CHAPTER 15

CHAGAS' HEART DISEASE

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1. INTRODUCTION

Chagas' disease is a protozoan infection caused by Trypanosoma cruzi and is mainly acquired by vectorial transmission in endemic areas, when infected excreta of Triatominae bugs are inoculated into the sting site or neighbouring mucous membranes of the victim. The insect vector acquires the disease from feeding on infected animals (wild and domestic) and harbours the parasite in the gastrointestinal tract. Less common mechanisms of transmission include transfusion, congenital transmission, organ transplantation, laboratory accident, breast feeding, or oral contamination (1). Multiplication of the parasite through its amastigote form is obligatory in the intracellular milieu. It initially occurs within cutaneous macrophages, followed by rupture of the infected cells and subsequent release of trypomastigote forms in the blood stream, and then invasion of remote sites (2). Parasite multiplication may occur in every tissue, but neat predilection is observed towards the myocardium, skeletal and smooth muscle, and the nervous system. When transmitted by the insect vector, skin swelling produces the typical entry lesions known as chagomas (3).

2. EPIDEMIOLOGY

The true prevalence of Chagas' heart disease is unknown (4). A review of serological surveys for Chagas' infection among blood donors conducted during recent years in several countries on the American continent disclosed a highly variable rate of prevalence, from 0 % to 63 %, with an average of 3 % (5). Only limited cross-sectional epidemiological studies have been carried out in some countries. This yields widely variable prevalence of both morbidity and mortality even within each country (6), probably because of marked variations in the genetic background, parasite strain, climate, social and economic situation and related hygienic and alimentary conditions, and health care measures. Despite those limitations, it is well recognised that Chagas' disease is a major public health problem in endemic areas of 18 South American countries, where global human infection prevalence estimates are in the range of 16 to 20 million, with another 70 to 90 million people being considered at risk (7).

The main manifestations of disease include: megaoesophagus, megacolon, and cardiac involvement, by far the most serious of all. The prevalence of digestive
involvement is highly variable even among endemic regions; some surveys in central Brazil show an average rate of 6 - 8 %. Chagas' myocarditis is the most common form of cardiomyopathy in Latin American countries. It is estimated that over 750 thousand years of productive life are lost annually, because of premature deaths due to this disorder.

Formerly considered a rural disease, Chagas' disease is now ubiquitous because of conspicuous changes in social patterns in most countries, mainly as a result of rural-urban migration. Chagas' disease also occurs in non-endemic areas where it may be acquired by blood transfusion (8). A serologic study in 205 Latin American immigrants to the United States found a prevalence of \( T. cruzi \) infection of 4.5 %. This leads to an estimate of at least 400,000 to 500,000 infected people now living in the United States (7).

Prevention has been manly achieved by improving the quality of housing and the use of residual pesticides to suppress home transmission. Good results have been reported from some countries, and constitute a highly cost-effective public health policy. However, widespread application of such programs is hindered by financial limitations. It is also necessary to prevent transfusion transmission in both endemic and non-endemic areas. Etiologic treatment of Chagas' disease is well indicated for the acute cases, arguable for patients with recent infection (e.g. children) and has no demonstrated scientific proof of effectiveness to change natural history when the clinical syndromes are fully established (9).

3. CLINICAL FORMS AND NATURAL HISTORY

Chagas' heart disease presents two different phases, acute and chronic. The long period (10-30 years) between the acute condition and the appearance of clinically manifest chronic Chagas' heart disease is known as the indeterminate form of the disease and constitutes one of its most intriguing features. There is no clarification as to why approximately 50 % of chagasic patients do not evolve from the indeterminate phase to the serious clinical manifestations of chronic heart involvement throughout their lives (3, 10). In the chronic phase, common symptoms include fatigue, exertional dyspnea, palpitation, dizziness, syncope and chest pain. Such symptoms indicate curtailment of the cardiac reserve (including minor early signs of diastolic dysfunction), or the presence of ventricular dysrhythmias, and of atrium-ventricular block. Chest pain is most commonly atypical for myocardial ischaemia, but may mimic an acute coronary syndrome (11). Sudden unexpected death may occur even in previously asymptomatic patients. It is usually precipitated by physical exercise, and its mechanism is typically ventricular fibrillation, with complete atrium-ventricular block remaining as secondary possibility. Chagasic patients usually present electrocardiographic alterations, including conduction disturbances, ventricular arrhythmias, and abnormal Q-waves, suggesting extensive areas of myocardial fibrosis. In special, the combination of right bundle branch block and left anterior hemi-block is very typical in chronic Chagas' heart disease. Studies in endemic areas show that not more than 10 % of the acute cases have clinical manifestations compatible with the diagnosis of acute chagasic myocarditis.