Syncope during resistance exercise in an individual with type 1 diabetes

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Received: 3 April 2013
Accepted in current form: 10 May 2013

Case report

Here we detail a case study of a 25-year-old male with T1DM who experienced a single case of exercise-induced syncope during an unaccustomed acute bout of RE. He had a body mass of 86.5kg, height 1.9m, HbA1c 27mmol/mol (4.6%), and body fat 16.5%; diagnosed in November 2010, he was free from diabetes-related complications, and treated with basal-bolus insulin (aspart [NovoRapid] and glargine [Lantus], respectively).

The subject was a participant in a local research ethics committee approved study exploring the metabolic and cardiovascular effects of RE in individuals with T1DM. After a preliminary session to determine maximal strength using a three-repetition maximum assessment one week before the RE trial, he attended the exercise laboratory at 6am after an overnight fast having taken usual basal insulin (glargine 14U) the night before to perform an acute RE session. The exercise session comprised three sets of 10 repetitions of a range of upper and lower body exercises performed at 65–70% of the participant’s maximal strength determined from the preliminary session. The isotonic exercises in the session were bench press, leg extension, shoulder press, pectoral deck, squats, lateral pull-down, seated row and lunges, performed in that order. The subject was encouraged to regularly breathe during the session and maintain hydration; no valsalva manoeuvres were performed during the session. After each exercise the participant rested for 60 seconds, and between each circuit rested for 2 minutes. Cardiovascular data were obtained using a BioHarness™ (Zephyr Technology Co. Annapolis, MD)

Abstract

Although metabolic and cardiovascular effects of resistance exercise in type 1 diabetes (T1DM) remain poorly explored, research employing type 2 diabetes suggests glycaemic and cardiovascular benefits. However, this intense exercise carries some risks.

Here we describe the cardiovascular and metabolic responses of a newly diagnosed, previously sedentary T1DM individual experiencing syncope during an unaccustomed acute bout of resistance exercise.

The cause of this exercise-induced incident was attributed to inappropriate cardiovascular control and lack of habituation to accompanying acid-base disturbances. Careful consideration of exercise intensity and progression in previously sedentary T1DM performing resistance exercise sessions is warranted. Copyright © 2013 John Wiley & Sons.

Practical Diabetes 2013; 30(7): 290–293

Key words

glycaemic; metabolic; heart rate; syncope; resistance exercise; type 1 diabetes

Introduction

Physical activity is a favourable non-pharmacological intervention for the management of type 1 diabetes (T1DM). Regular exercise can lead to improvements in health-related factors including reduced glycosylated haemoglobin (HbA1c), delayed micro- and macrovascular complications, increased insulin sensitivity, improved glucose clearance and a predisposition to decreased mortality rate (for review). While most of the above-mentioned health benefits have been attributed to aerobic exercise training, regular resistance exercise (RE), or ‘strength training’, can also promote better health of both type 2 diabetes (T2DM) and T1DM. Furthermore, although RE is promoted by the American College of Sports Medicine (ACSM) for use in T2DM in line with specific exercise session characteristics, in the context of T1DM guidelines are less defined. It therefore remains unclear as to the acute effect of RE in T1DM.

Case report

Here we detail a case study of a 25-year-old male with T1DM who experienced a single case of exercise-induced syncope during an unaccustomed acute bout of RE. He had a body mass of 86.5kg, height 1.9m, HbA1c 27mmol/mol (4.6%), and body fat 16.5%; diagnosed in November 2010, he was free from diabetes-related complications, and treated with basal-bolus insulin (aspart [NovoRapid] and glargine [Lantus], respectively).

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and blood metabolic data gathered from GEM3000 (Instrumentation Laboratories Ltd, UK).

On arrival to the laboratory (room temperature 25.3°C, relative humidity 53%, barometric pressure 1018 mmHg), a resting venous blood sample was collected via an indwelling cannula and kept patent with 0.9% saline solution. Baseline markers were representative of a rested state (Table 1; glucose 5.1 mmol/L, lactate 0.5 mmol/L, pH 7.34, heart rate [HR] 60 bpm, plasma adrenaline [A] 0.11 nmol/L, plasma noradrenaline [NA] 1.90 nmol/L). Venous bloods were subsequently sampled at the end of each set of eight exercises and for 1 hour following cessation of exercise. After two full circuits (i.e. two sets of eight exercises), glucose rose by 0.3 mmol/L to 5.4 mmol/L, lactate concentration increased to 17.0 mmol/L, blood pH was 7.06 and HR was 176 bpm.

