# PNEUMOCYSTIS PNEUMONIA

W. J. PEPLER, B.Sc., M.D., *Institute for Pathology, University of Pretoria, Pretoria*

Since Ammich[1] in 1938 first described 14 cases of the condition known as interstitial plasma-cell pneumonia, a large number of reports have appeared from various European countries describing this severe respiratory disease which affects mainly premature and undernourished infants. In 1952 Vanek and Jirovec[2] demonstrated in the lungs of their cases of interstitial plasma-cell pneumonia large numbers of parasites, which they identified as *Pneumocystis carinii*, and since then Hamperl[3] has confirmed the presence of these parasites in all 7 of Ammich's cases from which slides were still available for examination.

In 1953 Deamer and Zollinger[4] published a review of the literature on this condition and at that stage the disease seemed to be confined to Europe. However, since then cases have been reported from Britain,[5,6] the USA,[7,8] Canada,[9,10] South America[11] and Australia.[12]

To our knowledge no cases of this disease have been reported from Africa and in view of the fact that the condition shows a definite predilection for undernourished infants, it seemed advisable to draw attention to this condition here, because now that it has appeared on the scene, it should theoretically be seen with increasing frequency.

## CASE REPORT

A premature Bantu male infant, aged 6 days and weighing 4 lb. 12 oz., was admitted to hospital on 7 January 1958 with the diagnosis of imperforate anus. On the same day a colostomy was done under general anaesthesia. The postoperative course was uneventful and the baby gradually put on weight. On 23 March 1958 he suddenly developed an acute attack of gastro-enteritis with a temperature of 103°F. At the same time slight dyspnoea and cyanosis was noted and on examination he was found to have signs of pneumonia. Tetrex and penicillin were given and the temperature returned to normal. Subsequently a ventral hernia developed next to the colostomy wound and the child gradually deteriorated and died on 28 March 1958.

*Postmortem Findings*

Examination 24 hours after death showed a fairly well-nourished but slightly dehydrated and cyanotic Bantu male infant with a colostomy over the middle of the abdomen. Next to the colostomy opening a sausage-shaped hernial sac could be seen hanging over the abdomen. The hernial sac contained approximately an 8-inch segment of congested colon. The imperforate anal canal (± 2 inches) was confirmed and further congenital anomalies, viz. atresia of the left ureter with hydronephrosis, opening of the left jugular vein into the left atrium and an accessory toe on the left foot, were also noted.

The only other significant gross findings were confined to the lungs. There was no pleural reaction or fluid. The cut surfaces of both lungs showed an identical picture, viz. small areas of collapse at their bases and small nodular whitish areas, 1-3 mm. in diameter and more noticeable near the bases of the lungs. The intervening lung tissue was firmer than normal. The bronchi were normal and no pathological changes were noted in the hilar and tracheobronchial lymph glands.

*Microscopy*. Sections of the lungs showed the presence of a patchy interstitial infiltration of numerous plasma cells, some lymphocytes and large mononuclear cells (Fig. 1). The alveoli and alveolar ducts in the affected areas were filled with a faintly eosinophilic, slightly granular material (Fig. 2), which in some areas acquired a distinct honeycomb appearance. Closer inspection revealed the latter areas to

