Original articles

Dynamic Left Ventricular Outflow Tract Obstruction in Critically Ill Patients

J. M. BROWN*, W. MURTHA†, J. FRASER‡, V. KHOURY§
*Anaesthetic Department, Frenchay Hospital, Bristol, UNITED KINGDOM
†Intensive Care Unit, Royal Alexandra Hospital, Edmonton, Alberta, CANADA
‡Intensive Care Unit, Princess Alexandra Hospital, Brisbane, QUEENSLAND
§Department of Cardiology, Princess Alexandra Hospital, Brisbane, QUEENSLAND

ABSTRACT

Objective: *To review patients managed in an intensive care unit diagnosed with dynamic left ventricular outflow tract obstruction without hypertrophic cardiomyopathy.*

Patients and methods: *Dynamic left ventricular outflow tract obstruction (DLVOTO) is characteristically associated with hypertrophic cardiomyopathy, although it has also been described in patients without this disorder. We reviewed patients managed in two intensive care units over a one year period who were hypotensive and/or resistant to catecholamine infusions.*

Results: *During the one year period nine critically ill patients were found on echocardiography to have DLVOTO. None of the patients had a prior history of hypertrophic cardiomyopathy (HOCM) or echocardiographic evidence of asymmetrical septal hypertrophy and in three patients HOCM was specifically excluded by prior or convalescent echocardiography or by post mortem analysis. We found the risk factors for DLVOTO included left ventricular hypertrophy, hypovolaemia and use of positive inotropic agents. All patients responded to an increase in intravascular volume, reduction in infused inotropic agents with one requiring metaraminol to maintain blood pressure*

Conclusions:. *Dynamic left ventricular outflow tract obstruction without hypertrophic cardiomyopathy is not an uncommon cause of hypotension resistant to catecholamines in critically ill patients. The diagnosis is important because management which includes fluid loading, vasopressors and reducing catecholamine infusions, differs from the management of other causes of shock.* (**Critical Care and Resuscitation 2002; 4: 170-172**)

Key words: dynamic left ventricular outflow tract obstruction, sepsis, echocardiography, cardiogenic shock

Dynamic left ventricular outflow tract obstruction (DLVOTO) is classically associated with hypertrophic obstructive cardiomyopathy, although it has also been described in a number of other conditions. For example, there are case reports of DLVOTO occurring after myocardial infarction[1] (causing cardiogenic shock refractory to standard therapy[2]) and in patients with acute myocardial ischaemia without infarction.[3] It has also been described following mitral valvuloplasty with atrial fibrillation[4] and during dobutamine stress tests.[5]

Reduced left ventricular end-diastolic dimensions due to hypertrophy and hypovolaemia are recognised precipitants of DLVOTO, as are rapid ejection velocities produced by an enhanced contractile state and peripheral vasodilation. The clinical features of reduced cardiac output, systolic murmur, relatively high pulmonary venous pressure and a low systemic blood press-ure may easily be mistaken for cardiogenic shock due to other causes.

Standard treatment for cardiogenic shock includes

Correspondence to: Dr Julian Brown, 21 Pembroke Rd, Bristol, BS8 3BA, England (email: brownjules@doctors.org.uk)

preload and afterload reduction and positive inotropic agents, all of which have the potential to worsen DLVOTO. In the critically ill patient where low cardiac output states are common, a high index of suspicion is needed to make the diagnosis and alter treatment. We reviewed patients who were hypotensive and/or resistant to catecholamine infusions to consider the physiology, aetiology, risk factors, diagnosis and management of DLVOTO in the critical care setting.

PATIENTS and METHODS

We reviewed a series of patients managed in two intensive care units over a one year period who were hypotensive and/or resistant to catecholamine infusions. Transthoracic or transoesophageal echocardiography were used to assess the presence of DLVOTO. If DLVOTO was recorded, intravenous fluids, reduction in catecholamine infusions and vasopressor use (if the blood pressure was not maintained) were used in an attempt to reduce the obstruction and correct hypotension.