The participant continued on to the third circuit of exercise, with controlled breathing and displaying no visible symptoms relating to pre-syncope at this point. However, after partially completing the eighth exercise, upon standing the participant complained of feeling nauseous and dizzy. He began to display clammy, pale skin and experience shallow breathing. The participant was immediately seated and a blood sample and blood pressure taken. Blood glucose concentration was 6.3 mmol/L, lactate concentration was 17.0 mmol/L, blood pH was 7.06 and an HR of 179 bpm.

After 1 hour we observed maintenance in blood glucose concentration (6.2 mmol/L) but incomplete recovery of blood acid-base balance (lactate 9.9 mmol/L, pH 7.28). Blood pressure was 113/50 mmHg at 15 and 30 minutes post-exercise, respectively. The participant then remained in the laboratory for a further 40 minutes during which he ate breakfast, self-administering a usual dose of insulin aspart (3U). As this subject was a participant who had completed a 75-minute resting trial on a separate occasion, two weeks before the exercise trial, we present his rested data at equivalent time points for comparison (Table 1; Figure 1).

### Table 1. Metabolic responses before, during and after resistance exercise. CON refers to the resting trial; 3 sets refers to the exercise trial in which the participant completed 2 sets and 7 exercises prior to syncope

<table>
<thead>
<tr>
<th></th>
<th>Pre-exercise</th>
<th>Exercise</th>
<th>Post-exercise (min)</th>
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<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>1st set</td>
<td>2nd set</td>
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<tr>
<td><strong>Glucose (mM)</strong></td>
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<tr>
<td>CON</td>
<td>4.6</td>
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<td>3 sets</td>
<td>5.1</td>
<td>4.8</td>
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<td><strong>pH</strong></td>
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<tr>
<td>CON</td>
<td>7.34</td>
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<td>3 sets</td>
<td>7.34</td>
<td>7.1</td>
<td>7.06</td>
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<td><strong>Lactate (mM)</strong></td>
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<tr>
<td>CON</td>
<td>0.4</td>
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<td>–</td>
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<tr>
<td>3 sets</td>
<td>0.5</td>
<td>14.0</td>
<td>17.0</td>
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<td><strong>HCO3 (mM)</strong></td>
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<tr>
<td>CON</td>
<td>30.2</td>
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<td>3 sets</td>
<td>31.8</td>
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<td><strong>Base excess (mM)</strong></td>
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<tr>
<td>CON</td>
<td>4.4</td>
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<td>–</td>
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<tr>
<td>3 sets</td>
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<td>-18.1</td>
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<td><strong>K+ (mM)</strong></td>
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<tr>
<td>CON</td>
<td>3.8</td>
<td>–</td>
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<tr>
<td>3 sets</td>
<td>3.6</td>
<td>3.7</td>
<td>3.8</td>
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<tr>
<td><strong>Plasma insulin (pmol/L)</strong></td>
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<tr>
<td>CON</td>
<td>144.7</td>
<td>–</td>
<td>–</td>
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<tr>
<td>3 sets</td>
<td>144.2</td>
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Case report
Syncope during exercise in type 1 diabetes

Discussion
Resistance exercise induces very different physiological and metabolic responses compared to aerobic exercise. Our exercising T1DM subject displayed large cardiovascular changes and severe metabolic acidosis to an exercise volume that is typically prescribed to novice–intermediate individuals, in reference to ACSM guidelines for those with and without T2DM. With a view to facilitating the appropriate prescription of RE, we describe a number of predisposing factors that may influence the likelihood of experiencing a syncopal episode in response to RE.

Firstly, lack of familiarisation or pre-conditioning to performing this intense activity may have predisposed this individual to experiencing a syncopal episode. Common experience suggests that exercise-induced acid-base disturbances (Table 1) can cause dyspnoea and light-headedness, which are symptoms closely linked to pre-syncope. Interestingly, in contrast to the other T1DM participants involved in the study (who performed exercise at the same relative intensity and volume), both blood pH and lactate responses were in the upper range of values (cohort range: peak lactate 8.2–19.5mmol/L; nadir pH 6.94–7.29; n=8). This range clearly highlights the diversity in the metabolic consequences of exercise, most likely reflective of habitual levels of physical fitness – thus placing emphasis for the requirement to identify a T1DM patient’s exercise capacity prior to prescribing non-supervised exercise.

