SISTEMIESE LUPUS ERITEMATOSE

Lupus eritematose is reeds in sy plaaslike diskoïede vorm bekend vir nagenoeg honderd-en-twintig jaar. Kaposi het in 1872 daarop gelet dat konstitusionele simptome dikwels daarmee gepaard gaan, maar die beskrywing van 'n niebakteriële endokarditis in twee gevalle met lupus eritematose deur Libman en Sacks in 1924 bevestig die betekenis van 'n verspreide, of sistemiese vorm van lupus eritematose[1] (S.L.E.).

Die term kollageensiekte is in 1942 deur Klemperer, Pollack en Baehr[8] ingevoer *slegs* om daarop te dui dat 'n groep siektes van onbekende etiologie 'n gemeenskaplike patologiese kenmerk het, nl. beskadiging van die intersellulêre stof in bindweefsel, die ‚kollageen'.[1]

Die ontdekking van die ‚L.E.-sel' en ‚-verskynsel' in 1948 deur Hargreaves *et al.*[7] het 'n laboratoriumtoets in die hande van die klinikus geplaas om verdagte gevalle wat diagnostiese probleme het, te help ontdek en sodoende 'n vroeër diagnose te bewerkstellig.

Tot ongeveer 1950 is S.L.E. dus as 'n progressiewe en noodlottige siekte beskou. Jessar *et al.*[2] toon egter in 1953 dat uit 'n betreklike groot reeks, spontane afname van die siekte in ongeveer een derde van die gevalle voorkom.

Dit is bekend dat die steroïede 'n groot invloed op hierdie siektetoestand het, maar 'n noukeurige waardebepaling van die spesifieke indikasies en beperkings van hierdie soort terapie is nog nie bevredigend gelewer nie.[3] Die kwessie van swangerskap en S.L.E. is ook nog nie finaal opgelos nie.[4]

Die etiologie van die siekte is, soos reeds genoem, nog in duisternis gehul. Soos met enige toestand van onbekende etiologie, ontbreek dit ook hier nie aan hipotetiese verklarings nie. Onder die vroegste verklarings is die rol van bakterieë as etiologiese faktor ingesluit, maar hierdie verklaring het gou verdwyn. 'n Hipersensitiwiteitsreaksie skyn tans 'n sterker moontlikheid te wees en wel omdat die serologiese toetse vir sifilis dikwels antikomplimentêr is in hierdie siekte. Die toename van S.L.E. val verder ook saam met die toenemende gebruik van antibiotiese middels, en die L.E.-verskynsel is dan ook al gedemonstreer by persone wat sensitief is vir penisillien. 'n Verhoogde globulien in die serum in hierdie gevalle versterk ook verder die hipersensitiwiteitshipotese.[1] Die beskrywing van twee onlangse gevalle na mesantoïen-terapie is miskien 'n verdere punt ten gunste van dié teorie.[5]

Die fotosensitiwiteit van hierdie pasiënte het 'n moontlike verwantskap met die wanmetabolisme van porfirie ter sprake gebring, maar geen bewyse hiervoor is gevind nie. Endokrien-versteurings is vermoed en 'n abnormale sellulêre metabolisme met verhoogde produksie van gammaglobulien het ook aandag geniet.[1]

In 'n onlangse artikel, wat hopelik die voorloper van ander soortgelyke ondersoeke sal wees, probeer McCombs en Patterson[3] om faktore wat die verloop en prognose van S.L.E. beïnvloed, te ontleed. Uit 'n reeks van sewe-en-sewentig pasiënte vind hulle dat die siekte baie meer seldsaam is by mans as by vrouens, maar dat die mans 'n baie ernstiger beeld vertoon en 'n veel hoër sterftesyfer het. As die diagnose voor een-en-twintig jarige ouderdom gemaak word, is die prognose ernstig, maar diegene by wie die siekte na die ouderdom van vyf-en-veertig jaar ontdek word, het 'n veel gunstiger prognose.[3] Tekens van renale aantasting, selfs by minimale urinêre afwykings, dui op die ernstigste prognose.

Kortikosteroïede terapie verhoed nie die voortskreidende aard van die renale letsel nie, maar het skynbaar tog 'n voorkomende uitwerking. Poliserositis en miokarditis, in die afwesigheid van renale patologie, reageer gunstig op terapie met steroïede.[3]

In sommige gevalle was swangerskap skynbaar 'n presipiterende faktor, maar, as die siekte egter reeds aanwesig is en bewys van renale aantasting afwesig, kan swangerskap met veiligheid onderneem word met 'n redelike verwagting op 'n normale baba.[3]

In die gevalle wat met steroïede behandel is in die genoemde reeks, was die dood te wyte aan nierversaking of die komplikasies van terapie met steroïede in die algemeen.[3]

In 'n siekte wat beslis toeneem, soos blyk uit 'n vergelyking tussen drie twee-jaar periodes (1938 - 1939, 1948 - 1949, en 1954 - 1955) in Swede,[6] behoort ons alle informasie aangaande die etiologie, die natuurlike verloop, en die waarde van steroïede of enige ander vorm van terapie, te verwelkom. Aangesien penisillien, apresolien, en nou ook mesantoïen[5] in sommige gevalle as oorsaaklike faktore voorkom, moet ons verwag om ook nog velerlei ander skadelike middels teen te kom.

