Systolic anterior motion of the mitral valve with left ventricular outflow tract obstruction: A case report in the perioperative setting

Movimiento sistólico anterior de la válvula mitral con obstrucción del tracto de salida del ventrículo izquierdo: un caso clínico en el perioratorio

Dear Editor:

An 84-year-old man with a history of hypertension and diabetes mellitus was diagnosed as having a gastric tumour and proposed for resection. Once in the operating room we first performed a thoracic epidural block with a test dose of lidocaine 2%, 3 mL. Tracheal intubation was carried out with a fibreoptic guidance under spontaneous breathing due to an anticipated difficult airway under sedation with remifentanil and the use of 200 mg of topical lidocaine. Then, we used 50 mg of propofol as the induction drug and desflurane, fentanyl and 0.15% epidural bupivacaine for anaesthesia maintenance. Severe cardiovascular collapse suddenly appeared which was managed with a titrated infusion of norepinephrine. During surgery the patient presented with different episodes of severe hypotension despite increasing norepinephrine doses. A low-dose dobutamine infusion showed no response. The epidural infusion was stopped too. A transesophageal echocardiography (TEE) showed a moderate-to-severe septal hypertrophy with systolic anterior motion of the mitral valve (SAM) and left ventricular outflow tract obstruction (LVOTO). Dobutamine was then discontinued and norepinephrine and volume infusion rates increased. Postoperatively the patient was transferred to the intensive care unit in where a PICCO (Pulsion Medical System, München, Germany) catheter was placed to guide haemodynamic therapy. Global end-diastolic volume (GEDV) and systemic vascular resistances (SVR) were in the low limit of normality despite the use of high doses of vaspressors, and stroke volume variation (SSV) was 14%. Volume expansion was achieved and progressive withdrawal of norepinephrine could be done successfully. The patient awoken and was extubated without any complications 24h later, and transferred to a surgical ward. Afterwards a transthoracic echocardiography was performed, which showed a moderate-to-severe hypertrophy without LVOTO.

SAM is characterized by a displacement of mitral leaflets, especially the anterior one, towards the septum during systole causing LVOTO.\(^1\),\(^2\) Classically, hypertrophic cardiomyopathy has been linked with this entity, but there is growing evidence that other conditions such hypertensive heart disease, diabetes mellitus, myocardial infarction and stress cardiomyopathy can act as predisposing factors to develop this alteration, which is known to be dynamic.\(^1\) Most patients with predisposing factors do not show features of SAM and LVOTO unless a triggering situation producing a decrease in cardiac preload happens, which in turn causes a modification in ventricular shape and in the relationship between mitral valve, left ventricular septum and the outflow tract. Moreover, narrowing of the left ventricular outflow tract may cause mitral regurgitation (MR).\(^1\),\(^2\)

The pathophysiology is not yet fully understood. Some researchers proposed that the increase in flow velocities may exert some traction in mitral leaflets through a Venturi effect,\(^1\),\(^2\) but recent data showed that Venturi forces present in the left ventricular outflow tract are much smaller in their magnitude than previously assumed.\(^2\) Sherrid et al. showed that the initial SAM begins very early in systole at normal flow velocities by a drag force causing an obstruction which would be responsible for the creation of a gradient.\(^3\) This gradient then becomes the major force acting on the mitral leaflet.\(^2\),\(^3\) Also, a decrease in preload is known to be a key factor in the development of SAM and LVOTO in most settings, such as the operating room or the ICU.\(^1\),\(^2\) When this phenomenon occurs, there is a modification in chamber size and in anatomical relationships between mitral valve apparatus and the direction of flow in the left ventricular outflow tract, making it easier to initiate the drag necessary for pushing mitral leaflets towards the septum.\(^2\) At this point not only the anatomical changes are responsible for the start of this alteration, but the hyper-contractility, initially compensating the decrease in preload, may worsen this situation due to an increase in flow velocities in the outflow tract. Several studies have reported evidence of LVOTO during dobutamine stress echocardiography and case reports have shown that inotropic drugs may aggravate SAM and LVOTO.\(^1\),\(^2\) This situation has also been described in cases of increased endogenous catecholamine production such as pheochromocytoma\(^1\) and authors have proposed that LVOTO seen in patients with myocardial infarction might be related to compensatory hyperkinesis in non-infarcted segments.\(^1\) Diabetes mellitus has also been linked to SAM. A group of poorly controlled diabetic patients who were compared to healthy controls both at rest and during an infusion of isoproterenol showed 6.5 times more SAM than the control group. In this

