Hyperventilation and Raynaud's disease

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Summary: A 42 year old woman with long standing Raynaud's disease, unresponsive to medical and surgical treatment, was noted to have a typical history of the hyperventilation syndrome. Rewarming of the hands following cold challenge was markedly prolonged in the presence of hypocapnia. It is suggested that hyperventilation may have an aetiological role in maintaining digital artery spasm in Raynaud's disease, which would benefit from recognition and treatment.

Introduction

Raynaud's disease is an idiopathic condition which is often familial, and commoner in females, characterized by intermittent symmetrical attacks of pallor, and/or cyanosis affecting the digits. It is precipitated by the cold and often begins in early adulthood. Chronic hyperventilation is a condition in which fluctuating hypocarbia produced by upper chest breathing occurs inappropriately in response to exercise or emotion producing many varied symptoms, many of which mimic or exaggerate concomitant organic disease.¹ A case is reported of a patient with severe Raynaud's disease who also had the hyperventilation syndrome, and an aetiological association is suggested.

Case report

A 42 year old housewife first developed typical features consistent with Raynaud's disease in her late teens. All screening tests for other causes of Raynaud's phenomenon, such as connective tissue disease or obstructive arterial lesions were negative. Her symptoms were not alleviated by simple measures such as warm clothing and gloves, and she quickly progressed to bilateral cervical sympathectomy. At surgical follow-up, she continued to complain of typical symptoms but was in addition noted by an ex-student of this hospital to be hyperventilating. She was therefore referred to our unit.

She gave a typical history consistent with a diagnosis of the hyperventilation syndrome, viz: inability to get enough air into her lungs, frequent sighing,

perioral paraesthesiae, dizziness and stabbing chest pains.² In addition she reported that these symptoms often coincided with her episodes of digital artery spasm. Her resting respiratory pattern was predominantly upper thoracic in nature, and irregular in frequency, punctuated by frequent sighing. This pattern was grossly exaggerated when the patient was exposed to the cold.

Her resting end tidal (et) CO₂, measured by an IL 200 infra-red mass spectrometer (calibrated with 5% CO₂ from a Corning medical gas cylinder and corrected daily for barometric pressure) was 28 mm Hg (normal 35–45 mm Hg). A forced hyperventilation provocation test was performed at 60 breaths per minute for 3 minutes and the *P_{et}* CO₂ was required to fall below 19 mm Hg by the end of the test. The rate of return of the *P_{et}* CO₂ plotted and the value at 3 minutes after the hyperventilation was used to derive a ratio of the resting *P_{et}* CO₂ to the *P_{et}* CO₂ 3 minutes post-provocation. This ratio was 1.52.³ Following provocation she reported symptoms of stabbing chest pain, dizziness and tingling in her upper limbs, typical of those that she experienced in daily life. There were no skin changes in the digits, nor did the skin temperature fall, as documented by a skin probe.

A cold challenge was performed where the patient's hands were cooled to 18°C and typical painful finger blanching was produced. Her resting *P_{et}* CO₂ was 27 mm Hg and this remained less than 30 throughout the procedure. The dominant respiratory pattern was noted to be upper thoracic. She also complained of chest pain during this time, although a continuous standard 6 lead electrocardiogram revealed no ST/T wave abnormalities. The rate of return of the skin temperature to normal was 30 minutes.

The same cold challenge was repeated to 18°C, where typical blanching of digital ischaemia was evident. On this occasion, however, she was shown

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Figure 1 Cold challenge of the digits to 18°C. The upper trace shows the time taken for rewarming of the digits in the presence of hyperventilation and hypocapnia (30 minutes). The lower trace shows the much shorter time taken for rewarming (18 minutes) when the patient was not hyperventilating and the *Pet*, CO₂ was normal. — Skin temperature; --- *Pet*, CO₂.

how to breathe in a slow, relaxed and controlled abdominal fashion. The resting *Pet*, CO₂ was 33 mm Hg and did not fall as had previously been the case. The rate of return of the skin temperature to normal was much faster than on the first occasion (18 minutes) and she did not experience any chest pain (see Figure 1). There was no difference in the warming up rates of *Pet*, CO₂ levels in a normal subject when faced with the same test.

She was given training in slow controlled abdominal breathing techniques such that it became her dominant respiratory pattern – even whilst talking or presented with any upset.⁴ She was, in addition, given counselling about coping with daily problems and encouraged to obtain adequate sleep, as has been described before.⁵ During a 6 month follow-up she reported that her Raynaud's disease had improved considerably, especially in terms of severity and duration of attacks, but also in terms of frequency.

Discussion

This woman had typical features of the chronic hyperventilation syndrome and she well describes the characteristic symptoms of 'difficulty getting enough air into the lungs', dizziness and chest pain as well as circumoral paraesthesiae. In addition, she had resting hypocapnia and reproduced her typical symptoms following the provocation test. The derived ratio greater than 1.5 is also consistent with a diagnosis of hyperventilation, according to the criteria of Haddonk & Beumer.³

The patient also had typical and relatively incapacitating Raynaud's disease, which had failed to show much improvement despite surgical intervention.

There is a good reason to consider that the pathophysiological basis of Raynaud's disease is an increased sensitivity to arterial vasospasm,⁶ and there are many reports in the literature of its association with other conditions in which arterial constriction is considered to play a predominant aetiological role, such as migraine⁷ and variant angina pectoris.⁸

Hyperventilation has been strongly implicated in producing arterial spasm, possibly due to altered ionized calcium levels secondary to the systemic alkalosis produced by the associated hypocapnia. Forced hyperventilation can provoke spasm of both cerebral¹⁰ and coronary arteries,¹¹ and in many cases there is evidence of a more generalized vasoconstrictive effect, including digital ischaemia.¹² The association of spontaneous hyperventilation and variant angina has also been reported.¹³

We suggest that this case demonstrates the role that chronic hyperventilation may have in maintaining digital artery spasm and we further postulate that on some occasions it may even induce it. Chronic hyperventilation is a commonly missed disorder, which may go unrecognized in many patients with Raynaud's disease, adding considerably to their morbidity.

Specific enquiry about the characteristic symptoms of hyperventilation, perhaps reproduced by a provocation test, should become routine in the assessment of such patients, and where found, breathing retraining may have an important therapeutic role.

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