RESULTS

Nine patients were identified with DLVOTO over the one year period. Demographic details of each patient are given in table 1. Table 2 lists the echo-cardiographic findings. None of the patients had a prior history of hypertrophic cardiomyopathy (HOCM) or echocardiographic evidence of asymmetrical septal hypertrophy and a post mortem specifically excluded HOCM in patient 1.

Eight of the nine patients were receiving inotropic infusions at the time the DLVOTO was diagnosed. Six patients had a reduced systemic vascular resistance due to either sepsis or a systemic inflammatory response syndrome when the diagnosis was made.

All patients were treated with intravenous fluids. In six of the nine patients an increased infusion of intravenous fluids allowed the inotropic agents to be weaned off. In one patient a metaraminol infusion was used. Patient 3 did not respond to therapy and died shortly after the diagnosis of DLVOTO was made. In our series of patients the mortality was 44%.

Table 1. Demographic data of nine critically ill patients with dynamic left ventricular outflow tract obstruction

Patient number	*Age*	*Sex*	*Admission diagnosis*	*Sepsis*	*Previous hypertension*	*Survival*	*Positive inotropic agents*
1	68	Male	Ruptured aortic aneurysm	Yes	Yes	Yes	Ad
2	40	Male	Liver transplant	Yes	No	No	Norad + Ad
3	59	Female	Subarachnoid bleed	No	No	No	Nil
4	67	Female	Nephrectomy	No	Yes	Yes	Dop
5	80	Male	Influenza pneumonia	Yes	No	Yes	Norad
6	68	Male	Peritonitis	Yes	No	No	Norad
7	64	Female	Peritonitis	Yes	Yes	No	Dop
8	79	Female	Diverticulitis	Yes	No	No	Dop
9	63	Male	Ruptured aortic aneurysm	No	No	Yes	Dop + Ad

Dop = dopamine, Norad = Noradrenaline, Ad = Adrenaline.

Table 2. Echocardiographic findings of the nine critically ill patients

Patient number	*Outflow tract gradient (mmHg)*	*Mitral regurgitation**	*Left ventricular hypertrophy***
1	20	nil	moderate
2	100	severe	mild
3	16	moderate	moderate
4	25	moderate	mild
5	not recorded	moderate	mild
6	not recorded	nil	nil
7	78	mild	moderate
8	not recorded	nil	nil
9	218	moderate	mild

* Subjective evaluation based on left atrial dimensions, mitral early diastolic velocity and 2 D colour flow
** normal < 1.1 cm, mild 1.1 – 1.4 cm, moderate 1.5 – 1.7 cm, severe > 1.7 cm

DISCUSSION

Dynamic left ventricular outflow tract obstruction may occur in the presence of a number of cardiovascular physiological disturbances including reduced circulating volume, peripheral vasodilation and increased myocardial contractity (e.g. excessive use of inotropic agents). Management of this disorder includes increasing the patients intravascular volume (i.e. increasing preload), reducing inotropic infusions (i.e. reducing myocardial contractility) and increasing peripheral resistance (i.e. increasing afterload). Beta adrenergic recepor blockers are often used in patients with hypertrophic obstructive cardiomyopathy but in the majority of our patients, myocardial contractility could be reduced by reducing the exogenous inotropic agents. Pressors may be used to maintain adequate blood pressure as inotropes are weaned and, in one of our patients, metaraminol was used to achieve this.

As seven of our nine patients were older than 60 years and all were aged over 40, the age and presence of left ventricular hypertrophy (which existed in 7 of the 9 patients) appeared to be the major predisposing factors for left ventricular outflow tract obstruction. Typical findings include systolic anterior motion (SAM) of the mitral valve (figure 1) and a pressure gradient across the left ventricular outflow tract (figure 2).

