CASE REPORT

Syncope: Clinical Presentation of Bronchial Asthma

S Williams-Phillips[1], M Tulloch-Reid[2]

ABSTRACT

Syncope, first described by Hippocrates, can be differentiated into neurological, cardiac and non-cardiac in origin, and this differentiation is of prognostic significance. The cardiac causes of syncope, which can be structural, electrophysiological or infectious, have a relatively poor prognosis and are associated with ethnicity, geographic location and sudden cardiac death. In decreasing frequency, the cardiac causes are hypertrophic cardiomyopathy, anomalous coronary arteries, Marfan Syndrome and dilated cardiomyopathy. Electrophysiological causes include supraventricular causes, Wolf-Parkinson-White syndrome, ion channelopathies, long QT syndrome and Brugada syndrome. The index case with bronchial asthma presented with syncope. There is an increased morbidity and mortality of this specific group of patients, if undiagnosed and not optimally treated; hence the need for a high index of suspicion and early diagnosis, after exclusion of cardiac and more common neurological causes. This is the first documented case of syncope secondary to bronchial asthma in an Afro-Caribbean.

Keywords: bronchial asthma, syncope

Síncope: presentación clínica del asma bronquial

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RESUMEN

El síncope, primeramente, descrito por Hipócrates, se puede clasificar como neurológico, cardíaco y no cardíaco atendiendo a su origen, y esta diferenciación tiene importancia pronóstica. Las causas cardíacas del síncope – que pueden ser estructurales, electrofisiológicas o infecciosas – tienen un pronóstico relativamente pobre y se asocian con la etnicidad, la localización geográfica y la muerte cardiaca repentina. En frecuencia decreciente, las causas cardíacas son la cardiomiopatía hipertrófica, las arterias coronarias anómalas, el síndrome de Marfan y la cardiomiopatía dilatada. Las causas electrofisiológicas incluyen las causas supraventriculares, el síndrome de Wolf-Parkinson-White, las canalopatías iónicas, el síndrome de QT largo, y el síndrome de Brugada. El caso índice con asma bronquial se presentó con síncope. Hay una mayor morbilidad y mortalidad de este grupo específico de pacientes, si no se diagnostica y no se trata de forma óptima. De ahí, la necesidad de un alto índice de suspicacia y diagnóstico temprano, luego de la exclusión de las causas cardíacas y las causas neurológicas más comunes. Éste es el primer caso documentado de síncope secundario al asma bronquial en un afrocaribeño.

Palabras clave: Asma bronquial, síncope

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INTRODUCTION

Hippocrates was the first to describe and name syncope, which, for the purpose of this case report and discussion, is defined as the transient loss of consciousness and postural tone for less than two hours with spontaneous recovery (1). The main causes of syncope are neurological, cardiac and non-cardiac. The most common cause in adolescents is orthostatic hypotension, also called neurocardiogenic syncope and has a classic history with immediate recovery when in the supine position and can be easily diagnosed with accurate history and examination (2). Excluding neurological causes, the differentiation of syncope into cardiac and non-cardiac origin is of prognostic significance as cardiac causes of syncope have a relatively poor prognosis. In decreasing frequency, the cardiac causes are usually hypertrophic cardiomyopathy, anomalous coronary arteries, Marfan Syndrome and dilated cardiomyopathy. However, in Italy, the most common cause of cardiac syncope and sudden cardiac death is arrhythmogenic right ventricle dysplasia, indicating a difference in the ethnicity and the geographic location of syncopal cardiac lesions (1–4).

Cardiac causes also include obstructive lesions on the left side of the heart and aortic stenosis where cardiac output and coronary blood flow can be severely impaired. Tumours (such as left atrial myxomas) may intermittently reduce left ventricular filling. Congenital coronary lesions leading to disruption of blood flow need to be identified from inflammatory, autoimmune, such as Kawasaki disease, and coronary flow obstruction, whether or not due to atherosclerotic plaques in familial hypercholesterolaemia (2).

There had been documentation of mitral valve prolapse with mitral regurgitation, causing syncope, with no clearly reproducible proven reasons, for this occurrence.

