

South African Medical Journal

Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL

CHRONIC HEPATITIS

On the whole, acute viral hepatitis is a benign condition with a favourable outcome, the vast majority of patients being restored to good health. However, several complications are recognized, the most serious being massive necrosis of the liver; if the patient survives this, he will develop post-necrotic scarring. Although diffuse fibrosis (Laennec's cirrhosis) is an uncommon consequence, it now appears that in England it is more often due to hepatitis than to alcoholism.¹

The meaning of the term 'chronic hepatitis' has long been debated. Very often it has mistakenly been applied to other conditions, e.g. post-hepatitic hyperbilirubinaemia or established cirrhosis. It has now been investigated for some years by workers at the Walter and Eliza Hall Institute in Melbourne; they describe the entity as a condition where the patient has prolonged jaundice, pyrexial episodes, hepatomegaly and gross splenomegaly, spider angiomata, low albumin level and raised gamma-globulin level in the serum, and a high erythrocyte sedimentation rate.² In all cases they had histological confirmation of the diagnosis. Young women predominated in their series.

Joske and King³ later described the occurrence of the lupus-erythematosus (L.E.) cell phenomenon in this type of hepatitis; and others have confirmed this observation.⁴ Mackay *et al.*,⁵ from the same unit, have reported 7 cases of active chronic hepatitis in which L.E. cells were demonstrated; some showed other evidence of systemic lupus erythematosus (S.L.E.), but this was never gross. They have called this syndrome 'lupoid hepatitis'. The same workers performed liver biopsy on 7 cases of S.L.E. and encountered mild liver damage in only 3. This form of chronic hepatitis is thought to be related to S.L.E., though not to be part of the typical disease. It is suggested that in some cases of hepatitis the components of altered liver cells become antigenic and stimulate the production of antibodies that damage the liver and other tissues.⁵ As this type of reaction is known to occur in Hashimoto's disease, where the serum contains precipitins to thyroglobulin and thyroid tissue,^{6, 7} the suggestion seems reasonable, even though we do not know how and why the hypersensitivity comes about.

As cirrhosis develops, and becomes part of the picture, the

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CHRONIESE LEWERONTSTEKING

Oor die algemeen is akute virusontsteking van die lever 'n goedardige kondisie met 'n gunstige afloop; die meeste pasiënte herwin hul gesondheid. Verskeie komplikasies is egter bekend waarvan massiewe lewernekrose die ernstigste is; orlewe die pasiënt hierdie verwikkeling, word na-nekrotiese littekens oorgehou. Hoewel verspreide fibrose (Laennec se sirrose) 'n seldsame gevolg is, blyk dit nou dat dit in Engeland meer dikwels deur lewerontsteking as deur alkoholisme¹ veroorsaak word.

Die betekenis van die woorde 'chroniese lewerontsteking' word reeds lankal beredeneer. Dit is reeds dikwels verkeerdlik gebruik om ander toestande soos bv. hiperbilirubinemie ná lewerontsteking, of gevinstige sirrose, te beskrywe. Hierdie siekte is gedurende die afgelope paar jaar bestudeer deur werkers aan die Walter en Eliza Hall-instituut in Melbourne; hulle beskrywe die siekte-eenheid as 'n kondisie waarby die pasiënt ly aan langdurige geelsug, koorsaanvalle, lewervergrotting en kwaai milt vergroting, 'spinnekop-vatgeswelle', 'n lae albumien- en verhoogde gammaglobulien gehalte in die serum, en 'n hoë eritrosiet-besinkingspoed.² Hulle het by al hulle gevalle histologiese bevestiging van die diagnose verkry. Die meeste van die gevalle in hulle reeks was jong vroue.

Joske en King³ het later die voorkoms van die lupus-erythematosus (L.E.) sel-verskynsel by hierdie soort lewerontsteking beskryf, en ander werkers het hierdie waarneming bevestig.⁴ Mackay *et al.*,⁵ uit dieselfde groep, het 7 gevallen van aktiewe chroniese lewerontsteking waar L.E.-selle aangetoon was gerapporteer; by party pasiënte was daar ander tekens van sistemiese lupus erythematosus (S.L.E.) maar dit was nooit ernstig nie. Hulle het hierdie sindroom 'lupoid hepatitis' genoem. Hierdie werkers het ook 'n lewerbiopsie op 7 gevallen van S.L.E. uitgevoer en het ontdek dat daar by slegs 3 van die pasiënte lige lewerbeskadiging was. Daar word gemeen dat hierdie vorm van lewerontsteking verwant is aan S.L.E., hoewel dit nie as deel van die tipiese siekte voorkom nie. Dit word voorgestel dat, by sommige gevalle van lewerontsteking, die bestanddele van veranderde lewerselle antigenies word en die produksie van teenliggaampies stimuleer wat die lewer en ander weefsels beskadig.⁵ Aangesien dit bekend is dat hierdie soort reaksie ook by Hashimoto se siekte voorkom, waarby die serum presipitiene vir tiro-globulien en skildklierweefsel bevat,^{6, 7} skyn dit 'n redelike voorstel te wees, al weet ons nie hoe of waarom die oorgenoegelheid ontstaan nie.