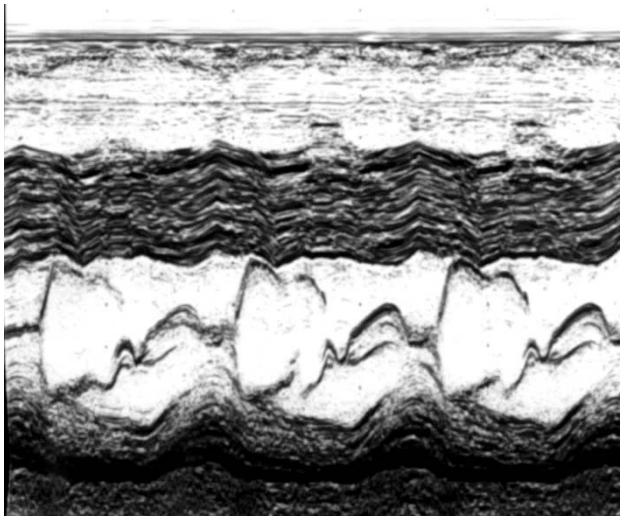

Figure 1. M-mode echocardiogram showing anterior movement of the mitral valve during systole.

We believe that it is important to make the diagnosis of DLVOTO in the critically ill hypotensive patient as the management is different from usual therapy for cardiogenic shock. We would also advocate echocardiography in any patient in shock who appears refractory to inotropic therapy.

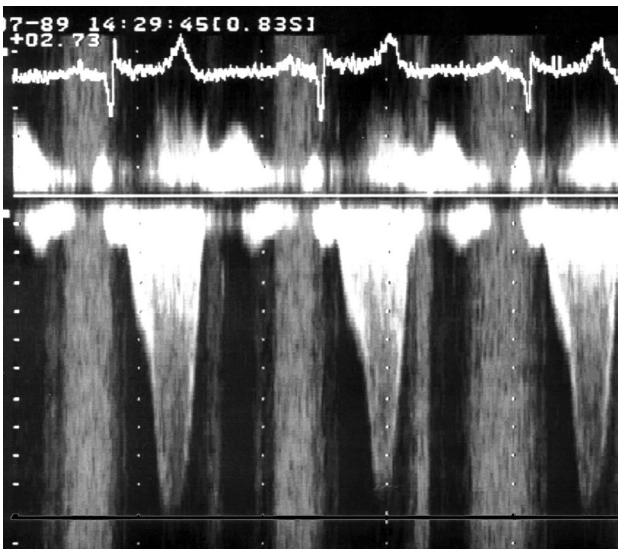

Figure 2. Continuous wave Doppler echocardiogram showing increased velocity across the left ventricular outflow tract due to dynamic obstruction.

Received: 7 August 2002
Accepted: 20 August 2002

REFERENCES

1. Armstrong WF, Marcovitz PA. Dynamic left ventricular outflow tract obstruction as a complication of acute myocardial infarction. Am Heart J 1996;131:827-830.
2. Haley JH, Sinak LJ, Tajik AJ, Ommen SR, Oh JK. Dynamic left ventricular outflow tract obstruction in acute coronary syndromes: an important cause of new systolic murmur and cardiogenic shock. Mayo Clin Proc 1999;74:901-906.
3. Keegan MT, Sinak LJ, Nichols DA, Lanier WL. Dynamic left ventricular outflow tract obstruction in acute coronary syndromes [letter]. Mayo Clin Proc 2000;75:216-217.
4. Vaughn-Whitley K, Mooss A. Dynamic left ventricular outflow obstruction related to atrial fibrillation. Chest 1992;102:1618-1619.
5. Pellikka PA, Oh JK, Bailey KR, Nichols BA, Monahan KH, Tajik AJ. Dynamic intraventricular obstruction during dobutamine stress echocardiography. A new observation. Circulation 1992;86:1429-1432.
6. Turnage WS, Zeller FA, Divertie GD. Dynamic left ventricular outflow tract obstruction. Diagnosis by transesophageal echocardiography in a critically ill patient. Chest 1993;104:1629-1631.