---


R. Navarro Suay\(^a\), M.Á. García Aroca\(^a\), S. Castillejo Pérez\(^b\) y E. López Soberón\(^b\)

\(^a\) Servicio de Anestesiología y Reanimación, Hospital Central de la Defensa Gómez Ulla, Madrid, España

\(^b\) Servicio de Cardiología, Hospital Central de la Defensa Gómez Ulla, Madrid, España

* Autor para correspondencia.
Correo electrónico: r.navarro_suay@yahoo.es (R. Navarro Suay).

http://dx.doi.org/10.1016/j.redar.2013.09.014

---
group of patients SAM was indicative of a hyperkinetic state.1

Definitive diagnostic of SAM and LVOTO is echocardiographic1,2 and TEE has been used in different surgical settings to guide fluid therapy. Echocardiographic dynamic indexes such as vena cava diameters correlate well with fluid responsiveness.4 Dynamic intravascular indexes such as SVV could be a good alternative to monitor haemodynamic therapy.5

In conclusion, SAM and LVOTO could be the final outcome of several entities acting by different paths.1 Decreasing the left-ventricular gradient, increasing preload and minimising heart rate are the key goals of therapy. If volume expansion and vasopressor therapy are conducted without success, β-block therapy may be initiated carefully to maximize the benefits and avoid adverse effects.2

References


R. Mellado*, M. Vendrell, X. Borrat, J. Balust

Servicio de Anestesiología y Reanimación, Hospital Clinic, Barcelona Spain

*Corresponding author.

E-mail address: rmellado@clinic.ub.es (R. Mellado).

http://dx.doi.org/10.1016/j.redar.2013.09.011

Neumoperitoneo masivo por perforación gástrica postintubación traqueal difícil

Massive pneumoperitoneum due to gastric perforation after difficult tracheal intubation

Sr. Director:

En anestesia y reanimación cardipulmonar (RCP), la perforación yatrogénica del estómago es una complicación infrecuente, aunque puede darse en situaciones de sobreinsufilación gástrica por maniobras de ventilación incorrectas1-3, intubación esofágica, endoscopias4, uso de fibrobroncoscopio con insuflación de oxígeno por el canal de trabajo4, compresiones torácicas enérgicas, etc. Así pues, las manifestaciones clínicas y las complicaciones que podríamos esperar son las propias del neumoperitoneo.

Presentamos el caso de un varón de 38 años, sin antecedentes patológicos de interés, que acudió al hospital con herida laterocervical por arma blanca. Se realizó revisión quirúrgica, con anestesia local, observándose una herida de unos 10 cm de trayecto con lesión de la glándula submaxilar, sin afectación de grandes vasos. Durante el procedimiento apareció hemorragia incontrolable por movilización de un coágulo, por lo que se realizó anestesia general. Se llevó a cabo inducción de secuencia rápida y se procedió a realizar la laringoscopia. Esta mostraba edema de la base de la lengua y visión de un grado Cormack-Lehane IV, que requirió de 5 intentos de intubación inefectivos. Se colocó mascarilla laringea tipo Fastrach, obteniéndose ventilación suficiente. A su través, se realizaron 3 intentos de intubación fallidos y, finalmente, se consiguió intubación orotraqueal con fibrobroncoscopio. Se controló la hemorragia y se trasladó a nuestro centro. A su llegada a Urgencias, se mantuvo

Figura 1 Imagen de TAC toracoabdominal en proyección sagital, que muestra neumoperitoneo con compresión de las vísceras abdominales.