



THE AGA KHAN UNIVERSITY

eCommons@AKU

Section of Cardiothoracic Surgery

Department of Surgery

December 2005

A case of transient diabetes insipidus following cardiopulmonary bypass

Omer Ashraf
Aga Khan University

Hasanat Sharif
Aga Khan University, hasanat.sharif@aku.edu

Mansoor Shah
Aga Khan University

Follow this and additional works at: https://ecommons.aku.edu/pakistan_fhs_mc_surg_cardiothoracic

 Part of the [Surgery Commons](#)

Recommended Citation

Ashraf, O., Sharif, H., Shah, M. (2005). A case of transient diabetes insipidus following cardiopulmonary bypass. *Journal of Pakistan Medical Association*, 55(12), 565-566.

Available at: https://ecommons.aku.edu/pakistan_fhs_mc_surg_cardiothoracic/130

A case of transient diabetes insipidus following cardiopulmonary bypass

Omer Ashraf¹, Hasanat Sharif², Mansoor Shah³

Final Year Medical Student¹, Departments of Surgery² and Medicine³, The Aga Khan University Hospital, Karachi, Pakistan.

Abstract

Cardiopulmonary bypass surgery has been linked with a number of postoperative complications. One of the frequently reported physiological alterations is the relative diuresis seen in the immediate post-op period. Rarely reported though is the development of full-blown diabetes insipidus in such patients. The etiology is unknown and has only been hypothesized in the past. We present the clinical course of a 54 year old male who developed transient diabetes insipidus post bypass surgery with subsequent recovery following exogenous vasopressin administration. The physiological alteration leading to the development of diabetes insipidus in a small fraction of bypass patients remains unknown. We propose that the variation in natriuretic peptide levels in the post-bypass period could account for the transient event.

Introduction

Diabetes insipidus (DI) is rarely encountered after bypass surgery. The phenomenon is poorly understood. We present a patient who developed transient diabetes insipidus following cardiopulmonary bypass (CPB), and hypothesize further on the subject.

Case Report

A 54 years old male underwent an uneventful on-pump total arterial revascularization procedure for three vessel coronary artery disease. The procedure lasted 107 minutes, with aortic cross clamp time of 75 minutes. The minimum temperature during the procedure was 33°C.

On the first postoperative day his urinary output increased to around 250 ml/hour. There were no clinical signs of any other endocrine or neurological deficit and laboratory work up revealed serum electrolytes, creatinine and urea nitrogen to be within the normal range. He was not receiving any diuretics. Fluid replacement was initiated while he continued to have polyuria. On the fifth postoperative day, after a provisional diagnosis of DI was made, intranasal desmopressin (synthetic vasopressin) was initiated. His urinary output declined thereafter, taking another two days to return to normal. Since then he has remained symptom free with no complaints of altered urinary output, at follow up visits.

Discussion

CPB can produce a myriad of physiologic disturbances, including altered fluid, electrolyte and acid-base balance, renal compromise, embolic events as well as neu-

roendocrine changes. Despite the abundance of conflicting reports regarding the variations in antidiuretic hormone (ADH) levels during and after CPB, DI is rarely encountered in the post-bypass patient.

While the etiology remains to be established the pathology in this patient was seemingly ADH deficiency. This can be established from the rapid decline in urinary output coupled with a relative increase in its osmolality after desmopressin administration in this patient. Classified under central DI, a variety of causes could lower ADH levels in such patients.¹

The lack of any associated neuroendocrine findings and the transient nature of the event make a neurological cause an improbable one. As Kuan et al² had argued, this appears to be more of a functional discrepancy than a morphological lesion. They attributed the polyuria to a transient malfunction of the left atrial nonosmotic receptors for vasopressin release, owing to CPB and cold cardioplegia. This occurred in a fraction of patients already having selective osmoreceptor dysfunction. The time it took for the nonosmotic receptor to regain its function was the time it took for the polyuria to diminish.

The above mentioned theory is the only formally proposed pathogenesis regarding this subject in literature. Whilst being plausible, it does not offer a precise explanation as to what caused the left atrial nonosmotic receptors to lose and then recover their function in a matter of days. The lack of evidence based medicine on the issue leaves the room open for further hypothesis.

Volume loading during CPB results in stretching of the cardiac myocytes. This leads to an augmentation in the level of natriuretic peptides after CPB.³ Peaking early in the first week⁴ and then gradually declining, this occurrence has been known to be associated with post-bypass diuresis.³ The phenomenon can result in polyuria not only through the primary action of natriuretic peptides on kidney, but also through inhibition of the vasopressin neuraxis. This would result in low vasopressin levels subject to correction by exogenous vasopressin administration, as seen in this patient as well as the three patients reported by Kuan et al.² With the timely decline in natriuretic peptide release, the ADH levels would rise again and urinary output would normalize.

The physiology of natriuretic peptides is interlaced around a number of factors. The fact that it could lead to DI in a small fraction of patients undergoing CPB, could be due to a selective disturbance in any one of the associated

factors in that subset of population. Alterations in gene expression or receptor sensitivity are possible candidates. Further research is warranted to ascertain the above mentioned postulation and investigate the source of this suggested aberration in the natriuretic peptide physiology in a small subset of patients undergoing cardiac surgery.

In conclusion, our patient was another one in the small series of patients who underwent transient full-scale DI after CPB surgery. The clinical and laboratory picture appears to be suggestive of a physiological rather than an anatomical anomaly to be the cause of vasopressin deficiency in such patients. Even though diverse ideas may be conjectured, further understanding of the neuroendocrine inter-

actions of the arginine vasopressin system and their variation with cardiac bypass is required before a pathogenesis is conclusively established.

References

1. Laczi F. Diabetes insipidus: etiology, diagnosis, and therapy. *Orv Hetil* 2002;143:2579-85.
2. Kuan P, Messenger JC, Ellestad MH. Transient central diabetes insipidus after aortocoronary bypass operations. *Am J Cardiol* 1983;52:1181-3.
3. Schaff HV, Mashburn JP, McCarthy PM. Natriuresis during and early after cardiopulmonary bypass: relationship to atrial natriuretic factor, aldosterone, and antidiuretic hormone. *J Thorac Cardiovasc Surg* 1989;98:979-86.
4. Song MH, Kobayashi Y, Michi H. Clinical implication of atrial and brain natriuretic Peptide in coronary artery bypass grafting. *Asian Cardiovasc Thorac Ann* 2004;12:41-6.