ness knows what would be the response to opening a rheumatic clinic. Like their brethren world wide, general physicians and orthopaedic surgeons cheerfully acknowledge their fumbling ineptitude when faced with locomotor disease of this variety and magnitude. Ours is a 'low tech' discipline compared with most these days, and our clinical skills and experience remain par-
amount. Elements can be taught at all levels of medical and paramedical practice, however, with some expectation of benefit to patients. South of the Sahara and north of Zambesi there lies a rheumatological abyss. The International League Against Rheumatism (ILAR) has recently established an African regional group: (AIFAR),\(^1\) which expects to hold an inaugural congress in Cairo in 1991. This may prove a catalyst to re-establishing medical links between Britain, Europe, and Africa sundered in the post-independence era. From Africa and its peoples we may learn how to husband meagre resources, stifle the demands of minor ailments, and perhaps, through appropriate research, explore the link between environmental and genetic factors in a variety of rheumatic disorders.

P E McGILL
Rheumatology Department
Stobhill General Hospital
Glasgow G21 3JW


Increased serum cytidine deaminase activity in gout and articular chondrocalcinosis

Sir: Thompson et al studied the activity of cytidine deaminase in serum and synovial effusions of patients with rheumatoid arthritis,\(^1\) the activity after withdrawal of non-steroidal anti-inflammatory treatment,\(^2\) and the circadian rhythm of serum cytidine deaminase activity.\(^3\) Increased activity of cytidine deaminase was found in serum and synovial effusions of patients with rheumatoid arthritis. The authors assumed that cytidine deaminase released from damaged neutrophils diffuses from all inflamed joints into the blood, so that serum cytidine deaminase activity may provide an integrated measure of joint inflammation more specific than traditional measures, such as the erythrocyte sedimentation rate. Increased granulocyte turnover and activation are involved in the inflammatory process connected with crystal induced arthritis. This prompted us to study the changes of serum cytidine deaminase in gout and arthritic chondrocalcinosis. Measurements of cytidine deaminase activity were carried out with cytidine as a substrate, and the ammonia liberated was determined by a modified Berthelot reaction.\(^4\)

Fifty four patients with gout aged 32-57 years (mean 49), seven patients with chondrocalcinosis aged 56-69 years (mean 63) with proved crystals of sodium urate and calcium pyrophosphate dihydrate in synovial effusions, and 32 healthy controls aged 29-60 years (mean 47) were studied. Subjects with liver damage and increased serum creatinine were excluded.

Serum cytidine deaminase activity was significantly higher in patients with gout than in healthy controls (mean (SD) healthy 2.5 (0-7) units/ml, patients with gout 5.4 (2.9) units/ml, p<0.005). In three hospital in-patients the cytidine deaminase activity reflected a gouty attack (figure). The patients were receiving a purineless diet without treatment or they were receiving some steroids and anti-inflammatory drugs (isopropenyl acid, diclofenac). Patient No 2 had two attacks, following each other. Cytidine deaminase activity increased before the attack with the maximal value occurring during the attack and a subsequent decrease. Regardless of the basal values the appearance of acute arthritic syndrome in gout was preceded by an increase of cytidine deaminase. The patients with the primary hereditary form of chondrocalcinosis,\(^5\) and who developed secondary arthrosis, came from Velká Mača, a village located in southern Slovakia. Their serum cytidine deaminase values were significantly higher (5.3 (1.8) units/ml) than those of healthy controls (p<0.001). The highest values of cytidine deaminase (7.5 and 7.7 units/ml) were found in two patients with active arthritic syndrome (on knees, metacarpophalangeal joints, and wrists).

Our observations indicate that serum cytidine deaminase reflects the inflammatory activity not only in rheumatoid arthritis and other autoimmune, bacterial, and viral inflamm-

ancies but also in crystal induced arthritides, such as gout and chondrocalcinosis. Higher cytidine deaminase values were present in these patients with no evidence of arthritic syndrome, but the cytidine deaminase activity increased further with the appearance of acute arthritic syndrome. Phagocytosis by neutrophils of monosodium urate and calcium pyrophosphate dihydrate monocrystals can cause the release of lysosomal enzymes, as well as a higher turnover of neutrophils; damage and breakdown of these cells would cause increased cytidine deaminase activity. Our results correspond with the investigation of Thompson et al\(^6\) and extend their findings, showing that higher cytidine deaminase activities are also present in gout and chondrocalcinosis.


---


Increased serum cytidine deaminase activity in gout and articular chondrocalcinosis.

J Rovenský, M Stancíková, K Bosmanský and M Kovalancík

*Ann Rheum Dis* 1991 50: 659
doi: 10.1136/ard.50.9.659

Updated information and services can be found at: http://ard.bmj.com/content/50/9/659.citation

**Email alerting service**
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to: http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to: http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to: http://group.bmj.com/subscribe/