EXCEPTIONAL CASE

Central volume shift in acute heart failure revealed by blood volume monitoring during haemodialysis

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ABSTRACT

Central volume shift is one of the major pathophysiological mechanisms of acute pulmonary oedema in acute heart failure (AHF). Pathological vasoconstriction results in central volume shift; however, its onset and course have been rarely detected or recorded in clinical practice. We report an exceptional case of AHF developing during haemodialysis, with marked blood pressure (BP) elevation and paradoxical repeated reduction in blood volume (BV) detected by real-time BV monitoring, accompanied by worsening dyspnoea. This inverse correlation of BV and BP during haemodialysis indicates that the theoretical central volume shift was captured in real-world AHF.

Keywords: acute heart failure, acute pulmonary oedema, blood pressure, blood volume, central volume shift, haemodialysis, pathological vasoconstriction

BACKGROUND

Central volume shift is one of the major pathophysiological mechanisms of acute pulmonary oedema due to pathological vasoconstriction in acute heart failure (AHF). Interestingly, when AHF develops due to central volume shift, haemoconcentration rather than haemodilution is often observed in a blood test. Although there are a few reports of haemoconcentration occurring in acute pulmonary oedema and improving after treatment [1, 2], no reports have demonstrated its real-time onset and course. Herein, we report an exceptional case of central volume shift, whose onset and course were observed in an unusual decrease in blood volume (BV) along with a marked elevation in blood pressure (BP) during haemodialysis (HD).

CASE REPORT

A 45-year-old man on HD for the last 2 years for diabetes-related end-stage kidney disease developed sudden-onset dyspnoea 1 h into an HD session. Four months prior he developed AHF with BP elevation after HD. We lowered his dry weight to prevent recurrence of AHF; however, his BP remained high. Therefore, five antihypertensive medications were prescribed.

During the HD session in question, his BP suddenly rose from 176/97 to 227/121 mmHg, while his percutaneous oxygen saturation dropped to 82% on room air. Physical examination was remarkable for bilateral lung coarse crackles. There was no marked pitting oedema in the lower extremities. The patient...
FIGURE 1: The changes in BV and BP in the HD session. (A) BV began to decrease significantly at 1 h and 1.5 h after the start of HD (white arrows). A black arrow shows the timing of blood sampling. (B) Systolic BP markedly increased as the BV decreased (white arrows). UF, ultrafiltration; sBP, systolic blood pressure; dBP, diastolic blood pressure; mBP, mean blood pressure; PR, pulse rate; BT, body temperature.

was diagnosed with AHF based on the clinical symptoms and medical history.

A marked BV decrease is typically accompanied by a BP decrease during HD. Interestingly, however, BV, measured by an optical device (Nikkiso, Tokyo, Japan) at the time of symptom onset, was significantly decreased despite BP elevation (Figure 1). Therefore this suggested the onset of central volume shift. Nitrates and calcium channel blockers were intravenously administered for vasodilation and BP control. After treatment initiation, his BP decreased and the BV subsequently increased. However, after 30 min, the systolic BP increased to 228 mmHg and his dyspnoea worsened. Simultaneously, a BV decrease similar to the first one was observed. HD with continuous intravenous vasodilator administration continued for 5 h. Thereafter the patient was admitted to the intensive care unit. Computed tomography of the chest taken after HD showed bilateral diffuse ground-glass opacities, consistent with acute pulmonary oedema. Oral antihypertensive drugs were started on day 2 and the continuous intravenous vasodilators were discontinued on day 3. He was discharged on day 5 with stabilized BP and improved general condition.

DISCUSSION

Central volume shift refers to the redistribution of body fluids from the large vessels to the lungs with marked hypertension due to pathologic arteriovenous vasoconstriction. Mebazaa et al. [3] classified AHF due to fluid redistribution as clinical scenario 1. The fluid redistribution to the pulmonary vessels causes haemoconcentration in the peripheral vessels, even in heart failure, suggesting the presence of central volume shift. However, it is quite difficult to detect its onset and course in real time.

To the best of our knowledge, this is the first case in which the onset and course of central volume shift were captured during HD, by BV monitoring in real time. The principle of BV monitoring is to continuously measure the wavelength of light that correlates with haematocrit (Hct) during HD. BV decline is accompanied by increased Hct due to haemoconcentration and is often observed in intradialytic hypotension [4]. However, in this case the BV repeatedly decreased with simultaneous BP elevation. Based on these recurrent clinical events and a reproducible inverse correlation between BV and BP, BV decline along with BP elevation could indicate the onset of central volume shift during HD. However, blood tests showed no significant haemoconcentration (Hct of 28.7% before HD versus 29.8% after onset). This was probably due to the timing of the blood sampling. The blood test was performed >30 min after the onset of symptoms and BV may not have shown haemoconcentration at that time (Figure 1A).

In conclusion, the onset and course of central volume shift are difficult to identify clinically. This exceptional case shows that the theoretical central volume shift was captured in real-world AHF.
PATIENT CONSENT
We obtained written consent from the patient for the publication of this case report and have listed it in his medical records.

ACKNOWLEDGEMENTS
We would like to thank Editage (www.editage.com) for English language editing.

FUNDING
This case report was not funded by any companies.

CONFLICT OF INTEREST STATEMENT
None declared.

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