Echocardiography for management of low cardiac output state after axillary block in a dehydrated patient with aortic stenosis: case report

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Abstract: We report an episode of transient low cardiac output in a dehydrated elderly woman having an undetected clinically significant aortic stenosis, after an axillary block. Cardiac echography was determinant for management of this patient. Events were considered to be from hemodynamic origin.

Key words: Axillary block; aortic stenosis; transthoracic echocardiography.

Introduction

Elderly patients can have several undetected medical conditions, although looking “well” and not reporting any significant diseases. Those patients often do not have an active life anymore, preventing the anamnestic evaluation of the cardio-pulmonary function. This, added to some predisposing factors, can make them susceptible to an usually safe anesthesia technique. Peripheral nervous blockade is considered to be the safest anesthesia technique for high-risk cardiovascular patients. The vasodilation provoked by uncomplicated axillary perineural injection of local anesthetic (LA) is considered to be minor and to not have any general effects on systemic blood pressure and cardiac output, even for those patients. However, we observed a low cardiac output state triggered by this simple regional technique applied to an elderly woman having an undetected aortic stenosis (AS) and dehydration. Cardiac echography was necessary for diagnostic and management.

Case Presentation

A 87-old women was admitted for a left wrist fracture (Pouteau-Colles’). She had a history of hypertension and was currently taking beta-blocker (bisoprol 100 mg/day) and a combination of diuretics (altizide 15 mg/day and spironolactone 25 mg/day). She was living in an old people’s home, and she was said to be well by her family, but didn’t make any efforts anymore (never used the stairs, always the lift). Family noted two recent episodes of syncope that were assumed to be “minimal strokes” by the patient’s general practitioner. No further investigation had been made for this.

The patients looked calm and cooperative. She weighed about 70 kg for about 165 cm. Her blood pressure (BP) were 180/100 mmHg and heart rate (HR) 60/min. Physical examination and laboratory tests were normal. EKG showed regular sinus rhythm with complete right bundle branch block and inferior and anterolateral repolarisation abnormalities. Chest X-ray showed enlarged cardiac silhouette and clear lung hiles and parenchyma.

The patient fasted during eight hours. She did not receive any premedication. Peripheral IV line was inserted and 500 ml normal saline was given over one hour. Axillary blockade was performed with a nerve stimulator, with 40 ml ropivacaine 0,5% with 1/200000 epinephrine. The procedure was easy and no vascular puncture was noted. Ropivacaine was injected slowly, while continuous verbal contact was kept with the patient along with HR monitoring. No intravenous (IV) medication were given during or after the procedure.

Ten minutes after completion of the procedure, the nervous block was complete and we put the patient on the operating table. The patient started then to complain of “not feeling well”, but she...
could not explain why. She did not have any headache, metallic taste in the mouth, or any other sign of LA toxicity. Surgical procedure was completed in 25 minutes, while the patient was breathing pure oxygen. She was obsessed, keeping saying that her face mask prevented her to breathe, and that she was not well. She did not have any dyspnea. She was lying down in a flat position and had a respiratory rate of 14/min and SpO2 of 98%. Her vitals were stable, BP 130/90 mmHg, HR 72/min. When we put her back in bed, she had nausea and vomiting, along with pallor and systolic BP was 100 mmHg. She had no chest pain, but she started to complain of left arm pain, from the shoulder to the wrist, although this arm was still under anesthesia (anesthesia was confirmed to be excellent with cold test, from the elbow to the fingers). Transthoracic cardiac echography was realized and showed significant aortic stenosis (on parasternal long-axis view), moderate to severe ventricular hypertrophiy, good left ventricle global dynamics, with hyperdynamic small cavities that nearly completely collapse at the end of systole, which is a sign strongly suggesting hypovolemia. Patient was given fluid (500 ml of Hydroxyethyl Starch) and started to feel better, although she had another episode of vomiting). Systolic BP went back to 14. At this time, a systolic ejection murmur (2/6) was audible. The patient received more IV fluids and was then transferred to the ICU for overnight monitoring. She was transferred to the surgical ward the day after with a normal mental status. Transthoracic cardiac echography performed by the ward’s cardiologist showed only moderate AS (surface 1.3 cm²; peak gradient 30 mmHg) with good ventricular function and LV hypertrophy ; the left ventricular cavity was found to be small. Although the AS was estimated to be moderate, it was thought by the cardiologist to be possibly responsible of the observed symptoms, in presence of precipitating factors. Coronary arteries were not investigated because the family of the patient requested to not make any invasive procedure anymore.