Met die ontwikkeling van die sirrose, en namate dit deel word van die siektebeeld, vererger die prognose. Behandeling met steroïede, wat oënskynlik aangewese is vanweë die

prognosis is poor. Treatment with steroids, which seems indicated in view of the likelihood of a hypersensitivity reaction, is, unfortunately, of little help; transient subjective and biochemical improvement often occurs, but the histological appearances remain unchanged.⁸

Another aspect of 'chronic hepatitis' has been considered by Bongiovanni and Eisenmenger, who describe a chronic hepatic disorder of unknown aetiology which again occurs mostly in young women.⁹ In this, jaundice starts insidiously and varies in degree over a prolonged period; at the same time signs of hyperadrenalinism are noted, viz. hirsutism, acne, abdominal striae, obesity, amenorrhoea, mooning of the face, and high urinary corticoid levels. As in cases of 'lupoid hepatitis,' a high serum level of gamma globulin is the rule. In spite of the evidence of hypercortisolism the exhibition of ACTH has led to temporary improvement (a fall in the serum-bilirubin and serum-globulin levels, and a rise in the serum albumin). Some of the cases have developed typical Laennec's cirrhosis. The pathogenesis of the endocrine abnormalities is not yet established; a possible suggestion is that the liver in these cases is unable to degrade adrenocortical hormones.

It seems likely that these two syndromes^{5, 9} are expressions of the same disorder, for Wilkinson and Sacker⁴ have recorded a case of cirrhosis in a young woman who showed the features of both; at autopsy there was no evidence of S.L.E. This form (or forms) of chronic cirrhosis constitutes a rare disorder, but it is of great pathological interest, and it may well be that further studies of it will shed light on the pathogenesis of Laennec's cirrhosis.

1. Sherlock, S. (1955): *Disease of the Liver and Biliary System*. Oxford: Blackwell.
2. Saint, E. G., King, W. E., Joske, R. A. and Finckh, E. S. (1953): Quoted by Mackay *et al.*⁵
3. Joske, R. A. and King, W. E. (1955): *Lancet*, 2, 477.
4. Wilkinson, M. and Sacker, L. S. (1957): *Brit. Med. J.*, 2, 661.
5. Mackay, I. R., Taft, L. I. and Cowling, D. C. (1956): *Lancet*, 2, 1323.
6. Roitt, I. M., Doniach, D., Campbell, P. N. and Hudson, R. V. (1956): *Ibid.*, 2, 820.
7. Goudie, R. B., Anderson, J. R., Gray, K. G., Clark, D. H., Murray, I. P. C. and McNicol, G. P. (1957): *Ibid.*, 2, 976.
8. Last, P. M. (1957): *Med. J. Austral.*, 1, 672.
9. Bongiovanni, A. M. and Eisenmenger, W. J. (1951): *J. Clin. Endocr.*, 11, 152.

moontlikheid van 'n oorgevoeligheid-reaksie, help ongelukkig maar min; daar is dikwels 'n vervlietende subjektiewe en biochemiese verbetering, maar die histologiese verskynsels bly maar onveranderd.⁸

Bongiovanni en Eisenmenger bespreek nog 'n aspek van 'chroniese lewerontsteking,' en beskryf 'n chroniese lewer-aandoening van onbekende oorsprong wat ook meestal jong vrouens as slagoffers tel.⁹ By hierdie aandoening begin die geelsug sluipend en varieer dit ingraad oor 'n lang tydperk; terselfdertyd is daar tekens van oormatige byniewerking soos harigheid, aknee, strepe op die buik, vetsug, amenoree, 'maangesig', en hoë uriengehalte aan kortikoëde. Net soos by gevalle van 'lupoid hepatitis' bevat die serum gewoonlik baie gamma-globulien. Ten spye van die tekens van hiper-kortisonisme, het die ekshibitie van A.C.T.H. tydelike verbetering meegebring ('n daling in die serumgehaltes aan bilirubien en globulien, en 'n styling in serumalbumien). By party van die gevalle het tipiese Laennec-sirrose ontwikkel. Die patogenese van die endokrien-afwykings moet nog bevestig word; 'n moontlike voorstel is dat by hierdie gevalle die lewer nie in staat is om die bynierskorshormone af te breek nie.

Dit is waarskynlik dat hierdie twee syndrome^{5, 9} uitings is van een en dieselfde steuring, want Wilkinson en Sacker⁴ het 'n geval van sirrose gerapporteer—'n jong vrou wat die kenmerke van albei getoon het—maar by die lykskouing was daar geen tekens van S.L.E. nie. Hierdie vorm (of vorms) van chroniese sirrose is 'n seldsame siekte, dog patologies baie interessant, en dit is heel moontlik dat verdere bestudering daarvan lig sal werp op die patogenese van Laennec se sirrose.

1. Sherlock, S. (1955): *Disease of the Liver and Biliary System*. Oxford: Blackwell.
2. Saint, E. G., King, W. E., Joske, R. A. en Finckh, E. S. (1953): aangehaal deur MacKay *et al.*⁵
3. Joske, R. A. en King, W. E. (1955): *Lancet*, 2, 477.
4. Wilkinson, M. en Sacker, L. S. (1957): *Brit. Med. J.*, 2, 661.
5. Mackay, I. R., Taft, L. I. en Cowling, D. C. (1956): *Lancet*, 2, 1323.
6. Roitt, I. M., Doniach, D., Campbell, P. N. en Hudson, R. V. (1956): *Ibid.*, 2, 820.
7. Goudie, R. B., Anderson, J. R., Gray, K. G., Clark, D. H., Murray, I. P. C. en McNicol, G. P. (1957): *Ibid.*, 2, 976.
8. Last, P. M. (1957): *Med. J. Austral.*, 1, 672.
9. Bongiovanni, A. M. en Eisenmenger, W. J. (1951): *J. Clin. Endocr.*, 11, 152.