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Wide pulse pressure and Quincke's pulse in high-output heart failure

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SUMMARY

A 74-year-old man with a history of chronic alcohol use presented with progressive exertional dyspnoea and weight gain. On physical examination, he was noted to have wide pulse pressure, elevated jugular venous pressure, and alternating flushing and blanching of the nail beds in concert with the cardiac cycle, known as Quincke's pulse. Transthoracic echocardiography demonstrated normal biventricular systolic function and valvular function, but noted a dilated inferior vena cava. Right heart catheterisation revealed elevated filling pressures, high cardiac output and low systemic vascular resistance, consistent with high-output heart failure. Whole blood concentration of thiamine was low, confirming the diagnosis of wet beriberi. The patient abstained from alcohol use and was started on thiamine replacement therapy, resulting in narrowing of the pulse pressure over time and complete resolution of symptoms without the need for diuretic therapy.

BACKGROUND

Heart failure is one of the most common conditions seen in modern-day healthcare, with an estimated prevalence of 6.2 million people in the USA between 2013 and 2016.[1] It is most often associated with low or normal cardiac output along with high systemic vascular resistance. The diagnosis is made clinically, relying heavily on characteristic physical findings. Signs of venous congestion, such as elevated jugular venous pressure, are nearly ubiquitous; however, a minority of heart failure patients present with a high-output state, which may produce additional, unique physical findings, such as Quincke's pulse. When heart failure occurs in combination with high-output physiology, it is known as high-output heart failure.

Quincke's pulse is characterised by alternating flushing and blanching of the nail bed in concert with the cardiac cycle, and frequently occurs along with wide pulse pressure. It is most commonly associated with severe aortic valve insufficiency; however, it can also be seen in patients with coarctation of the aorta as well as in patients with high-output physiology. The presence of Quincke's pulse in a patient presenting with heart failure should raise suspicion for high-output heart failure. Recognising this subset of heart failure patients is critical because there are unique treatment options. Moreover, identifying and differentiating the specific underlying aetiology is crucial for initiating specific therapeutics.

CASE PRESENTATION

A 74-year-old man with obesity and chronic alcohol use presented to the hospital with a 2-month history of progressive exertional dyspnoea and weight gain. The patient reported drinking three to four glasses of wine daily for at least 10 years. Physical examination was notable for a body mass index of 34.3 kg/m^2, blood pressure of 177/56 mm Hg, dependent pitting peripheral oedema, jugular venous pressure of 18 cm H_2O with a normal waveform, unremarkable S1 and S2 without extra transient sounds or murmurs, and subungual capillary pulsations (Quincke's pulse) (video 1).

INVESTIGATIONS

Laboratory data were notable for haemoglobin of 100 g/L with a mean corpuscular volume of 103.3 fL. Transthoracic echocardiography demonstrated a dilated inferior vena cava with normal biventricular systolic function and normal valvular function, raising the suspicion for high-output heart failure. Subsequent right heart catheterisation (after some diuresis) revealed elevated resting intracardiac pressures (mean right atrial pressure 9 mm Hg, mean pulmonary arterial wedge pressure 16 mm Hg), high cardiac output (12.3 L/min by thermodilution) and low systemic vascular resistance (587 dynes/s/cm^{-5}), confirming the diagnosis. Whole blood concentration of thiamine diphosphate, the primary active form of vitamin B_1, was 39 nmol/L (reference 70–180 nmol/L).

DIFFERENTIAL DIAGNOSIS

The patient presented with symptoms and signs of heart failure, including exertional dyspnoea, weight gain, peripheral oedema and elevated jugular venous pressure. Given its prevalence, the diagnosis of traditional 'low-output' heart failure with preserved ejection fraction might be enticing in this case. However, the presence of wide pulse pressure and Quincke's pulse suggest a more nuanced diagnosis. These findings are most often associated with aortic insufficiency; however, the absence of a diastolic murmur and the presence of normal valvular function on echocardiography argue against this. Wide pulse pressure and Quincke's pulse are associated with a high-output state from any cause, and given the additional findings consistent with heart failure, high-output heart failure was suspected and subsequently confirmed with right heart catheterisation. The differential for the underlying cause of high-output heart failure is broad and includes obesity, liver disease, myeloproliferative disorders, lung disease, sepsis, severe anaemia, thyrotoxicosis, arteriovenous shunting and beriberi.[2] While obesity could account for the high-output physiology in this case, it should not be considered as the culprit until all other reasonable possibilities have been excluded. Given the history of chronic alcohol use, a thiamine