**DISCUSSION**

Anesthetetic management for non cardiac surgery of a patient having an AS can be challenging, especially if the AS is severe and symptomatic (1). In this case, elective non cardiac surgery should generally be postponed and valve replacement should be performed (2). If not possible, peripheral regional anesthesia is advised.

Our patient had significant aortic stenosis (i.e. less than 1 cm²). The difference between the evaluation of the ICU cardiologist and the ward cardiologist should be attributed to poor technical conditions. The two recent episodes of syncope described by her family and the visceral pain in the anesthetised arm suggested that the AS was clinically significant.

Our patient was in a severe dehydrated state, as confirmed by transthoracic cardiac echography. She used to drink very few, was taking diuretics and had fasted for more than eight hours. Moreover, efficacious beta-blockade made her unable to become tachycardic in response to hypovolemia. Furthermore, chronic hypertension can lead per se to an hypovolemic status.

Although our patient was obsessed, no other real sign of systemic toxicity were observed, although we tried to unmask them. Although no dosage of LA could confirm it, and although the procedure was easy, without vascular effraction and the ropivacaine contained adrenaline, some minor systemic resorption cannot be ruled out.

We believe that direct vasodilatation of the arm, and some minor systemic effects of LA (negative inotropic effects and some degree of systemic vasodilatation), were the precipitating events of this low cardiac output state.

We think that the observed events were primarily of hemodynamic origin because 1. clinical indices of significant AS were present ; 2. several precipitating factors (dehydration, beta-blockade) were present ; 3. we observed nausea and vomiting with pallor and hypotension, which are typical signs of low cardiac output frequently seen after spinal anesthesia ; 4. we were able to hear a clear systolic ejection murmur only after infusion of fluids ; 5. the patient improved after fluid infusion.

Some minor systemic resorption of epinephrine cannot be ruled out too. However, due to the very low concentration of epinephrine in this case, and to the beta-blockade of the patient, this is difficult to document. Moreover epinephrine has alpha and beta agonist actions that can be opposite. Beta-2 adrenergic effects could have participated to the global vasodilation, but alpha-1 effects could have provoked some minor degree of cerebral vasoconstriction, contributing to the cerebral hypoperfusion.

Transthoracic cardiac echography was an invaluable tool for the management of this patient. Tranesophagal echocardiography is used since a long time for hemodynamic management of patients undergoing cardiac surgery (3), and this
technique has extended to non cardiac surgery (4). Transthoracic echography, allowing repeated pro-, per- and post-operative evaluations, is now increasingly recognised as part of hemodynamic monitoring (5), although it is not a generalised practise. This technique has already been showed to be extremely useful patients with AS in the perioperative period (6). Moreover, in our patient, AS was unknown and the ejection murmur was not easily audible at the time of initial examination because of the hypovolemic status, which made cardiac echography the only technique that could help us to understand the underlying disease.

We did not find any similar case of hemodynamic compromise after an axillary block in the literature. Combined parasacred sciatic and lumbar plexus block in a patient with AS has been shown to induce some degree of hemodynamic instability (7), probably because of the greater volume of the vasodilated lower limb, and because of the possible epidural spread of the LA that can be observed.

CONCLUSION

Although peripheral neural blockade is generally considered to be a safe anesthesia technique for high-risk cardio-vascular patients, we observed that a single peripheral nervous axillary block was able to precipitate hemodynamic compromise in an elderly woman with several predisposing factors (dehydration, beta-blockade, chronic hypertension and unrecognized AS). The hemodynamic instability were obviously multifactorial, but triggered by vasodilation of the arm and some general vasodilation due to some minor resorption of LA. In our patient, cardiac echography were the most useful tool for evaluation and effective goal-directed treatment.

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References