

**EDITORIAL : VAN DIE REDAKSIE**

**EOSINOPHILIC GRANULOMA OF THE LUNG**

Formerly the Hand-Schüller-Christian disease and the Letterer-Siwe disease were thought to be primary disturbances of lipid metabolism associated with a disturbance of the intracellular enzymatic systems concerned with the formation of cholesterol. However, the close relationship of these conditions to eosinophilic granuloma of the bones has given rise to new aetiological concepts. Largely owing to the work of Mallory<sup>1</sup> and Lichtenstein,<sup>2</sup> it was appreciated that the 'pathologic common denominator' of all three conditions was a distinctive and apparently specific inflammatory histiocytosis. Lichtenstein proposed the term 'histiocytosis X' for the whole group of conditions.

Eosinophilic granuloma represents the mildest form of the malady, and many reports describe cases of eosinophilic granuloma in bones, either isolated, or associated with similar lesions involving soft tissues, lungs, kidneys and spleen.<sup>3-5</sup>

In 1952 Lackey *et al.*<sup>6</sup> first reported cases of eosinophilic granuloma limited to the lungs. Whether this is the sole situation, or whether lesions are also present in extra-pulmonary sites but are not clinically detectable, remains for the future to decide.<sup>7</sup> Eosinophilic granuloma involves the lung either in the form of discrete nodular masses of granulomatous tissue, or in the form of diffusely disseminated granulomatous tissue. Later, parenchymal fibrosis becomes marked. These changes are not restricted to the lung parenchyma, but may be seen in the tracheobronchial tree, the lymph glands and the pleura. Associated with the changes is cystic development, which results from granulomatous obstruction and later cicatricial constriction of the bronchiolar lumina. The resulting cysts vary in size from small blebs to giant bullae. The lungs may eventually present a honeycombed appearance.

Histological studies demonstrate early accumulation of sheets of histiocytes and an associated inflammatory-cell infiltration consisting mainly of eosinophils, but also containing plasma cells. The granulomatous phase, in which fibroblastic activity occurs, is followed by the xanthomatous phase, in which isolated histiocytes or nests of histiocytes

are transformed into foam-cells. The final stage—that of fibrosis—is the end phase of healing. There is no strict demarcation between the different stages and much overlapping occurs.

Clinically the condition affects any age and both sexes. The patients may be asymptomatic and detected only on routine screening of the chest, or they may present with chronic productive cough, haemoptysis, fever, night sweats and spontaneous pneumothorax. Later dyspnoea and extreme right-sided heart failure result from the extensive pulmonary fibrosis.

Early on, X-ray examination of the chest shows generalized reticulation associated with miliary mottling. Later, diffuse soft nodular infiltrations of various sizes are seen. Fibrosis may be extensive, and so may the cystic changes, which vary from fine scattered cystic reticulation to bullous degeneration and advanced honeycombing. The mediastinal and hilar lymph glands may be extensively diseased. The differential diagnosis is wide, and pulmonary tuberculosis, sarcoidosis, various fungus infections and the reticuloses must all be considered. Lung biopsy is essential.<sup>8</sup>

The outlook for a patient with eosinophilic granuloma of the lung is uncertain; but the dual danger of a 'spill-over' into the Letterer-Siwe or the Hand-Schüller-Christian disease on the one hand, and of the development of crippling pulmonary fibrosis on the other, demands a guarded prognosis. Treatment of this disease is still highly unsatisfactory. ACTH, cortisone and X-ray therapy have all been tried with varying degrees of success.<sup>9</sup> In general, however, the patients receiving various forms of therapy have fared no better than those receiving no therapy at all.

1. Mallory, T. B. (1942): New Engl. J. Med., 227, 955.
2. Lichtenstein, L. (1953): Arch. Path. (Chicago), 56, 84.
3. Weinstein, A., Francis, H. C. and Sprofkin, B. F. (1947): Arch. Intern. Med., 79, 176.
4. Parkinson, T. (1949): Brit. Med. J., 1, 1029.
5. Adams, P. and Kraus, J. E. (1950): Arch. Derm. Syph. (Chicago), 61, 957.
6. Lackey, R. W., Leaver, R. Y. and Farinacci, C. N. J. (1952): Radiology, 59, 504.
7. Thompson, J., Buechner, H. A. and Fishman, R. (1958): Ann. Intern. Med., 48, 1134.
8. Virshup, M. and Goldman, A. (1956): J. Thorac. Surg., 31, 226.
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**INFEKSIETOESTANDE VAN DIE MAAGDERMKANAAL**