Electrophysiological causes, whether supraventricular causes (such as Wolf-Parkinson-White syndrome) or ion channelopathies (such as long QT syndrome and Brugada syndrome) are equally important causes, which can be concealed on an electrocardiogram (1–3).

Despite the referring physician's differentiation into cardiac causes, it can be sometimes impossible, based on obligatory detailed careful history, physical examination, chest X-ray and presumably electrocardiogram, prior to referral, to make that differentiation. Hence, the cardiologist seeing these patients needs to bear this in mind in his or her evaluation. It is very important to note social history, including the use of illicit drugs (such as cocaine and caffeinated beverages), and travel history to areas where infectious causes of cardiovascular causes such as Lyme disease are in preponderance, such as in South America (2, 3). The literature suggests an increase in prevalence of hypertrophic cardiomyopathy among Blacks and ion channelopathies (particularly the long QT syndrome) among Caucasians, reinforcing the differences in ethnicity (1–4).

Neurologic causes would be referred to the neurology clinic after careful assessment by the referring physician, but they cannot be excluded with a normal electroencephalogram and computerized axial tomography scan. In the literature, there are 10–20% of cases of syncope where no identifiable cause is found (2–4).

Cardiac causes of syncope can occur in an electrophysiological and structurally normal heart on echocardiography, on electrocardiogram, at cardiac catheterization, magnetic resonance imaging of the heart, where there are concealed accessory pathways. Hence, transoesophageal pacing and transcatheter electrophysiological studies may be needed (2, 3).

Haematological (*eg* severe anaemia), biochemical and endocrine causes (*eg* diabetes: hypoglycaemia and hyperglycaemia) of syncope are rare in the adolescent age group, without other significant symptoms, which would lead to referral to the commensurate haematology or endocrine clinic, but have been documented and need to be excluded (2, 3).

Exercise-induced asthma, and in this case causing syncope, is a condition of respiratory difficulty (bronchoconstriction) that is related to histamine release, is triggered by aerobic exercise and can last for several minutes (5–17).

CASE REPORT

The index case was a 12-year-old Afro-Caribbean female with complaints of progressively worsening and of increasing frequency (now two to three times per week) of retrosternal chest pain for six years, occurring equally in the day or night. The symptoms were exacerbated by exercise and associated initially with nocturnal cough, tightness in the chest preceding palpitations for one year and four episodes of syncope occurring immediately following chest pain and tightness in the past year. The last episode occurred three days prior to being seen. Her history was assessed retrospectively after catastrophic response to a treadmill stress test, requested by a physician.

Of significance in her past medical history was that she had wheezing as a toddler with no recurrence, which was treated with oral salbutamol, but she was never diagnosed as having bronchial asthma.

There was no history to suggest congenital heart disease, pre-excitation syndromes, ion channelopathy or brugada syndrome, nor stress to substantiate a diagnosis of takotsubo cardiomyopathy. There was no travel history or use of medications, illicit drugs or highly caffeinated beverages prior to being seen. There was no history of surgical or interventional procedures.

Of significance in her family history were multiple first-degree members with bronchial asthma including her father, brother and sister. She was the sixth of seven children in the family. There was no family history of congenital heart disease, arrhythmias, sudden death, sudden infant death syndrome, deafness, thyroid disorder, tall or short stature.

On examination, there was no cardiorespiratory distress at rest. She was afebrile, had pink mucous membranes, anicteric, acyanotic with saturation of 100%. Her weight was 58 kg and her height was 168 cm, giving a body mass index of 20.5. Her resting lying blood pressure was 108/75 mmHg, pulse rate 98/minute and respiratory rate 28/minute. She had negative wrist and thumb signs, no arachnodactyly and normal arm span, height and upper and lower body ratios.

Her cardiovascular examination was normal and showed a pulse rate of 98/minute, regular, synchronous, with normal volume. There was no radio-radial or radio-femoral delay, and all peripheral pulses were palpable. Her jugular venous pressure was not elevated. There were no parasternal heave, thrill or palpable sounds. Her apex beat was normal in the fifth left intercostal space, mid clavicular line with normal character. Her heart sounds one and two were normal with normal pulmonary component of the second heart sound. There were no murmurs, clicks or abnormal heart sounds.

