases Unit
Clinical Sciences Building
Hope Hospital
Eccles Old Road
Salford, Manchester M6 8HD, UK


Anticardiolipin antibodies in drug addicted patients with AIDS

Sir: The presence of anticardiolipin antibodies and lupus anticoagulant has been found in up to 90% of homosexual patients with AIDS.\(^1\) \(^2\) There have been some attempts to relate anticardiolipin antibodies with Pneumocystis carinii infection,\(^3\) other opportunistic infections, and with neoplasm,\(^4\) but up to the present, anticardiolipin antibodies in patients with AIDS have only been associated with a poor outcome of the illness.\(^5\) \(^6\) In these patients anticardiolipin antibodies do not have the same clinical significance as they have in systemic lupus erythematosus.

As drug addicted patients with AIDS have a different spectrum of rheumatic manifesta-
tions, with less reactive arthritis, Reiter's syndrome, and HIV related arthritis, and more septic arthritis than homosexual patients with AIDS (Montenegro I et al, unpublished data), we studied the presence of anticardiolipin antibody in drug addicted patients with AIDS and its relation with clinical manifestations.

Anticardiolipin antibody was determined (Cheshire Diagnostic QACA enzyme linked immunosorbent assay (ELISA) kit) in 55 drug addicted patients. Forty three of these had been admitted to hospital because of acute problems related to AIDS (mainly infections) and the remaining 12, with less acute problems, were seen at the outpatient AIDS clinic in the hospital. Anticardiolipin antibodies were posi-
tive in 42 (76%)—31/43 (72%) of the hospital patients and 11/12 (92%) of the outpatients.

Clinical problems in the hospital patients were diverse, and we found no significant relation between infection and the presence of anti-
cardiolipin antibodies. Additionally, there was no relation between anticardiolipin antibodies and the antecedent or kind of infection in these patients. Of the 55 patients, 43 were in stage IV AIDS, with 34 (79%) being positive for anticardiolipin antibodies. The remainder were in other stages and anticardiolipin anti-
bodies were found in 11/12 (83%).

Our results show that the presence of anticardiolipin antibodies in drug addicted patients with AIDS is similar to that found in homosexual patients with AIDS. We found no relation between anticardiolipin antibodies and clinical manifestations (mainly infections) nor pathological antecedents in these patients. Similarly, there were no differences between outpatients and hospital patients. As with homosexual patients with AIDS, we found no correlation between anticardiolipin antibodies and recurrent thrombosis and thrombopenia. Probably, anticardiolipin antibodies are asso-
ciated with HIV infection itself or with an abnormal immune response which is not yet well defined.

JAVIER RIVERA
INDALECIA MONTENEGUO
JAVIER LOPEZ-LONGO
EDUARDO MALDONADO
LUIS CARRENO
Servicio de Reumatologia
Hospital General Gregorio Maranon
Madrid, Spain

Correspondence to: Dr J Rivera, Servicio de Reu-
matologia, Hospital Gregorio Maranon, Doctor Esquerdo 46, 28007 Madrid, Spain.

5 Cano CR T, Zon LI, Groopman EJ. Anticardi-

6 Mizutani W T, Woods V L, McCustan J A, Zwaal R J. Anticardiolipin antibodies in human immunodeficiency virus (HIV) infected gay males may be associated with a deteriorat-

Disabling ossification of the patellar tendon

Sir: A 42 year old lorry driver without any medical or traumatic history had had for two years pains in the right knee when walking. They had appeared simultaneously with the use of a new truck, on which the accelerator pedal was particularly stiff. After some months of limping clinical examination showed that the right patellar tendon was diffusely thickened and tender; a bulge sign was found, as was a crepitus and a marked reduction of the knee flexion. Erythrocyte sedimentation rate was 1 mm/1 h; fasting blood sugar, calcium, phosphorus, and alkaline phosphatase were normal. Synovial fluid contained 0–1 \(10^2\) leucocytes without crystals.

Lateral plain radiographs and tomograms (without signs of patella alta: length of the patellar tendon equal to the diagonal length of the patella)\(^7\) showed ossification of the tendon, which did not affect its distal third (fig 1).

Sonography showed that ossification was mostly in the lateral part and that the non-
ossified distal third was thickened in com-
parison with the tendon on the left side (anteroposterior thickness 9 mm v 6 mm). Radiographs and computed tomography also showed irregular enthesitic osteophytes of the patella with calciation and/or addition, a femoral and tibial osteopikositis. Radiographs of cervical, thoracic, and lumbar spine showed no abnormalities.

Arthroscopic examination showed the joint capsule was normal. During operative and a orthopaedic surgeon found that the right patellar tendon was wider than normal. The ossified mass was not adherent to the adjacent patella or tibia and could be easily dissected and removed. The patient recovered slowly and was able to resume his work three months later.

Pathological examination showed compact remodelled lamellar bone. In the mass of the bone, and on the proximal end, strips of fibrous cartilage were seen, which might be considered as an abnormal metaphasia of the tendon (figs 2A and B). Despite its distance from any insertion site this intermingling of bone and fibrous cartilage resembles those seen in enthesopathic hyperostosis—that is, in a location in which fibrocartilaginous bundles are normally found.\(^2\) On the distal end tendinous bundles intermingled with scar tissue associated with some degree of scar

Figure 1 (A) Plain radiograph; (B) tomogram.
remodelling were inserted (figs 2C and D). No mucoid degeneration or deposits of calcium pyrophosphate dihydrate crystals, haemosiderin, or cholesterol were found.

This segmental ossification of the patellar tendon must be distinguished from the bony islands found near patellar or tibial insertions, which are sequelae of Sinding-Larsen or Osgood-Schlatter diseases respectively. Similar ossifications have exceptionally been reported in patellar tendon but more commonly in the Achilles tendon. In the absence of local severe injury such ossifications have been attributed to microtrauma. In our case they seemed to be related to repeated pressing on a stiff pedal of the lorry. Pain of two years’ duration is compatible with development of ossification. The scar was not simple, however. Swelling of the entire tendon indicated by clinical examination and by sonography, which might be attributed to the persistence of the mechanical stress, has been commonly reported in ‘jumper’s knee’—that is, patellar tendinitis, in participants of various sports.

The cause of ossification, an event which seems exceptional in jumper’s knee, remains to be explained. We know of only one case of a high jumper in whom partial biopsy of a patellar tendon ossification showed an aspect similar to that seen on the distal pole in our case. The presence of enthesopathic hyperostosis and of osteopoiikilosis (a curious condition characterised by bone formation at the intersection of spongy bone trabeculae) suggests predisposing factors to ossification.

This case is presented in a journal for rheumatologists, not only for its diagnostic interest, but also because it introduces a conceptual discussion of the respective roles of local stress and general environment in tendon or ligament ossification processes.

We thank Dr. D. Pelet (orthopaedic surgeon, Lausanne) for clinical information as well as Mrs. M. Maunoir and Miss A. Polchouk for technical and secretarial assistance.

R. LAGIER
Department of Pathology (Osteoarticular Unit)
Geneva Medical School
Geneva, Switzerland

J-C. GERSTER
Rheumatology and Rehabilitation Centre
University Hospital
Lausanne, Switzerland

Correspondence to: Dr. R. Lagier, Departement de Pathologie, CMI, 1 rue Michel-Servet, 1211 Geneva 4, Switzerland.