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REPORT OF CASES

Pulmonary Hypertension, Hypereosinophilia and Filariasis

by

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Abstract

Reports have been made on three cases of pulmonary hypertension, hypereosinophilia with filariasis as the possible cause. Considering the Indonesian geographical location, it would be reasonable enough to perform further studies on hypereosinophilia cases. The possibility of filarial infestation is to be taken also into consideration. This condition should be treated as soon as possible to prevent or to slow down the consequences of continuing cardiovascular changes.

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Introduction

Filaria is a nematode parasite; its adult form lives in the lymphatic organ and usually causes a disease known as elephantiasis (Oemijati, 1968). The larvae, called microfilariae, are periodically released into the bloodstream, circulating to the lungs, liver, heart, etc. (Webb et al., 1960; Danaraj et al., 1966). Clinically, microfilariae do not always produce any symptom, sometimes only fever exists, but they can also create a more severe problem known as tropical eosinophilia, indicated by pulmonary symptoms, like frequent cough and wheezing, especially during the night with a high intensity of eosinophils in the blood.

Microfilariae are rarely present in the peripheral blood (D'Abrera, 1958), but a skin test against filaria is mostly positive. Many reports about cardiovascular changes in tropical eosinophilia were published. Aubertin and Gibrous (1921) found congestive heart failure in patients with hypereosinophilia and atherosclerosis of pulmonary artery. Jhatakia (1946) and Vismanathan (1963) found precordial pain in several patients with tropical eosinophilia. D' Abrera (1958) and Vakil (1961) stated that tropical eosinophilia was accompanied with rheumatic heart disease, and deteriorated the existing heart disease.

Congestive heart disease, cor pulmonale, and electrocardiographic changes in tropical eosinophilia were also reported by Johny and Ananthachara (1965). Menon (1963) reported on patients who recovered from congestive heart failure after being treated with diethyl carbamazine, known as a drug against filariasis. Obeyesekere and De Soysa (1970) associated pulmonary hypertension with filariasis and in 1974 Obeyesekere and Doris reported 2 cases of pulmonary hypertension where adult filaria were found.

This report concern 3 cases of pulmonary hypertension with hypereosinophilia. In one case microfilariae were found, while in the others filariasis was suspected due to a positive skin test against filaria.

Case report

* *Case One*

N., a 3-year-old Indonesian girl, was treated at the Pediatric Outpatient Department of the Cardiac Center in early December 1972. The chief complaints were frequent cough and laborous breathing, particularly during the night, which had lasted for one month. She was the youngest of 3 siblings in the family. The history of mental and physical growth was normal. No evidence of allergy, pulmonary tuberculosis, and rheumatic fever was detected. There was no history of cyanosis.

On physical examination her body weight was 14.1 kg. She looked rather pale and showed slight cyanosis. Her

blood pressure was 100/70 mm. Hg, her heart and pulse rate were equal, 100/minute. Jugular venous pressure was not increased. An increase of the right heart activity without thrill could be felt. Pulmonary second sound was closely split non-fixed, with a loud second component. An ejection systolic murmur grade 2/6 was heard over the pulmonary area. Both lungs were normal. Chest X-ray showed enlargement of the heart to the right and left, pulmonary artery segment was prominent with a slight paracardial infiltration. Electrocardiogram recorded sinus rhythm, right axis deviation, right atrial enlargement, and right ventricular hypertrophy.

Blood examination showed a hemoglobin level of 10.5 gm.%, total white blood cell 13,200/mm^3, and absolute eosinophil 6,468/mm^3. Blood sedimentation rate (Wintrobe) was 30 mm. in the first hour. Tuberculin test was negative. Negative LE cell, ASTO, titer was 100 TU. The skin test against filaria was strongly positive. No microfilariae were seen in the thick films prepared from capillary blood smear or from arterial blood collected during right heart catheterization. Liver biopsy was performed; the result showed non-specific reaction with hydropic degeneration and infiltration of monomorphonuclear and polymorphonuclear cells at the portal areas. Some of the cells were eosinophils. Cardiac catheterization and angiography confirmed the diagnosis of pulmonary hypertension. There was no intracardiac shunt. Unfortunately, she died 7 hours after the procedure was done.

* *Case Two*

A., a 10-year-old Indonesian male child, the third child of 4 siblings, was sent to the Outpatient Department of the Cardiac Center, on December 12, 1973, due to an enlargement of the heart and the presence of heart murmur. Mental and physical growth were normal, never suffered from any previous significant illness, neither rheumatic fever, congenital heart disease, nor allergic disease. The child was in a good nutritional state (weight 27 kg.), but looked rather pale. The blood pressure was 100/70 mm.Hg, pulse and heart rate 100/minute, and respiration rate 16/minute. Right heart activity was felt to be slightly increased without thrill. Pulmonary second heart sound was closely split with a loud second component. An ejection systolic murmur with a punctum maximum on the second left intercostal space was heard. Lungs were clear; liver and spleen were not palpable.

Chest X-ray showed an enlargement of the heart to the left and to the right side. The hili were enlarged with infiltration of the lung in the right paracardial region. Pulmonary vascular markings in the upper and middle lobe were prominently increased. Electrocardiogram recorded sinus rhythm, intermediate heart position with evidence of incomplete right bundle branch block. Blood examination showed a hemoglobin content of 9.5 gm.%, and white

blood cell of 8,200/mm^3; absolute eosinophil was 1,968/mm^3. Blood sedimentation rate was 14 mm in the first hour. Microfilariae were found in the blood. Cardiac catheterization revealed no intracardiac shunt; right ventricular pressure was 35 mm. Hg and pulmonary artery pressure 40/15 (high normal). Diethyl carbamazine was the drug of choice for filariasis and the second laboratory examination showed less eosinophils in the blood picture.

