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Case Report

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Rare Case of Chagas Disease

Assoc Prof. Adel Ekladious*

Faculty of Health and Medical Sciences, Internal Medicine, The University of Western Australia, Nedlands, Western Australia.

Bega- South East Regional hospital , NSW Australia

***Corresponding author**
Adel Ekladious, Faculty of Health and Medical Sciences, Internal Medicine, The University of Western Australia, Nedlands, Western Australia.
Email: ekladiou@gmail.com, Mob: 61499449905

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Abstract

Chagas is a multisystem disease transmitted from Trypanosoma cruiz, not uncommon in Latin America. This disease causes parasympathetic failure resulting in cardiac failure, arrhythmogenic, cardiomyopathy, sudden death, mega esophagus and mega colon. In this article, we present a case of chronic Chagas disease who had extensive investigation prior to reaching a correct diagnosis. We will highlight the symptomatology and treatment.

Case report

A 36-year-old lady emigrated from Mexico to America when she was 15 years old and currently resides in Australia. She is an intensive care nurse, married from a pilot and has a boy and a girl aged 12 and 15 years old. She does not smoke or drink. She takes regular laxative and proton pump inhibitor for constipation and esophageal reflux respectively. Her father died at the age of 60 and her cousin at the age of 45, both of which had sudden death without clear cause. Patient was athletic and used to run 10km on weekdays and 15km on the weekend. While she was exercising at home, she suddenly became unconscious for thirty seconds. The paramedics were called and the patient was admitted to high dependency unit for investigation and management. She denied any preceding chest pain, palpitation, cough, dizziness, giddiness, sweating, nausea, vomiting, diaphoresis, abdominal pain or shortness of breath.

On examination; pulse 70/min regular with good volume, blood pressure 120/70 with no postural drop. Temperature 37, respiratory rate 18/min, oxygen saturation was 100% on room air. Cardiac examination was unremarkable apart from wide split second heart sound. Chest and abdominal examination did not reveal any abnormalities. Examination of lymph nodes, skin, joints and muscles were normal.

ECG showed complete right bundle branch block. She did not have any old ECG for comparison, cardiac troponin was very slightly elevated, 0.5 ng/ml (0-0.4 ng/ml), D dimer 0.60 (<0.5). Other investigation included LFT, urea, creatinine, thyroid function tests, ESR, CRP, Metabolic panel were all either negative or normal.

Patient was commenced on fluids and low molecular weight heparin.

CTPA was arranged which was negative for PE. Echocardiogram was negative.

Patient was observed for two days with continuous telemetry and discharged home with follow up with her GP.

6 days later, the patient experienced a syncopal episode, 60 seconds in duration, thus readmitted to hospital. On arrival she was conscious but faecally incontinent. Physical examination was unremarkable. The patient was examined by the neurology senior registrar: normal cranial nerves, tone, strength, reflexes, coordination, superficial sensation and prospective all were unremarkable. The patient had normal gait, successful tandem gait, and nil incoordination. ECG showed old RBBB and CT head did not show any abnormality. MRI head was normal, EEG did not show any slowing, epileptiform abnormalities.

The patient was discharged home and asked not to drive for 6 weeks until her next appointment with cardiology and neurology.
Three days later, the patient experienced another syncopal episode, 45 seconds in duration. The patient was readmitted to the hospital and had a CT coronary angiography which did not show any calcium deposition - score was zero. Electrophysiological study showed ventricular tachycardia originating from endocardium in the inferior left ventricle. The patient underwent radiofrequency ablation. Cardiac MR showed late enhancement of Gadolinium which was interpreted as myocardial fibrosis. Transesophageal echocardiogram showed a very small aneurysm in the apex of the left ventricle. MIBG scan to exclude apical ballooning syndrome and pheochromocytoma revealed sympathetic and parasympathetic myocardial denervation.

In view of recurrent cardiac syncope and family history with two sudden deaths, it was agreed that the patient should have an implantable cardiac deliberator inserted. The patient did not have further syncopal episodes, however did have two electrical shocks over the first 6 months. The deliberator was programmed and

found the shocks was appropriate for ventricular tachycardia. The patient was reviewed by a cardiac electrophysiologist who started the patient on flecainide to reduce the electric shocks. Ten weeks later, the patient started to have difficulty with swallowing, which fluctuated during the day.

The patient was referred to a neurologist to rule out myasthenia gravis. Acetylcholine receptor antibodies were negative, MUCK antibodies were undetectable. MRI chest and mediastinum was negative for thymoma. Repeated nerve conduction study did not show any decremental response, thus myasthenia was ruled out. The patient had oesophageal motility studies, which reported lack of peristalsis and achalasia of the lower esophageal sphincter, elevation of the resting pressure, and increased response to stimulation of esophageal smooth muscle response to cholinergic medication (acetylcholine). Colonoscopy and multiple biopsies showed dilation and lengthening of sigmoid and rectum, and hyperactivity to acetylcholine. PCR was positive for Trypanosoma Cursi, Enzyme linked immunosorbent assay(ELISA) and hemagglutination assays was strongly positive for Trypanosoma Cursi, confirming Chagas disease.

