AMMONIA METABOLISM IN PULMONARY HEART DISEASE

By Aaron Valero, Gideon Alroy and Joseph Izkowitz

Confusion delirium and coma in pulmonary heart failure has been ascribed to hypoxia or cerebrospinal acidosis caused by respiratory failure. Since the clinical manifestations resemble those seen in hepatic encepalopathy, this study was undertaken to investigate the role of ammonia intoxication in the production of the neurological signs in pulmonary heart disease. Glutamine and NH_3 in the CSF, ammonia in arterial and venous blood were determined con-committantly with acid base balance and PO_2 in arterial blood and CSF.

Comparison was made between a control group (10 cases), alert hypercapnic (10 cases), drowsy hypercapnic (6 cases), and drowsy cirrhotic (6 cases).

It was found that the drowsy hypercapnic patients were those who had advanced liver congestion due to right sided heart failure. No statistical difference was found between the two hypercapnic groups in terms of pH, PCO_2 or PO_2. The glutamine in the CSF and blood ammonia leve's, however, were found significantly higher in the drowsy patients.

It is concluded that the neurological manifestations seen in pulmonary heart disease associated with respiratory failure, are caused mainly by ammonia intoxication resulting from liver congestion.

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PULMONARY HYPERTENSION WITH SPECIAL REFERENCE TO FILARIASIS AND ITS MANAGEMENT

By Ivor Obeyesekere

'Primary' pulmonary hypertension, which is rare in Western countries was found to be relatively common in Ceylon. There were many unusual features seen in Ceylon which suggested a filarial aetiology.

The clinical picture, results of special investigation, natural history and pathogenesis is described on the basis of 65 patients investigated in the Cardiology Unit, General Hospital, Colombo.

There were two distinct types, malignant and benign. Patients with the malignant type had a rapidly progressive illness of short duration and an invariably fatal outcome. Those with the benign form gave a long history, and, inspite of severe pulmonary hypertension were only slightly disabled. They appeared to tolerate the disease better. An important factor which determined the clinical course of the disease was the patency of the foramen ovale. This appeared to act as a safety valve permitting right-to-left shunt in times of stress. The therapeutic implications of this is discussed. A conspicuous feature was the severity of the pulmonary hypertension which was severe in every patient. One patient had the extremely high pressure of 220/112 mm Hg.

The mean absolute eosinophil count was 1790 per cu mm. This was significantly higher than in age-matched, sex-matched patients suffering from chronic rheumatic heart disease (p=0.25-0.01) and congenital heart disease p=(0.01-0.005). The raised eosinophil counts in these patients was not due to intestinal parasites (Obeyesekere and Soysa 1970). None of the established causes of a raised eosinophil count were present. Eight patients had counts over 3,000 per cu mm, levels generally associated with tropical pulmonary eosinophilia (T.P.E.). However, they did not have any of the clinical or radiological features of T.P.E. But, very similar to patients with T.P.E., the high eosinophil count dropped dramatically in response to diethyl carbamazine—an anti-filarial agent.

Eight patients gave a previous history of T.P.E. and five of clinical fi ariasis. The Filaria Complement Fixation Test was positive in 60% (9 out of 15), Fluorescent Antibody Test in 33% (8 out of 24) and the Intradermal Skin Test positive in all patients tested (10 out of 10).

A lymph node biopsy done on a female patient suffering from pulmonary hypertension and hypereosinophilia (11,525 per cu mm) demonstrated filarial worms with extensive eosinophilic infiltration of both the lymph gland and lung tissue (lung biopsy section). A male patient aged 35 years with pulmonary hypertension and hypereosinophilia (6075 per cu mm) had a nodule in his epididymis. Biopsy demonstrated filarial worms and surrounding eosinophilic infiltration.

Epidemiology. W. bancroft is endemic along the South Western coastal border of the island with pockets in some inland towns. 61 of the 65 patients lived in these endemic areas, the remaining four lived in areas close by.

During the acute phase of filariasis, microfilariae are liberated by the adult worm and find their way into the pulmonary circulation. It is therefore conceivab e that the microfilariae embolise the pulmonary capillaries setting into motion pulmonary hypertension in the same manner that minute pulmonary emboli consisting of blood clot do. The hypereosinophi ia could well be the result of an antigen antibody response to the dead microfilariae.

Since fi ariasis is endemic in certain areas in Ceylon, this might account for the high incidence of this disease, where unlike in the West it is almost as common in the male as it is in the female.

The management of pulmonary hypertension is discussed.

REFERENCE

Obeyesekere, Ivor and De Soysa, N.: "'Primary' Pulmonary Hypertension, Eosinophilia and Filariasis in Ceylon." British Heart Journal, 32,524-536, 1970.