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RESEARCH ARTICLE

SELF LIMITING TRANSIENT POLYURIA POST AORTIC VALVE REPLACEMENT

Dr. Akshaya Parthiban[1], Dr. Sameer Kadam[2], Dr. Aditya Nayak[3] and Dr. Vishwas Sathe[4]

1. PG Resident Year I, Department of General Surgery, MGM Medical College and Hospital,Navi Mumbai.
2. HOD, Department of General Surgery, MGM Medical College and Hospital, Navi Mumbai.
3. AssistantProfessor, Department of General Medicine, MGM Medical College and Hospital, Navi Mumbai.
4. Professor, Department of Anaesthesia, MGM Medical College and Hospital, Navi Mumbai.

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Abstract

Polyuria is not an uncommon perioperative complication, following coronary artery bypass surgery. Diabetes insipidus (DI) results from inadequate secretion of antidiuretic hormone (ADH) from the pituitary gland (Central DI)or the absence of the normal response by the renal tubules to ADH(Nephrogenic DI).Here we present a 55 years old male who underwent Aortic valve replacement. Postoperatively he developed polyuria and was diagnosed with Diabetes insipidus (DI). It's most likely due toalteration within the left atrial non-osmotic receptor during cardioplegia.It can also be a dysfunction of osmotic receptors in the hypothalamus because of transient cerebral ischemia resultingdue to microthrombi during Cardio-Pulmonary Bypass.The patient recovered from symptoms without any administration of vasopressin.

Introduction:-

Polyuria is not an uncommon perioperative complication, following coronary artery bypass surgery[1].Diabetes insipidus (DI)is characterized by hypotonic polyuria greater than 3 liters/24 hours in adults and persists even during water deprivation. This disease results from inadequate secretion of antidiuretic hormone (ADH) from the pituitary gland (Central DI)or the absence of the normal response by the renal tubule to ADH(Nephrogenic DI) other are dipsogenic DIand gestational DI[2]. In individuals undergoing cardiopulmonary bypass (CPB), several endocrine changes due to surgical stress can occur[3,4]. In the present report, we describe a case of transient polyuria that developed after aortic valve replacement.

Case Report

A 55 years old male underwent elective aortic valve replacement for severe aortic stenosis.He had no previous history of polyuria,polydipsia, or head injury. The patientis hemodynamically stable.Basic hematological and biochemical parameters were within normal limits.The procedure lasted for 5hours and 15 minutes with a CPB time of 170mins and an aortic cross-clamp time of 107mins.Cardiac protection was provided usinga cold-blood cardioplegic solution. There were no intra-operative episodes of hypotension.Intra-operative urine output was 1000ml.On POD1 patient started having a urine output of 100ml/hr with a total output of 2280ml/24hrs,followed by the next day he passed 5400ml/24hrs (Fig.1A).

Corresponding Author:- Dr. Akshaya Parthiban
Address:- PG Resident Year I,Department of General Surgery,MGM Medical College and Hospital, Navi Mumbai.

The patient was observed to be lethargic and complained of nausea.His mental status was not altered and there were no clinical signs of neurologic deficit. Serum glucose and sodium concentrations were 187 mg/dL and 139 mEq/L. The specific gravity of the urine was 1.005. Serum and urine osmolality was 278.41 and 158.22 respectively(Fig. 1B). The urine was also negative for protein, glucose, and occult blood. Other laboratory findings did not reveal any drastic changes compared to preoperative values.

On the second postoperative day, the patient's urine output continued up to 200-300ml/hr and he was referred to a nephrologist. Considering his laboratory values patient was diagnosed with diabetes insipidus. The patientwas managed with fluid replacement therapy.Serum sodium level was maintained within normal levels, and one episode of hyponatremia was noted.On POD7 he had a urine output of 1500ml/24hrs. The patient had no symptoms or signs of diabetes insipidus. The patientwas discharged on POD12.