The patient was counselled about options for treatment, and was started on benznidazole with repeat serology every 6 months including liver and kidney functions. The patient was also advised that if she planned to become pregnant, medication should be ceased. She would also require an echocardiogram every 6 months and Holter monitor twice a year. The patient seroconverted negative after one year, and advised continue medication, with ongoing follow up.

Discussion

Chagas disease is endemic in Latin America [1]. Disease is transmitted by faeces of kissing bugs via oral route or through a break in the skin of the host [2]. Blood transfusion or infected organ transplant can also transmit the disease [3]. There are many reported cases where an infection is transmitted from mother to fetus causing congenital Chagas disease [4]. Chagas disease is considered a parasitic zoonosis transmitted by Trypanosoma cursi, where the transmission to human is vector borne. It is estimated that around 8 million are infected worldwide. The disease is asymptomatic in the acute phase, and symptoms are vague and nonspecific. Migration has increased the number of infected patients around the rest of the world. The first two cases were reported by Dr Luis Mazotti in Mexico in 1940. Trypanosoma cursi, is an obligate intercellular protozoan flagellate. The vector is named Triatominae, which is responsible for its transmission. There are few strains of Trypanosoma curzi, which cause complications of variable severity. Most cases in developing countries are caused by transmission through blood transfusion or infected organ transplant. Very rarely, Trypanosoma curzi transmission can occur through occupational exposure. Chagas disease could be reactivated in immunosuppressed patients. Infection and chronicity of the diseases is mainly CD4 mediated and colonel populate CD8 [5]. Clinical symptoms are very broad, with many organ systems affected in variable degrees. Incubation period is 4-15 days, with most of patients asymptomatic in the acute phase.

Less than 5% may have pruritic skin rash, periocular oedema, conjunctival congestion, subcutaneous nodules, myalgia, arthralgia, lymphadenopathy, and hepatosplenomegaly, myocarditis with tachycardia, St Wave changes in ECG, galloping, premature atrial or ventricular beats, RBBB, pericarditis, and heart failure. Congenital Chagas does not give rise to symptoms in the acute phase. Chronic disease is very heterogeneous, characterized by involvement of the heart, which can cause sudden death due to involvement of the conduction system causing ventricular arrhythmia, myocardial fibrosis, catecholamine cardiomyopathy, apical micro ventricular aneurism, intramural thrombus, ischemic cardiomyopathy, dilated cardiomyopathy, progressive heart failure, A-V nodal disease, A-V block, thromboembolism, recurrent syncope, valvar disease. Chagas could be manifested by dysphagia, recurrent aspiration pneumonia, sever reflux and regurgitation, esophageal dysmotility, retrosternal pain due to intramural parasympathetic denervation causing hypertrophy of the muscles and loss of contractility [6-8]. Parasympathetic failure of the colon leads to dilation, impaired absorption and secretion, leading to constipation, abdominal distension, intestinal obstruction, volvulus, formation of fecalomas. Patient can have cardio embolic stroke at a young age due to atrial arrhythmia, cardiac dilation, apical aneurism and mural thrombus [9].

Diagnosis can be made on the suspected epidemiological background, history of blood transfusion in low-income countries or organ transplant, along with serology from the blood including Enzyme-linked immunosorbent assay, indirect hemagglutination, Wistron Blot test, upper and lower endoscopy and multiple biopsies, and PCR for Trypanosoma Cursi. clinicians should be a low threshold to investigate immunosuppressed patients. Micro-Strout test is not an expensive test. Where a sample of blood from the patient, the leucocyte fraction can be examined under light microscope in search for Trypomastigotes. This can be done in minimal resources laboratories [10].

Base line investigations, once diagnosis confirmed, included abdominal X ray, echocardiogram, ECG, Holter monitor, cardiac MRI, esophageal manometry. Treatment involved Benznidazole 7mg/kg/day divided into two doses. Side effects included skin rash, peripheral neuropathy, anorexia and weight loss, very rare marrow suppression, nausea and vomiting. Treatment in the acute phase may prevent the progress to chronic stage. Benznidazole is contraindicated in pregnancy [11,12]. Nifurtimox is an alternative oral medication, with an adult dose of 10mg/kg, divided into 4 doses. Common side effects are gastrointestinal irritability, peripheral neuropathy, myalgia, and neutropenia.

Conclusion

Chagas disease is very rare disease in developed countries. It rarely presents acutely. Reactivation in immunocompromised patient is not uncommon, and can present with cardiac syncope. Therefore, it should be considered in the differential diagnosis of cardiac syncope among immigrants. It is treatable in the acute phase to prevent chronic manifestation. Travel history is still of paramount importance.

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