

Swan Ganz Catheter for Diagnosis of Transient Central Diabetes Insipidus after Mitral Valve Replacement

Ishtiaq Sarwar,¹ Lok M. Sinha,² Ambreen Khan,³ Aftab Yunus⁴

Abstract

Transient Diabetes Insipidus (DI) occurring in a patient undergoing open heart surgery is a rare occurrence. In this case report, we are presenting a 30 years old female patient with past history of stroke who underwent redo mitral valve replacement developed polyuria. The diagnosis of hypovolemia was made with the help of swan ganz catheter. The patient responded to desmopressin and completely recovered seven days after surgery. It is possible that transient cerebral ischemia given her history of Stroke resulted in the dysfunction of osmotic receptors in the hypothalamus or hypothalamus – pituitary axis during Cardiopulmonary Bypass (CPB). Therefore, we concluded that central DI is a probable cause of polyuria after CPB.

Keywords: Transient Diabetes Insipidus, Swan Ganz Catheter, Mitral valve replacement.

Introduction

Diabetes insipidus is an uncommon endocrinologic disorder. It could be either Central, caused by lack of antidiuretic hormone (also called vasopressin) or Nephrogenic, resulting from failure of kidneys to respond to antidiuretic hormone. As a cardiac surgeon we come across different endocrinologic disorders, the most common being diabetes mellitus, hypothyroidism and hyperthyroidism.¹ Diabetes Insipidus is a very rare entity occurring in cardiac surgical patients.²⁻³ In this case report we will discuss the experience of transient diabetes Insipidus in a patient undergoing redo mitral valve replacement and role of swan ganz catheter in its diagnosis.

Case Report

The patient was 30 years old female with previous history of cerebrovascular accident with no gross residual neurologic deficit. Her past history was significant for open mitral valvotomy 16 years ago for pliable mitral stenosis. There was no prior history of polydipsia, polyuria, or head injury. In addition, the patient did not have a familial history of polyuria. Preoperatively, her blood pressure was 115 / 60 mmHg and heart rate was 85 beats per minute. Currently, she presented with sev-

Sarwar I.¹
Department of Cardiac Surgery, Mayo Hospital, King Edward Medical University, Lahore – Pakistan

Sinha L.M.²
Department of Cardiac Surgery, Mayo Hospital, King Edward Medical University, Lahore – Pakistan

Khan A.³
Department of Anesthesia, Mayo Hospital, King Edward Medical University, Lahore – Pakistan

Yunus A.⁴
Department of Cardiac Surgery, Mayo Hospital, King Edward Medical University, Lahore – Pakistan

ere mitral regurgitation with pulmonary hypertension (60 mm. Hg). Her only complaints were shortness of breath on exertion (NYHA III) and palpitations. Rest of her investigations were within normal limits.

She was taken to the theatre after informed consent and monitoring lines included a right radial arterial line and a central venous line inserted through right internal jugular vein. Redo sternotomy was done and mitral valve was replaced with 27 mm mechanical bileaflet mechanical prosthesis, preserving both the mitral leaflets, without any complications. The cross clamp time was 80 minutes and bypass time was 106 minutes. There was no inotropic support required postoperatively.

She was extubated in ICU after 4 hours of mechanical ventilation. However, she was reopened for bleeding during night and about 2 litres of blood and blood clots were removed, her blood pressure was maintained during the period. No bleeding point was found. She was extubated next morning. She passed more than 11 litres of urine postoperatively in first 15 hours. This high volume of urine was ignored as she have been transfused with 08 units of blood (3.6 litres) and around 5 litres of saline. Negative fluid balance was 3318 ml. She remained fine during the day, but became hypotensive and tachycardic during night. Her blood gases showed severe metabolic acidosis. She was started on inotropes and her acidosis was corrected. Her central venous pressure as measured from central line was 19 cm of water. As we could not get her echo done, a swan ganz catheter (Fig. 1) was inserted through left sub-clavian vein.

The basic aim of swan ganz catheter insertion was to check pulmonary artery pressures and make deci-



Fig. 1: Swan Ganz Catheter and its Sheath.

sions regarding start of milrinone infusion for pulmonary hypertension. The right atrial pressure was 2 mm. Hg and pulmonary artery pressure was 30/12 mm. Hg. She was given IV fluids and inotropes were stopped. She again started passing high volumes of urine. Her serum sodium was 155 mmol/L and urine specific gravity was 1002. Provisional diagnosis of Diabetes Insipidus was made. She was treated with desmopressin nasal spray and her urine output slowly lessened. She was treated with desmopressin for 03 days and then stopped. She made good recovery and was discharged on 7th postoperative day in satisfactory condition.

