MULTIPLE SEROSITIS IN LEPROSY[1]
REPORT OF A CASE

By Y. Y. Ying, M.D., D.T.M.

National Medical College, Shanghai.

Multiple serositis, or the so-called Pick's syndrome, was first described by van Deen (2) in 1846. A number of cases was subsequently reported by various authors. Curschmann (1), in 1884, studied the condition from the pathological viewpoint and named it "Zuckergussleber." Not until Pick's report in 1896 (6), however, did it begin to attract much attention. Since then the condition has been more commonly called "Pick's disease" or "pericarditic pseudocirrhosis of the liver." A thorough review of the literature, with a collection of cases, was made by Kelly (4) in 1903, and this has been a very useful reference to those who are interested in the subject. The disease does not seem to be extremely rare, for Reed (7) found 15 cases (0.4 percent) in a review of 3,900 autopsies at the Massachusetts General Hospital.

The exact etiology of the condition is still very poorly understood and has occasioned considerable discussion. The medical profession at present is inclined to accept either of the following factors, or a combination of them, as the cause of this syndrome: (*a*) An infection by an organism that is of low virulence but capable of provoking marked fibrosis in a serous membrane in the event of lowered resistance, inherent or acquired. (*b*) A toxin of unknown nature and source. Various diseases such as tuberculosis, rheumatic infection,

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syphilis, typhoid fever, *B. coli* infection, pertussis, malaria, and gall-bladder diseases have from time to time been held to be etiological factors in some of the cases. The importance of tuberculosis has been particularly emphasized by various authors. McWeeney (5) considered attenuated human or bovine organisms as the causative agent. Owing to the frequent association with chronic nephritis, Hale-White (3) suggested the possibility of an unknown toxin as the etiological factor. Cultural proof was lacking in most of the cases.

Literature concerning leprosy as a possible etiological factor of multiple serositis seems to be extremely scarce, if there is any at all; no particular reference to it has been found in any of the available publications. Very recently a case of advanced leprosy with Pick's syndrome came to my notice. As *My. leprae* may possibly be an important contributing factor in the causation of polyserositis in this patient, a report of the case may not be out of place.

CASE HISTORY

HOSPITAL NO. 1844. The patient, female, age 19, single, factory worker by occupation, was admitted to the hospital complaining of abdominal distention, edema and multiple ulcers of legs, cough and orthopnea of three and one-half months' duration. About 11 years ago she began to notice anesthetic patches on the upper and lower extremities, loss of eyebrows and erythematous puffiness of the face. Though the condition has steadily progressed of late, she has continued her work. One year ago she had cervical adenitis that suppurated and resulted in the formation of chronic discharging sinuses. Family history negative for tuberculosis; the patient had never had chronic cough or hemoptysis. About 6 months ago she began to have irregular fever and impairment of health, and the conditions noted on admission developed.

Physical examination revealed advanced mixed leprosy, discharging sinuses at the neck, pericardial and right-sided pleural effusion, marked ascites, marked enlargement of liver, edema and multiple shallow ulcers of both legs, and a perforating ulcer of the right big toe. The temperature ranged from sub-normal to 39°C., the respiration was increased and orthophetic, and the pulse rate was accelerated.

Laboratory findings: Erythrocytes 4,500,000, hemoglobin 75 percent; leucocytes 9,100 to 11,000; polymorphonuclears 79 percent, lymphocytes 19 percent, large mononuclears 1 percent, eosinophils 1 percent. Wassermann negative twice, Kahn positive once and weakly positive once. Urine repeatedly negative. Feces contained *Entamoeba coli* cysts and *Trichuris* ova. Sputum negative for tubercle bacilli.

A total of 3,950 cc. of clear yellowish pleural fluid was obtained (three tappings); specific gravity 1,015-1,016, cell count 450 to 1,890 per cu. mm., lymphocytes predominating (89-100 percent); no microörganisms in smears; guinea-pig inoculation positive for tuberculosis. Abdominal puncture gave 3,200 cc. of clear

yellow fluid of specific gravity 1,014, with 790 cells per cu. mm., lymphocytes predominating. Wassermann reaction and bacterial culture negative. Chemical analysis showed 1.74 percent albumin and 1.5 percent globulin. Smear of the sediment contained no microörganisms. Pericardial fluid (330 cc.) was clear, yellowish, specific gravity 1,015, cell count 300 per cu. mm., lymphocytes 86 percent, albumin 1.33 percent, globulin 0.42 percent. Numerous acid-fast bacilli were found in smear, some in clumps and some scattered. Guinea-pig inoculation negative. Skin section histologically leprosy. Nasal smear positive for *My. leprae*. Smears from the discharging sinuses and leg ulcers negative for acid-fast organisms.

Remarks.—There is no doubt that the patient has advanced leprosy. It is equally clear, as indicated by the effusions in all three of the serous cavities and marked enlargement of the liver, that she is also suffering from multiple serositis. The question of the probable causative factor of the multiple serositis in this case naturally arises. The fluids obtained from the cavities were obviously all of the nature of transudates, as indicated by their specific gravity and protein contents. Because the guinea-pig inoculation with pleural fluid was positive for tuberculosis, one is justified in considering that condition as the contributing factor. The matter is complicated by the finding of numerous acid-fast bacilli in the pericardial fluid which did not infect a guinea-pig. As a rule tubercle bacilli are so few in the serous transudates, even in the presence of extensive lesions, that their presence cannot be demonstrated without resorting to animal inoculation. Considering the number and morphology of the organisms found in the pericardial fluid, and the negative guinea-pig inoculation, it is quite reasonable to regard these organisms as leprosy bacilli, probably set free suddenly by rupture of a leproma of the pericardium. While one would naturally hesitate to draw any conclusion as to whether tuberculosis or leprosy was the chief offender, there are grounds for assuming that *My. leprae* did play an important role in this case, and it may be expected to do the same in other instances among lepers.

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