'n Toestand wat nie so lank gelede nie nog net as 'n mediese wetenswaardigheid beskou is, het nou 'n belangrike siekte geword, en in die nabye toekoms mag dit selfs 'n baie ernstige probleem word.

1. Talbott, J. H. en Ferrandis, R. M. (1956): *The Collagen Diseases.* New York en Londen: Grune & Stratton.
2. Jessar, R. A., Lamont-Havers, R. W. en Ragan, C. (1953): Ann. Intern. Med., **38**, 717.
3. McCombs, R. P. en Patterson, J. F. (1959): New Engl. J. Med., **260**, 1195.
4. Friedman, E. A. en Rutherford, J. W. (1956): Obstet. & Gynec., **8**, 601.
5. Lindqvist, T. (1957): Acta med. scand., **158**, 131.
6. Svanborg, A. en Sölvell, L. (1957): J. Amer. Med. Assoc., **165**, 1126.
7. Hargreaves, M. M., Richmond, H. en Morton, R. (1948): Proc. Mayo Clin., **23**, 25.
8. Klemperer, P., Pollack, A. D. en Baehr, G. (1942): J. Amer. Med. Assoc., **119**, 331.

HUMAN CAPILLARIASIS AND ITS TREATMENT

In July 1957 an article by Cochrane, Sagorin and Wilcocks[1] was published in this *Journal* reporting a case of human infection with *Capillaria hepatica* in the person of a White child, fifteen months old when the illness began, living on the small holdings in the outskirts of Vanderbijlpark, Transvaal. Only four human cases had previously been reported in the medical literature, and this was the first case to be recorded in South Africa. The illness began with pyrexia

in June 1956 and during the following three or four months the child was extremely ill, with a swinging temperature. The outstanding findings were enlargement and other changes in the liver, a high degree of eosinophilia, and maximally positive tests for disordered liver function. The eosinophil count ranged from 6,630 to 32,526 per c.mm. (25 - 78% of the total white cells) and the serum gamma globulin reached 5 g. per cent. Liver biopsy showed large numbers of characteristic ova in the portal tract, surrounded by dense cellular infiltration, which in some areas had been replaced by fibrosis, with marked distortion of the liver pattern and degeneration of liver cells. The general appearance suggested that the ova were those of a species of Trichuris, but on further study they were recognized as of *Capillaria hepatica*. Capillaria belongs to the same family as Trichuris (whipworm), which it closely resembles.

This nematode occurs in many animals, and in South Africa it has been found in rats, mice, rabbits, hares, gerbilles and meerkats. At the Institute for Medical Research, Johannesburg, nineteen out of a random group of forty rats received for examination were found to be infested with Capillaria. Where the human patient was living, rats and mice as well as other animals moved freely, and one field rodent (mastomys) trapped at her home in November 1956 showed extensive infestation with Capillaria.

The ova of *Capillaria hepatica* are not discharged in the faeces, and only escape from the liver of the host on its death, when, in rats, they are released through cannibalism on the part of these rodents. Whether the rat which eats the liver develops the disease, or whether the ova must first pass through this animal to be ingested by another, appears to be uncertain. In any case, the embryonated eggs pass to the caecum and thence to the liver, where the worms mature, lay their eggs, and then die and disintegrate. It is interesting to note that geophagy has been recorded in the few known human cases, and that the child infected at Vanderbijlpark is stated to have been 'a prodigious earth-eater'.

In the treatment of their case our authors were faced with the fact that all the four previous cases reported had ended fatally. They therefore rejected the drugs that had been given in those cases and, influenced by certain clinical and histological resemblances to bilharziasis, they selected an antimony preparation, sodium antimomyl gluconate ('triostam'), which had been reported as effective against the haematobium and mansoni bilharzia and against trypanosomes. Large intravenous doses over seven days appeared to clear up the condition and, in a follow-up article[2] which appears in the present issue, Cochrane and Skinstad report that the child is in excellent health and shows no clinical signs of the severe illness she passed through three years ago. On exploratory laparotomy the liver is of normal size and appearance, and on biopsy the only significant pathological change is some 'not very marked' periportal fibrosis. The liver function tests are in strong contrast with those of 1956, the eosinophils now being four per cent and the gamma globulin 0·9 g. per cent. This is the first recovery amongst the five human cases reported.

It will be noted that this study, which is an important contribution to our knowledge of this rare disease in man, in the fields both of diagnosis and treatment, is based on cooperation between laboratory workers of the South African Institute for Medical Research and the clinicians.

1. Cochrane, J. G., Sagorin, L. and Wilcocks, M. G. (1957): S. Afr. Med. J., **31**, 751.
2. Cochrane, J. G. and Skinstad, E. E. (1960): *Ibid.*, **34**, 21.