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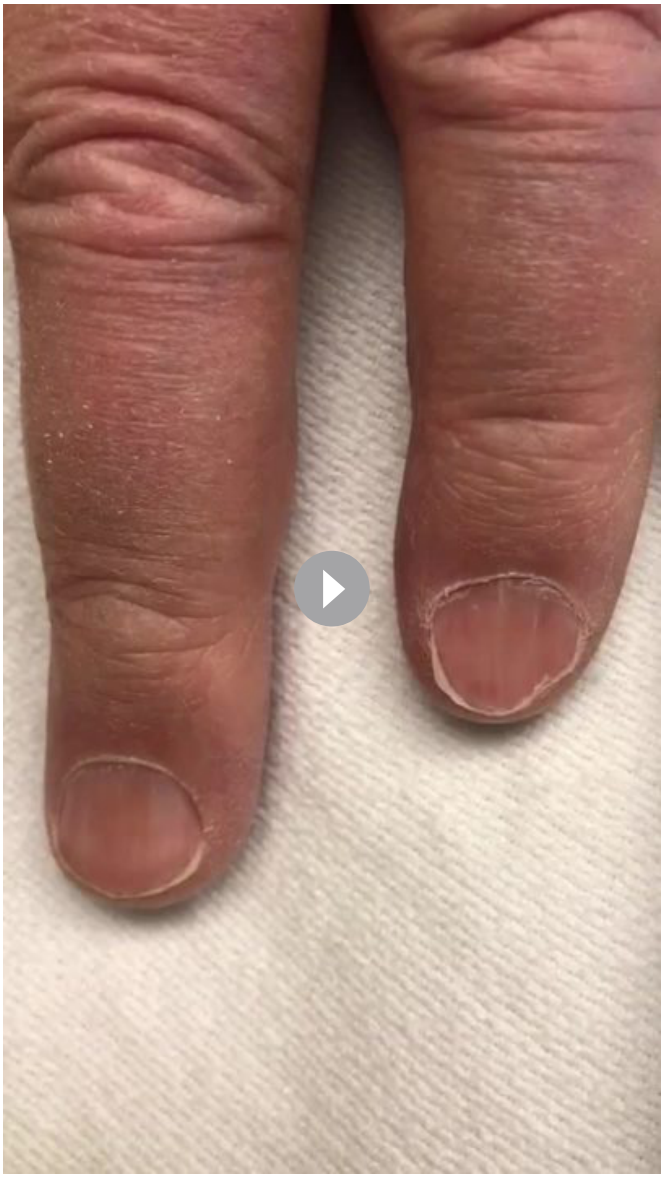

Video 1 Quincke's pulse.

level was checked and found to be low, confirming the diagnosis of wet beriberi.

TREATMENT

The patient was initially treated with diuretics to achieve euvolaemia as well as intravenous thiamine, receiving a total of 500 mg over the course of 5 days. He abstained from alcohol use and was started on oral thiamine replacement therapy (100 mg three times a day). Ongoing diuretic therapy was not required.

OUTCOME AND FOLLOW-UP

Alcohol abstinence and thiamine replacement therapy resulted in narrowing of the pulse pressure by nearly 40 mm Hg over 28 weeks (figure 1) and complete resolution of symptoms without the need for diuretic therapy.

DISCUSSION

Wide pulse pressure and Quincke's pulse are most often associated with aortic valve insufficiency; however, these physical findings are indicative of hyperdynamic circulation from any cause: in this case,

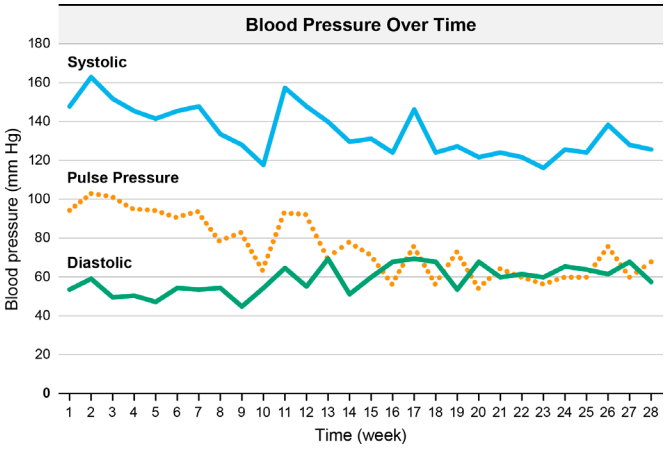

Figure 1 Blood pressure and pulse pressure trends over time with alcohol abstinence and thiamine repletion in a patient with wet beriberi.