Fig-1.A:-

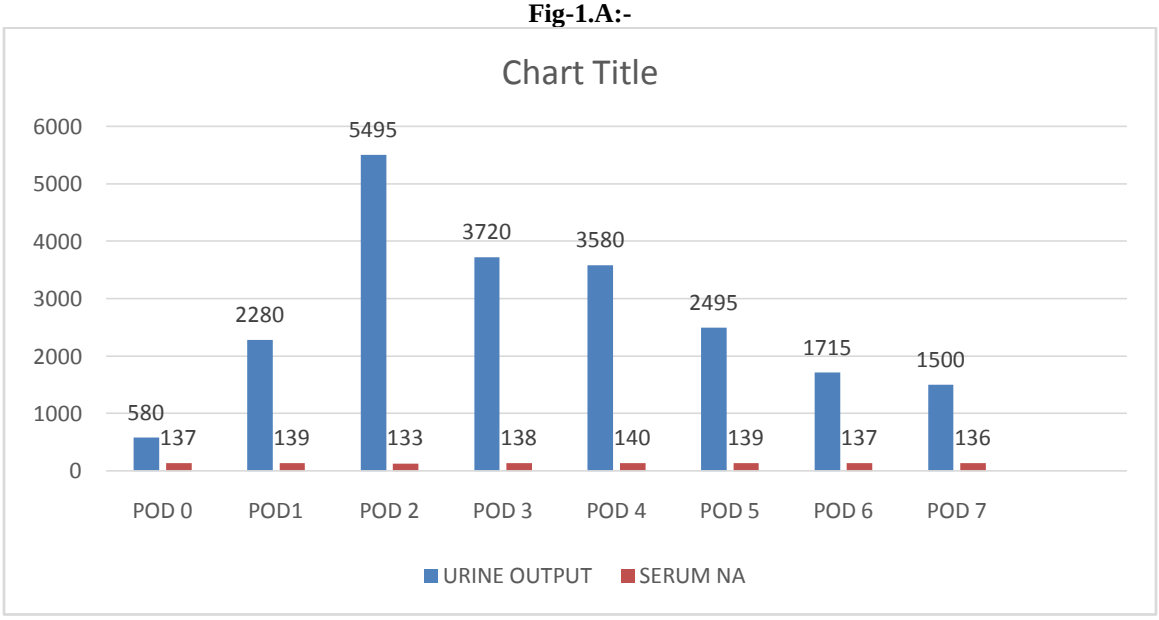
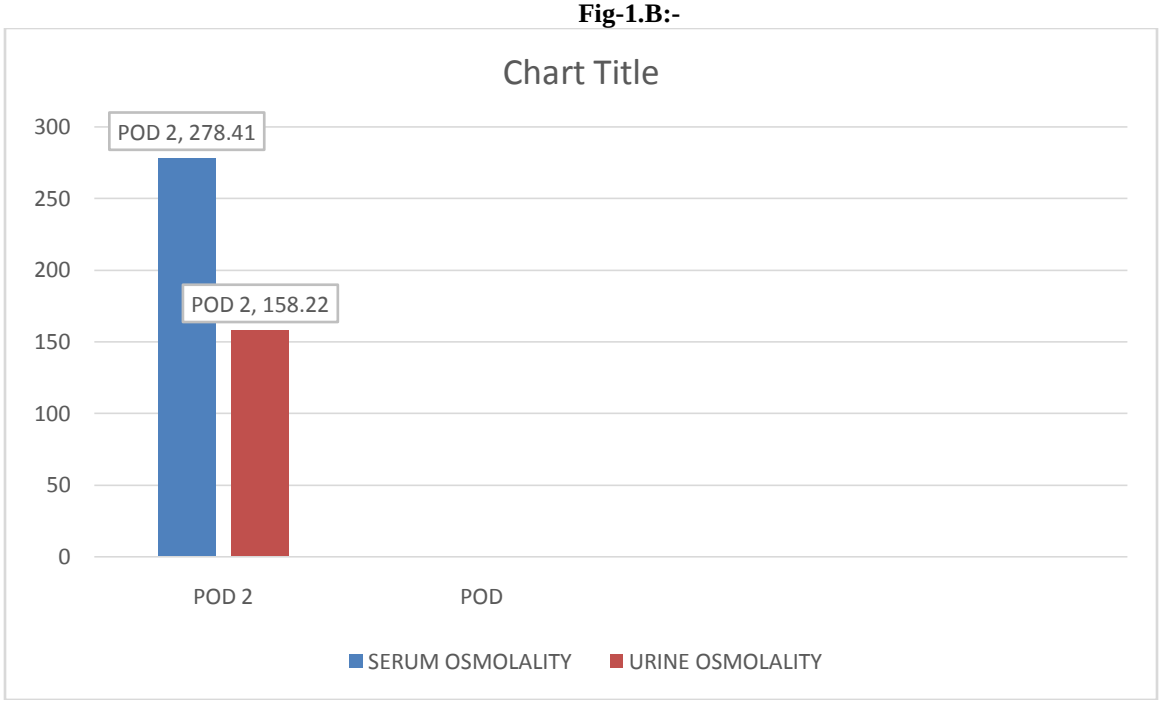