Secondly, a warm environment can accentuate the physiological stress induced by exercise. Ambient temperature in the laboratory (25°C) was higher than American Heart Association guidelines of 20–22°C. Exercising in a hot environment reduces the body’s effectiveness at dissipating heat, placing an additional stressor on the subject. As such, the coordinated movement of blood from core to periphery to aid in thermoregulation may have been dysregulated due to an inability to cool the peripheral blood on its return to the core because of the warm environment. Additionally, the greater proportion of blood volume moving to the periphery compromises sufficient volume to central organs and can reduce oxygen delivery to the heart and brain. In our case, this response may have contributed to the loss of consciousness. Performance of RE requires adoption of upright, seated and supine positions. Although the participant was familiarised to each exercise technique in an introductory session, he was not habituated. Quickly alternating between these positions can accentuate fluctuations in central and peripheral blood pressure as large volumes of blood are moved to and from working muscles. Lack of acclimatisation to this exercise type can cause syncope as the body fails to adapt quickly enough to the stress of exercising in alternate positions with insufficient blood flow.

Cardiovascular data provide a dramatic illustration of the impact of syncope on exercising heart rate. As a result of performing RE the peak heart rate was 179bpm. Indeed, syncope in non-diabetic individuals in response to strenuous exercise is usually conducive to failure of neural mechanisms associated with bradycardia or asystole. We observed an 8–10-fold increase in resting adrenaline (A) and noradrenaline (NA) concentrations in response to the RE protocol; that was also apparent when compared to equivalent time-points in the control trial (rest: A 0.04, NA 2.20nmol/L; 0 minutes post-exercise period: A 0.07, NA 1.00nmol/L). In line with our findings, RE has previously been found to provoke a strong catecholamine response especially from the sympathetic hormones adrenaline and noradrenaline. Through their actions on α- and β-adrenoceptors, catecholamines influence sympathovagal balance and aid in regulating blood flow and total peripheral resistance. Typically, increased sympathetic activity results in concomitant vagal withdrawal, but high circulatory catecholamine concentrations can dysregulate sympathovagal balance, resulting in disruption of baroreflex control and blood flow accordingly. The syncopal episode lasted approximately 3 minutes and reduced peak heart rate by 122bpm to 27bpm in the face of a strong sympa-ho-adrenal stimulus.

Although syncope in response to unfamiliar exercise is not uncommon in those without diabetes, we highlight that diabetes-related factors may have increased the sensitivity of the participant to the physiological responses elicited by exercise, thence leading to a syncopal episode. In diabetes, a reduction in stroke volume compared to those
without diabetes may impair cardiac function and exacerbate loss of cerebral and peripheral blood flow. Moreover, there have been reports demonstrating elevated exercise-induced acidosis when compared to fitness-matched non-diabetic individuals. Thus, exercise that would normally be prescribed to an individual without TIDM may impose a relatively greater metabolic and cardiovascular stress on an individual with TIDM.

The inability of the body to self-regulate changes in glycaemia is borne in TIDM, and in turn predisposes patients to symptoms associated with hypo- or hyperglycaemia. Although hypoglycaemia is a significant cause for concern in the exercising TIDM individual, blood glucose values were euglycaemic and remained slightly higher than rest over the recovery period.

In conclusion, we detail an episode of syncope in a newly diagnosed TIDM individual unaccustomed to regular RE. The cause of this intense exercise-induced incident may be attributed to a redundancy in cardiovascular control in a warm environment alongside large acid-base perturbations. Caution should be taken when implementing new exercise programmes in individuals unaccustomed to this form of exercise, even if they adhere to current exercise guidelines. It may be prudent to start lifting lower weights at first, gradually progressing as habituation to acid-base disturbances improves.

Acknowledgement
This project has been made possible by the EU’s Convergence European Regional Development Fund through the Welsh Assembly Government.

Key points
- Regular resistance exercise is promoted for use in those with and without diabetes for benefits related to physical well-being.
- Guidelines for resistance exercise are available to those with and without diabetes, but in the context of type 1 diabetes guidelines are less defined.
- This case study highlights the metabolic impact of an American College of Sports Medicine recommended resistance exercise session relevant to those with and without diabetes in an individual with type 1 diabetes.
- Reports of this nature are essential to those with type 1 diabetes as well as healthcare professionals so that exercise can be safely undertaken and appropriately prescribed, respectively.

Declaration of interests
There are no conflicts of interest declared.

References