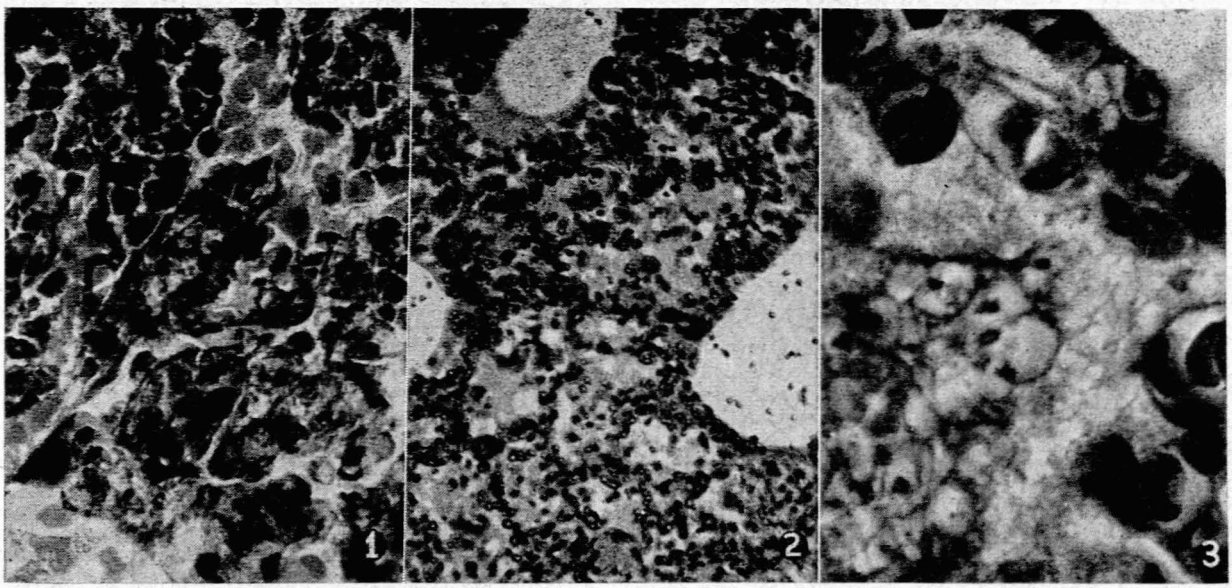


*Fig.* 1. Section of lung tissue showing interstitial infiltration of inflammatory cells. H & E × 40.
*Fig.* 2. Eosinophilic granular material filling many of the alveoli in the affected areas. H & E × 400.
*Fig.* 3. Parasitic cysts with nuclei in one of the alveoli. H & E × 900.

be composed of small parasitic cysts which varied in size and in many of which small nuclei could be seen (Fig. 3). The mucous capsules of these protozoa stained strongly positive with the periodic-acid-Schiff technique. In addition the parasites were also seen in large alveolar phagocytes and in occasional giant cells lying free in the alveoli. Parasites were also present in phagocytes in some of the smaller bronchioli. There was no evidence of inclusion disease or parasites in the other viscera.

## DISCUSSION

The various clinical and pathological findings in pneumocystis pneumonia have frequently been discussed in detail over the past few years and, therefore, only the salient features will be reviewed.

*Geographical distribution* The disease now seems to have a world-wide distribution, but with the highest incidence still in some of the European countries especially Switzerland, Czechoslovakia, Germany, Finland, Austria and Poland. In these countries the disease has on occasion assumed epidemic proportions in hospital wards.

*Clinical course.* The condition usually starts clinically as a common cold and, in fatal cases, the patients rapidly develop dyspnoea, cyanosis, loss of appetite and die from terminal asphyxia in from a few days to 3 weeks. The majority of cases, however, apparently recover slowly.

There is usually little or no temperature, but a mild leucocytosis has been observed in many cases. Clinically however, there is a well-marked discrepancy between the striking radiological changes and the absence of physical symptoms in the chest. No absolute diagnostic laboratory methods have as yet been devised, but Vivell[13] and Navratil *et al.*[14] have claimed good results with complement-fixation tests using lung extracts of proven cases.

*Predisposing factors.* The disease shows a marked predilection for premature and malnourished infants although occasional cases have been described in adults.[15-18] Practically all these adult cases have had some associated disease of the reticulo-endothelial system and, because of this and the presence of agamaglobulinaemia in some of their cases, Bird and Thomson[6] have suggested that a diminution of gamma globulins also plays a predisposing role. Both sexes are equally affected and there is no seasonal incidence. Baar[5] believes that infection of the patients with the salivary-gland virus may render them more susceptible to infection with *Pneumocystis carinii.*

*Aetiological agent.* The majority of workers accept the protozoal aetiology of this type of interstitial pneumonia. Vanek *et al.*[19] have described the parasite as a round or oval mass of chromatin 1-3 $\mu$ in diameter and enclosed in a mucous capsule 5-10 $\mu$ in diameter. Reproduction apparently takes place by division into 2, 4, 6 and finally 8 of these spore-like structures in a cyst wall. Absence of suitable test animals and failure to culture the parasites have, however, resulted in differences of opinion on the developmental stages of the parasites and their exact classification. The parasites are also found in the lungs of cats, rabbits, rats, mice, sheep, goats and guinea pigs, where they do not seem to give rise to any lesion.

*Pathological findings.* Macroscopically, the lungs of the fully developed cases are usually enlarged, heavy and airless, except for areas of compensatory emphysema near the anterior borders. On section the surfaces are moist and solid and the colour may vary from pale red to light yellow or grey. The hilar lymph nodes are apparently not affected. Microscopically the lesions are well demonstrated in the present case and need no further description.

*Therapy.* Practically all the known therapeutic measures have been tried without any real success.

## SUMMARY

The first South African case of *Pneumocystis carinii* pneumonia is described.

The salient clinical and pathological features are briefly reviewed.

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