Fig-1.B:-



Discussion:-

Here we have presented a rare case of transient polyuria which developed post Aortic valve replacement. Changes such as fluid, electrolyte, and acid-base imbalances; renal complications, embolic events, and neuro-endocrine changes can be induced by CPB[5].

Paraventricular and supraoptic nuclei of the hypothalamus produce ADH along the hypothalamus-pituitary axis and are stored in the pituitary gland.The control of ADH synthesis and release consists of two ways; osmotic and non-osmotic. Variation in extracellular fluid (ECF) volume acts as a non-osmotic stimulus.Decreased extracellular fluid levels, stimulate the release of ADH. This is conveyed throughvolume receptors which are situated in the left atrium,aortic arch, and carotid artery.In an osmotic stimulus increase in plasma osmolality results in the release of ADH from the hypothalamus.ADHis released by both, osmotic and non-osmotic stimuli, ADH released acts directly on kidney[6].

Sheehan and Murdock described postpartum pituitary necrosis in patients who were in hemorrhagic shock after parturition[7].This also reinforced that necrosis withinthe posterior pituitary is also a possible cause of diabetic insipidus[8].In our study, we couldn'tforget the possibility of temporary cerebral ischemia ensuing in osmotic stimulus receptor disturbance or hypothalamus-pituitary axis even though no excessive or permanent posterior pituitary harmoccurred.MRI brain was not done in this case so posterior pituitary damage couldn't be excluded.

According toa fewreports[9,10,11], almost 1%to 6% of people have a prevalence of developing cerebral ischemia during open-heart surgery. It is also evident that 6%to 12% of CABG patients have subclinical carotid stenosis more than 50% preoperatively;however,they don't have any bruit on clinical examination[12]. Since we no longercarry out a carotid doppler study, subclinical carotid stenosis couldn't be excluded.Transient subclinical cerebral ischemia can also be induced by CPB[13].

In our diagnosis, we hadtaken into consideration other causesof polyuria such as iatrogenic fluid overload, osmotic diuresis, and recovery from acute tubular necrosis. These situationshad been excluded based ona review of the surgeon's prescription and renal function test.

Next,we have to consider non-osmotic disturbance. A decrease in left atrial volume results in diminished vagus parasympathetic stimulation eventually leading to the release of ADH.An increase in the left atrial volume causes viceversa. During CPB the left atrial non-osmotic receptor function could be transiently altered by a standstill heart resulting in suppression of ADH release.Other factors contributing to polyuria are myocyte rigidity and an increase in natriuretic peptide secretion due to volume loading during the procedure. DI post-cardiac surgery is transient and responds to desmopressin replacement[1].

Our case is one of the casesreported in adults where desmopressin was not required.

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