By 'n vorige geleentheid<sup>1</sup> het ons aangetoon hoedat sulke siektetoestande soos maagkoers heeltemal onder beheer gebring kan word hoofsaaklik deur die toepassing van gesondheidsmaatreëls. Ons het die geskiedenis van hierdie siekte in sommige van die ouere Westerse lande geskets en daarby gewys hoedat die sterfesyfer van maagkoors met meer as 99 persent gedaal het in gebiede waar bevredigende beheer uitgeoefen word oor watervoorsiening, waar 'n stelsel van waterbeheer bestaan, en waar daar faciliteite is vir die afsondering van besmetlike gevalle en vir die beheer van voedselhantering in al sy aspekte.

Behalwe maagkoers, waarna ons hierbo verwys het en wat in 'n groot mate onder beheer gebring is deur die doeltreffende toepassing van gesondheidsmaatreëls, is daar nog 'n groot aantal infeksiotoestande van die maagdermkanaal wat as siektelepog nog een van die hoofoorsake van sterftes in ons land vorm. So skryf Truswell,<sup>2</sup> byvoorbeeld, 'Maagdermonsteking word aangegee as die gesertifiseerde oorsaak van dood in 721 gevallen in Kaapstad vir die jaar geëindig op 30 Junie 1955'. Hansen<sup>3</sup> sê: 'Dit word bereken dat in een gebied in Suid-Afrika 10 persent van die Bantoe-kinders gedurende hul eerste lewensjaar aan diaree-toe-

stande beswyk'. Robertson<sup>4</sup> verklaar: „Die kindersterfesyfer vir infeksietoestande van die maagdermkanaal was in 1958 by Blankes 3·1 per 1,000 kinders wat lewendig gebore is, en by nie-Blankes was die syfer 42·1. . . . Dit is nie moontlik om soortgelyke syfers te gee vir die insidensie van die siekte nie aangesien gevalle van infeksietoestande van die maagdermkanaal nie siektes is wat aangemeld moet word nie'. Ook sterf daar jaarliks honderde babatjies aan enteritis en duisende persone aan sogenaamde somer-diaree.

Die bewyse bestaan dus dat toestande soos diaree en enteritis nog baie algemeen voorkom. En daarby weet ons dat hierdie siektes in 'n groot mate deur vlieë versprei word. Na analogie van die uitwerking wat die strenge toepassing van gesondheidsmaatreëls op die verminderde voorkoms van maagkoers gehad het, kan ons nie anders nie as om dergelike maatreëls aan te beveel by die bestryding van enteritis en diaree. Trouens, hierdie siektes behoort deur wetgewing aanmeldbare siektes gemaak te word, want dit sal ons dan beter in staat stel om, onder andere, 'n nasionale program van bestryding van vlieë dwarsoor die land te onderneem.

Ten spyte van die feit dat die huisvlieg 'n weerstand

ontwikkel het teen 'n groot aantal insekdodende middels wat vandag gebruik word, kan daar tog baie gedoen word om hierdie plaag doeltreffend te bestry. In 'n artikel wat ons elders in hierdie uitgawe plaas, word die rol van die ‚sosiale spinnekop' by die bestryding van infeksietoestande deur die huisvlieg beskryf. Dit is verblydend om te weet dat 'n probleem soos die bestryding van vlieë alreeds met sukses aangepak word selfs op uitgestrekte plase.

Die artikel waarna ons nou net verwys het handel maar net oor een moontlike metode van benadering van die probleem van die bestryding van vlieë. Indien ons sukses verwag, moet die probleem egter op veel groter skaal aangepak word soos byvoorbeeld gedoen word deur die instandhouding van 'n malaria-veldpersoneel in die Transvaal vir die uitwissing van malaria. En, indien ons die hoop koester om nie net vlieë suksesvol te bestry nie, maar al die maniere waarop en middels waardeur die infeksietoestande waaroor ons skryf versprei word, sal ons 'n doelgerigte en volgehoue poging op 'n landswye basis moet daarstel.

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2. Truswell, A. S. (1957): *Ibid.*, 31, 446.
3. Hansen, J. D. L. (1957): *Ibid.*, 31, 452.
4. Robertson, I. (1957): *Ibid.*, 31, 441.