high-output heart failure from thiamine deficiency. The association between hyperdynamic circulation and thiamine deficiency has been recognised for over a century, with descriptions of wide pulse pressure and Quincke's pulse in patients with wet beriberi first appearing in Western journals in the 1920s.[3 4] Since then, these findings have been associated with other causes of high-output heart failure, including thyrotoxicosis and arteriovenous fistula.[5 6]

Making the distinction between traditional heart failure and high-output heart failure is important. Most patients with heart failure have low or normal cardiac output with high systemic vascular resistance, while a minority present with high cardiac output and low systemic vascular resistance. There are many overlapping symptoms and signs between these conditions (eg, dyspnoea, peripheral oedema), however 'low-output' heart failure is significantly more common. Therefore, when patients present with the clinical syndrome of heart failure, clinicians are at risk of premature closure and settling on a diagnosis of low-output heart failure before recognising the pivotal and unique signs of high-output heart failure. This is particularly true when

Patient's perspective

This was my first encounter with Oregon Health & Science University. I was quickly diagnosed with congestive heart failure. During the first 3 hours, I met more doctors than I had met in my entire life. I was introduced to a great team of specialists who worked together to ensure that solving my main problem didn't cause other problems. But had it not been for one of the doctors (Dr André Mansoor), the root cause of my problem might never have been found. After multiple physical examinations, Dr Mansoor asked me if I noticed that my fingernails were blinking. Thinking that he was joking, I responded 'well of course they are, it is a sign that my batteries are low and need charging'. Seeing that he was serious, I looked but couldn't really see anything. He then took out his phone and took a video and showed me and I couldn't believe what I was seeing. This information was key for him to get permission for other tests that discovered that I had a rare issue with thiamine deficiency, which of course I had never heard of. Getting me onto B1 brought my thiamine level back to normal and I have been fine ever since. Today, I think back on how discovering this problem would have been impossible had it not been for Dr Mansoor's physical examination and I am grateful for his persistence.

echocardiography demonstrates a normal left ventricular ejection fraction and patients are given the diagnosis of heart failure with preserved systolic function.

Evidence of hyperdynamic circulation in a patient presenting with the clinical syndrome of heart failure should raise concern for high-output heart failure. Signs not only include wide pulse pressure and Quincke's pulse, but also warm and well-perfused extremities, bounding pulses (Corrigan's or water hammer pulse), systolic bruit over the carotid arteries, pistol shot sounds over the femoral arteries (Traube's sign), cervical venous hum, enlarged apical impulse and a loud first heart sound.[3–6] Clinical manifestations related to a particular cause of high-output heart failure (eg, painful glossitis in a patient with thiamine deficiency) may also be present.

Once the diagnosis of high-output heart failure has been established, it is important to identify the underlying cause in order to guide treatment, which can sometimes be curative.[3 4 7] In this case, whole blood concentration of thiamine diphosphate was 39 nmol/L (reference 70–180 nmol/L), confirming the diagnosis of wet beriberi from thiamine deficiency. This allowed for a specific treatment plan, including alcohol cessation and thiamine replacement. Additionally, some medications typically prescribed for low-output heart failure may be contraindicated in patients with high-output disease. For example, medications with vasodilatory properties, such as ACE inhibitors /angiotensin receptor blockers and some beta-blockers may further lower systemic vascular resistance in patients with high-output heart failure and are not recommended.[7]

Learning points

- Wide pulse pressure and Quincke's pulse are most commonly associated with aortic valve insufficiency; however, these findings are indicative of hyperdynamic circulation from any cause.
- High-output heart failure is uncommon, but should be suspected when heart failure patients present with the physical findings of hyperdynamic circulation.
- Making the distinction between high-output and low-output heart failure is critical because there are important differences in treatment and prognosis.

This case demonstrates the importance of distinguishing high-output heart failure from other more common forms of heart failure, and the critical role that physical examination plays in this process. The observation of wide pulse pressure and Quincke's pulse can provide a pivotal clue to the diagnosis of high-output heart failure, particularly when echocardiography is uninformative. If these important features of the case had gone unnoticed, the opportunity for curative treatment would have likely been missed.

Correction notice This article has been corrected since it has been published online. The value of haemoglobin is now changed from 0.1g/L to 100 g/L.

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