

Age specific incidence of antibodies.

(16.3%) and West Nile (17.4%) than between tanapox and Chikungunya (6.8%), and there are similarities between tanapox and West Nile in sex, tribe, and age specific incidence. The distribution of antibodies in the villages showed the same similarities except that tanapox is absent from Kipao. Ten sera showed antibodies to two viruses but there was no evidence that this was due to anything other than chance.

Discussion

The examination of sera collected from the inhabitants of the Tana River valley in 1971 showed that 16.3% had neutralizing antibody for tanapox virus in their sera. The levels of

antibody found in comparison with those observed in sequential samples of sera from persons who had clinical infection with tanapox virus in a primate centre in America, and the presence of antibody in two persons under 10 years of age in the Tana River valley, indicate that tanapox infection has continued to occur in that area since the recorded outbreak in 1962. Examinations of sera from vervet monkeys in Ethiopia and Kenya have shown that 15 to 20% contain antibody to tanapox virus (Downie, unpublished observations) and it seems likely that monkeys form the reservoir from which the inhabitants of the Tana River valley become infected. The method of transmission is unknown. Monkeys are not eaten in this area but are sometimes caught and kept as pets. It is unlikely, however, that handling of this nature would maintain such a high level of infection. Transmission by biting insects is a possibility and certain similarities in the distribution of antibodies to tanapox and West Nile viruses in the local population supports the suggestion that a culicine mosquito might be the vector.

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MEDICAL MEMORANDA

Strongyloidiasis of Respiratory Tract Presenting as "Asthma"

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The nematode parasite *Strongyloides stercoralis* is familiar as a common parasite of the intestinal tract, especially in tropical and subtropical areas.

It shares with some other intestinal nematodes the ritual of passing through the respiratory tract during its stage of migration after entry into the body, before achieving full maturity in the intestines. It is not generally appreciated, however, that it may settle down in the respiratory tract, mature, and "produce progeny" there (Fülleborn, 1914; Faust, 1935). The manifestations of respiratory strongyloidiasis need to be more widely known, and the possible consequences of inappropriate treatment when the correct diagnosis is missed deserve emphasis.

We report a case of pulmonary strongyloidiasis presenting

with wheezing and chronic cough which had been treated as asthma before the condition was recognized.

Case Report

A Nigerian girl aged 15 complained of chronic cough and wheezing of relatively sudden onset of six months' duration. She had recently been receiving treatment without improvement for asthma and later for suspected tuberculosis.

There was no family history of asthma or tuberculosis. On examination, she was breathless and wheezed audibly, but otherwise looked well and was afebrile.

Chest examination showed scattered rales and rhonchi more noticeable on the left side than on the right. No other abnormal signs were noted.

The chest radiograph was normal and the Mantoux tuberculin test was negative.

Microscopical examination of unstained sputum and centrifuged deposits showed large numbers of strongyloides larvae, and ova in various stages of embryonation (see fig. 1). All larvae were of the rhabditiform type, measuring 150 by 20 microns and showing the characteristic short narrow buccal cavity and club-shaped oesophagus followed by a bulbous dilation (fig. 2). No filariform larvae were seen.

The stool showed some ova of hookworms, but no strongyloides larvae or ova. The urine was normal. Haemoglobin was 12.5 g/100 ml, W.B.C. 10,300/mm³. The differential leucocyte count showed an eosinophilia of 23% with 34% neutrophils and 42% lymphocytes.

She was treated with thiabendazole (25 mg/kg) for two days. This produced no improvement in symptoms or in sputum parasite content. She was then treated with diethylcarbamazine (Hetrazan) 12 mg/kg/daily for 18 days. The wheezing discontinued, and the cough stopped from the fourteenth day of treatment. Sputum