* *Case Three*

E.M., a 9-year-old Indonesian female child, the eighth child of 10 siblings, was admitted to the hospital on April 1972 due to frequent coughing and shortness of breath and heart murmur. Mental and physical growth were normal. Neither cyanosis nor allergic disease, lung tuberculosis, rheumatic fever could be derected. One month prior to admission the child suffered from fever, cough, and paroxysmal wheezing during the night. She was treated with antibiotics and symptomatic drugs, but with no improvement.

On examination, she was an undernourished child, weak, pale, and dyspnoeic. Her blood pressure was 100/70 mm.Hg, pulse and heart rate 60/minute, rather deep but no cyanosis. Jugular venous pressure was normal. Right heart activity was increased. Second heart sound was closely split, with a loud pulmonary component. An ejection systolic murmur grade 2/6 was heard over the pulmonary area. Gallop rhythm was audible. There were moist and dry rales in both lungs and also wheezing. The liver was palpable 2 cm. below the costal margin, firm without tenderness. Thorax X-ray showed enlargement of the heart to the right and left side, the apex reaching the thorax border. Pulmonary artery segment was prominent. The diameter of the blood vessels, particularly on the hilar region, was enlarged and there was an increase in vascular marking; while in the periphery it became smaller and decreased. Electrocardiogram recorded sinus tachycardia, right atrial enlargement, right ventricular hypertrophy, and complete right bundle branch block.

Laboratory findings showed a hemoglobin level of 12.5 gm.%, total white blood cell 18,800/mm^3, and absolute eosinophil 3,008/mm^3, ASTO titer was 100 TU and tuberculin test was negative. No filariae were seen in thick films prepared from capillary blood or from arterial blood collected during right heart catheterization. Skin test against filaria was positive. Pulmonary hypertension with heart failure was considered. Tropical eosinophilia was suspected. Digitalization had been given, but the result was not satisfactory. She was taken home because her father refused to let her stay in the hospital any longer.

After one year she returned to the hospital in a deteriorated condition. She was easily exhausted. On auscultation diastolic murmur was audible. Total

eosinophil count was 18,876/mm^3, total white blood cell 28,660/mm^3, and hemoglobin level 11,2 gm.%. Right heart catheterization was performed which revealed an increase in right ventricular and pulmonary artery pressure. There was no pulmonary-ventricular pressure gradient. There was also no intracardiac shunt, but the catheter could pass the foramen ovale to the left atrium. Liver biopsy revealed widening of the sinusoid and central vein and infiltration of the cells, particularly eosinophil cells. No signs of hepatic cirrhosis could be found.

After filarzan was given for 2 periods of five days, improvement was noticeable. The cyanosis, cough, and dyspnoeic respiration disappeared. Blood test showed an eosinophil decrease of 20%. On further family study, micro filariae were detected in four other members of the family that lived in the same house.

Discussion

This report concerns the relation of filarial infestation and cardiovascular changes in Indonesia; while in Malaysia, India, and Ceylon the hemodynamic and pathology anatomy had already been discussed previously. With regard to the geographic location of Indonesia, which is in the neighboring areas of these countries and known as one of the filarial endemic area, it is worthwhile to give more attention to the study of this disease. The hazard caused by this parasite is not only elephantiasis, but also serious conditions such as pulmonary hypertension, heart failure, etc., which may be fatal in severe cases. The developing pulmonary hypertension in filariasis has been proved on animal studies, as reported by Dissanaike and Paramanathan (1961) who found a new type of filaria, the Brigia (Brugiella) buckleyi on Lepus nigricollis singhala (wildhare). They also live in caninens, as reported by Detweiler et al. (1960) and Patterson and Luginbuhl (1963). Smith and Jones (1957) described that vascular changes in canines' lungs have the same characteristic as pulmonary hypertension in human.

Comparing the forementioned cases with those compiled by Obeyesekere and De Soysa (1970) and Obeyesekere and Doris (1974), it was found that both pulmonary hypertension and tropical eosinophilia patients had antibody against filaria, but microfilaria in the blood was mostly not found. On the contrary, in our symptomless case, when pulmonary artery pressure was still 40/15 mm. Hg, microfilaria in the blood was positive. In the third case pulmonary hypertension was neither caused by congenital heart defect (R-L shunt), nor by lung processes, since the lung function was within normal limits. It was most likely caused by filarial infestation, confirmed by the existence of microfilaria in other members of the family and by the fact that the family had lived in the filarial endemic area.

The possible deteriorating process of filarial infestation from the early stage as in the second case to the late stage as in the other 2 cases has been considered. In a severe condition, cardiac catheterization and angiocardiographical risk are considerably high, as in the first case; death occurred after those procedures due to the accumulation of contrast in the lungs, causing severe hypoxia. A similar condition was also reported by Obeyesekere and De Soysa (1970) and Obeyesekere and Doris (1974). The total blood eosinophils is a useful parameter to make further investigations on the possibility of filarial infestation in patients with pulmonary symptoms, which may have serious consequences if the process continues.

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