Her respiratory system examination was normal with vesicular breath sounds. The index case had a syncopal episode during a treadmill stress test where she had marked rhonchi with markedly decreased air entry with prolongation of expiratory phase. Her abdominal, cardiovascular, skeletal and central nervous system examinations were normal.

Haematological and thyroid function tests were normal. Electrocardiogram and chest X-ray were normal for age.

Her echocardiogram showed a structurally normal heart with normal origin and course of coronary arteries. Specifically, there was no evidence of Ebstein's anomaly, corrected transposition, atrial septal aneurysm, mitral valve prolapse, mitral stenosis, cortriatrum, supravalvar mitral membrane, superior nor inferior sinus venosus defects, no dilated coronary sinus or unroofing of coronary sinus, non-compacted left ventricle, arrhythmogenic ventricular dysplasia, Uhl's anomaly, normal aorto-left atrium ratio. Modified Bruce protocol treadmill stress test confirmed severe exercise-induced bronchial asthma causing syncope. Her pre-test electrocardiogram was normal with a normal sinus rhythm. Her pre-test heart rate was 99/minute and blood pressure was 108/70 mmHg. The maximum heart rate achieved was 144/minute (69% of maximum predicted of 208/minute). Heart rate and blood pressure responses to exercise were appropriate. The maximum blood pressure was 135/90 mmHg. It returned to normal (119/66 mmHg) after eight minutes in the recovery period. There were no arrhythmias or ischaemic changes.

The treadmill stress test was aborted at stage 1 at six minutes, when the patient developed and complained of dyspnoea, chest pain with shortness of breath and signs of respiratory distress with tachypnoea and respiratory rhonchi, which were the limiting factors. There was markedly reduced air entry with prolongation of expiratory phase with saturations falling from 100% to 84% when syncope ensued. The patient was given oxygen, laid in the supine position, and an intravenous line set up with normal saline.

Nebulized salbutamol and ipratropium bromide were instituted with initial minimal improvement. Intravenous aminophylline and hydrocortisone and continued nebulization were given to the patient with good effect. Within the following eight minutes, the patient was sitting upright with appropriate response to verbal commands. She was allowed to recover until her heart rate and blood pressure and saturation level returned to the normal pre-test levels. The dyspnoea, chest pain, signs of respiratory distress and rhonchi in the lung fields bilaterally were completely aborted with the treatment given.

The patient walked unaided from the institute and had a post-treadmill chest X-ray done which was normal. Severe exercise-induced bronchial asthma was confirmed. Optimal management of her bronchial asthma with rescue inhalers and prophylaxis were recommended. Recreational physical activities but no competitive or elite sports were advised.

DISCUSSION

The patient had a strong family and clinical history supportive of a diagnosis of bronchial asthma. If she were seen prior to the treadmill stress test that led to the diagnosis of syncope secondary to bronchial asthma, it would have been prudent to do lung function

testing – spirometry, bronchodilator testing. Her syncopal attacks being induced by the bronchial asthma exacerbations would have been reproducible by metacholine challenge testing, which however would be considered to be risky given the severity of her symptoms. Tilt table testing would have been helpful to rule out orthostatic hypotension (2–14).

The incidence and causes of syncope vary according to age, ethnicity, country of origin, family genetic predisposition and association with specific types of congenital and electrophysiological heart disease. The demise of a child from syncope causes devastating effects on families, hence the need for the identification of treatable causes. The patients and their parents who are identified with lesions that are most likely genetically linked or familial would need to be advised on moral, ethical and legal grounds, and investigations should be pursued. The current literature is replete with Caucasians with syncope in the adolescent period. However, there is none specific on the Black or Afro-Caribbean population with syncope secondary to bronchial asthma. The literature has no study on adolescent Blacks or Afro-Caribbeans with syncope (1–29).

This index case supports the need for detailed history (including family history and appropriate investigations) and a high index of suspicion to facilitate the identification of the myriad of secondary causes including bronchial asthma (in this case, a rare presentation with exercise-induced syncope). This would lead to appropriate and optimal management, which will help to reduce morbidity and mortality.

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