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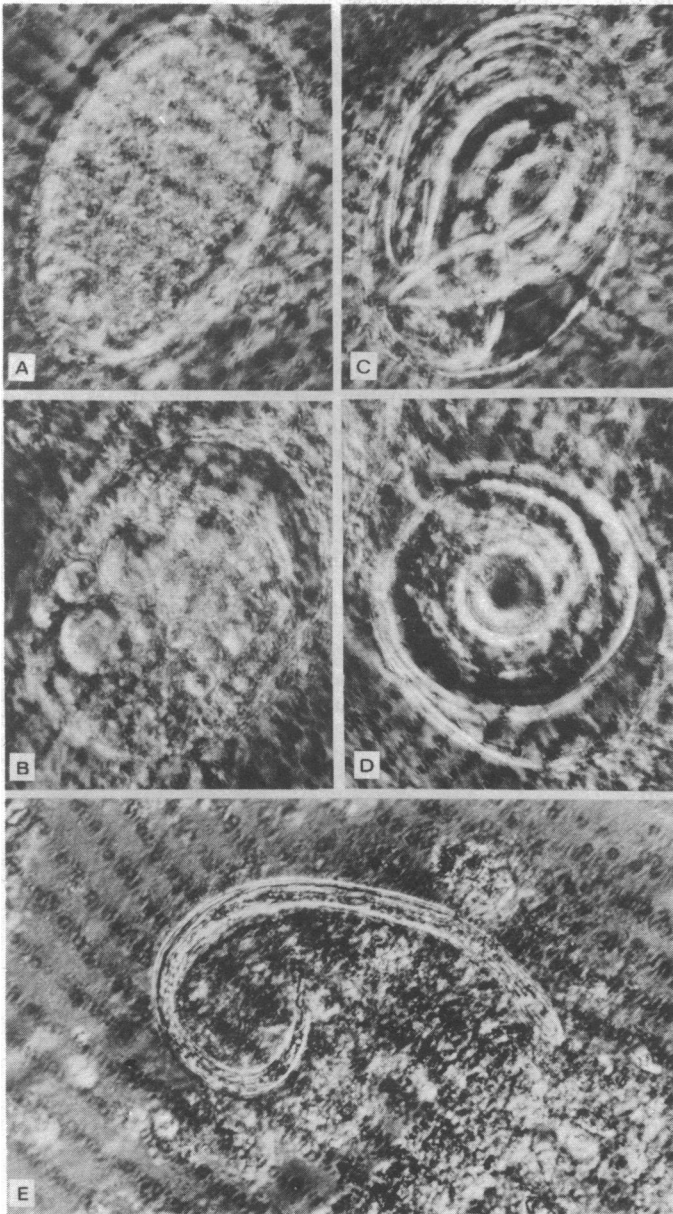


FIG. 1—Ova of *strongyloides* in various stages of development with mature rhabditiform larva and cells in surrounding medium (sputum specimen). A. Undeveloped ovum. ($\times 586$.) B. Developed ovum. ($\times 610$.) C. Ovum with mature larva. ($\times 586$.) D. Ovum with mature larva. ($\times 610$.) E. Mature rhabditiform larva surrounded by cells in the sputum. ($\times 578$.)

examination on the thirteenth day showed only 0-2 larvae and no eggs per low power field. She was then discharged, but treatment with diethylcarbamazine was continued for seven days more.

When seen eight weeks later, she looked well, but had a slight cough without wheezing. *Strongyloides* eggs were again present in the sputum and there were 1-3 larvae per low power field. She was then given further thiabendazole treatment (25 mg/kg daily) for two weeks in an attempt to eradicate the parasite completely.

At the time of writing she had not reported for a follow-up examination, and efforts to trace her have been initiated.

Comment

Strongyloides larvae may rarely appear in the sputum under the following three conditions:

(1) Filariform larvae (second stage migratory larvae) may appear during the course of the initial migration from the skin or buccal mucosa through the lungs to the intestines (Faust *et al.*, 1970).

(2) In autoinfection (Fulleborn, 1914, De Paola, 1961)—



FIG. 2—Mature rhabditiform larva of *strongyloides* surrounded by cells in sputum. ($\times 3,180$.)

for example, in severe debilitating diseases or malnutrition or during corticosteroid or other immunosuppressive therapy in a patient already suffering from intestinal strongyloidiasis.

(3) Rhabditiform larvae (first stage non-migratory larvae) together with *strongyloides* eggs may be coughed up in the sputum persistently in the situation in which for some inexplicable reason filariform larvae mature, and multiply in the lungs instead of proceeding to the intestines (Faust, 1935). The present patient belonged to this group.

Treatment in this case with standard doses of thiabendazole was unsuccessful and diethylcarbamazine had to be substituted with some apparent benefit. Seabury and Savoy (1971) had a similar unfavourable experience with thiabendazole given for three weeks in a fatal case of pulmonary strongyloidiasis in a 70-year-old man.

Fortunately, our patient did not appear to have received corticosteroids at any time for her presumed asthma. This might have aggravated her condition. This is because there is evidence that a relatively harmless *strongyloides* parasite can be converted into a dangerous pathogen with pronounced invasive tendencies by the administration of steroids or other immunosuppressive drugs to the host (Willis and Nwokolo, 1966; Rivera *et al.*, 1970; Fagundes *et al.*, 1971).

Physicians who treat asthma often add corticosteroids to their antiasthmatic regimens whenever standard therapy is unsuccessful. This practice could be dangerous if the "asthma" is associated with pulmonary strongyloidiasis and steroid therapy is prolonged. Because of this, it is suggested that asthmatic patients from areas where strongyloidiasis is endemic should have sputum examinations for *strongyloides* before they are subjected to a course of steroids, most especially where an eosinophilia is found in the blood.

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