Discussion

Diabetes insipidus (DI) is characterized by excessive urination and thirst. The cause of diabetes insipidus is either deficiency of vasopressin (central or neurogenic) or inability of the kidneys to respond to vasopressin (Nephrogenic). Central diabetes insipidus usually occurs with hypopituitarism. The hypopituitarism has been reported in 19 percent of patients with ischemic stroke and it may present as an isolated deficiency in most cases.⁴ In cardiac surgical patients central diabetes insipidus results probably from dysfunction of osmotic receptors in hypothalamus or hypothalamus – pituitary axis from transient cerebral ischemia during cardiopulmonary bypass or decreased vasopressin release from pituitary by altered left atrial non-osmotic receptor function by cardiac standstill.²⁻³

In this patient there was history of cerebrovascular accident with complete recovery that decreased the threshold of ischemia in current surgery. During cardiopulmonary bypass she most probably had oedema / Ischemia around damaged area of brain leading to decreased vasopressin release from posterior pituitary. Her urine output was significantly increased postoperatively as compared to other patients following cardiopulmonary bypass due to haemodilution. She became dehydrated and went into hypovolemic shock. Her central venous pressure was high giving false impression of right heart failure secondary to pulmonary hypertension. The swan ganz catheter was inserted to see pulmonary artery and right heart pressures. The right atrial pressures were 2 mm Hg, right ventricular pressures were 30/1 mm Hg and pulmonary artery pressures were 30/12 mm Hg. Thus the swan ganz catheter made it possible to rule out pulmonary hyperten-

sion, right heart failure and diagnosis of hypo-volemia was made. She was given IV fluids and then desmo-

pressin nasal spray for suspected diabetes insipidus.

The swan ganz catheter is inserted percutaneously by seldinger technique through internal jugular or subclavian vein (Fig. 2). It allows direct simultaneous measurement of pressures in right atrium, right ventricle, pulmonary artery and the filling pressure (“Wedge” pressure) of the left atrium. It is used for direct / indirect measurement of several hemodynamic parameter such as Cardiac Output (thermo-dilution method), Cardiac Index, Systemic Vascular resistant in critically ill unstable patients and aids in decision making capability.

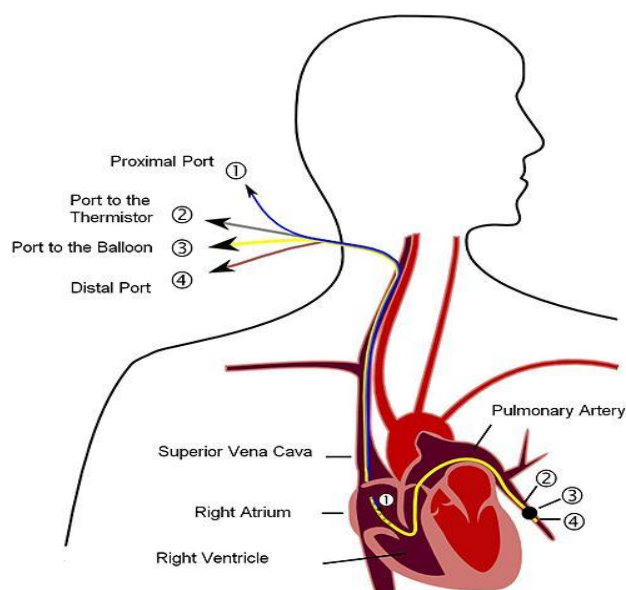


Fig. 2: Diagram showing insertion of Swan Ganz Catheter through right internal jugular vein.

The use of this catheter is limited⁵ by the complications namely haematomas, infection, balloon rupture, arrhythmias,⁶⁻⁷ pulmonary artery rupture,⁸⁻⁹ and knotting of catheter.¹⁰⁻¹¹

Conclusion

Although there are other methods for diagnosis of Central Diabetes insipidus such as MRI, In Pakistan in general, and other developing countries where Cost

comes first, Swan Ganz Catheter is cost effective simple bed side procedure for assessment and treatment of such rare self limited endocrine disorder following open heart surgery.

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