



Shock Following Percutaneous Coronary Intervention As A Manifestation Of Diabetes Insipidus

Younes Nozari¹, Abbas Andishmand², Akbar Shafiee^{3,4}

1-Department of Interventional Cardiology, Tehran Heart Center, Tehran University of Medical Sciences, Tehran, Iran

2-Department of Cardiology, Afshar Heart Hospital, Shahid Sadoughi University of Medical Sciences, Yazd, Iran.

3-Department of Community Medicine, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran.

4-Department of Cardiovascular Research, Tehran Heart Center, Tehran University of Medical Sciences, Tehran, Iran.

ABSTRACT

The shock syndrome following percutaneous coronary intervention (PCI) is a life-threatening complication, accompanied by poor outcomes. The most common causes of this problem are bleeding and severe left ventricular systolic dysfunction; although rare complications could happen, they are still challenging. In this article we reported a 47-year-old man who suffered from complications following PCI, manifested as unexplained shock, which was resistant to conventional management.

Overestimating the role of the patient's underlying disorder as the cause of the hypotension led to delay in diagnosis and treatment; however, obtaining a thorough familial history revealed diabetes insipidus (DI) as the most probable cause of the hypotension despite normal electrolyte levels at the time of admission. The patient dramatically responded to DI management.

Persistent hypotension or shock after PCI could result from an unusual or rare etiology. Precise history taking and attention to clinical findings are essential for an accurate diagnosis and timely treatment of this life-threatening condition indeed.

ARTICLE INFO

Date of acceptance: 18 May, 2018

KEYWORDS

Shock syndrome; Diabetes insipidus; percutaneous coronary intervention

CORRESPONDING AUTHOR

Akbar Shafiee

Research Assistant, Department of Cardiovascular Research, Tehran Heart Center, North Kargarst, Tehran, Iran.

Tel: +98 21 8802 9257

Fax: +98 21 8802 9256

Email: dr_shafiee@alborzi.com



CITE THIS PAPER AS

Y.Nozari, A.Andishmand, A.Shafiee. Shock Following Percutaneous Coronary Intervention As A Manifestation Of Diabetes Insipidus. *Sch Med Stud J*.2019;1(1):21-24.

INTRODUCTION

Shock or circulatory failure following percutaneous coronary intervention (PCI) is a life-threatening condition that requires immediate diagnosis and management [1]. Shock can result from a wide variety of factors and mechanisms, including hypovolemic, cardiogenic, obstructive, and distributive etiologies. In the setting of PCI, periprocedural hypotension with hemodynamic impairment is commonly due to major bleeding or left ventricular systolic dysfunction [2,3].

In the present study, we report a rare case with unexplained, severe refractory hypotension immediately following a successful PCI procedure.

CASE PRESENTATION

A 47-year-old man was referred to our hospital for elective PCI in a stable condition. The patient had a history of recent anteroseptal myocardial infarction. His coronary artery disease risk factors were systemic hypertension and low serum's high-density lipoprotein. The medications that he used were aspirin, nitrate, atorvastatin, metoprolol, and clopidogrel. He declared no history of specific medical condition. In addition, his physical examination was unremarkable, and his hemodynamic condition was stable. The patient's serum high-density lipoprotein was 34 mg/dl, Na was 140 mg/dl and K was 3.4 mg/dl. Other laboratory tests, including the cardiac serum markers were within normal ranges. The ECG showed sinus rhythm and QS, ST-segment elevation, and T-wave inversion at V1 to V4 leads. Two-dimensional echocardiography showed severe left ventricular systolic dysfunction with an ejection fraction of 25-30% and septal, apical hypokinesia. He was treated by clopidogrel 600 mg, aspirin 325 mg, and atorvastatin 80 mg before the PCI procedure.

PROCEDURAL COURSE

After prep, drape, and under local anesthesia with lidocaine 2%, an arterial sheath (6F) was inserted in the right femoral artery, following a guiding catheter (XB, 3.5, 6F, Cordis, USA) was engaged in the left main coronary artery. After an intracoronary injection of nitroglycerin 100 µg, selective coronary angiography revealed a total occlusion of the left anterior descending artery at the mid portion with a thrombolysis in Myocardial Infarction (TIMI) flow grade of 0. The diagonal artery had 99% stenosis at the mid portion with a TIMI flow grade of 3.

Intravenous unfractionated heparin 7500 u was used as a procedural anticoagulant. An intermediate guidewire (Asahi Intec, Japan) was negotiated easily and positioned distally. The lesion was thereafter predilated to a diameter of 2 mm with a 20-mm long, compliant balloon (Maverick, Boston Scientific, USA), and a TIMI flow grade of 3 was established. The operator opted for a 3-mm diameter and an 18-mm long drug-eluting stent (Xience Prime, Abbott Vascular, USA), which was subsequently deployed successfully.

The next step was to perform PCI on the diagonal artery. Primarily a hydrophilic guide wire (Pilot 50, Abbott Vascular, USA) was passed across the tight lesion of the diagonal branch, and then a bare metal stent (3 mm in diameter and 18 mm in length) (Multilink, Abbot Vascular, USA) was deployed.

After the procedure, the patient complained of weakness,

chest pain, and perspiration and his blood pressure dropped to 80/60 mm Hg, which was attributed to multiple injections of nitroglycerin. Nevertheless, his hemodynamic was relatively stabilized via a free infusion of 500 ml isotonic saline. Final coronary angiograms revealed good expansion of both stents with normal antegrade flow grades and without any dissection or perforation. The patient was subsequently transferred to the Post-Catheterization Unit.

POST-PROCEDURAL COURSE

The patient's post-procedural course was complicated with prolonged hypotension and retrosternal chest pain with agitation and severe perspiration. The blood pressure was 70/50 mm Hg, the heart rate was 120 beats per minute, flat jugular venous pressure, and respiratory rate of 30 per minute; however, no pulmonary rale was auscultated. The patient also had severe abdominal distention, but there was no visible bleeding. Bed-side abdominal ultrasonography showed severe distention of the urinary bladder, for which a urinary in-dwelling catheter was inserted and 700 ml clear urine was drained.

Serial ECGs demonstrated no new findings except for sinus tachycardia, and there were no significant changes in hemoglobin and hematocrit. Echocardiography ruled out cardiac tamponade and recent wall motion abnormality. Fluid therapy was initiated for the clinical assessment of hemodynamic signs. After infusing 2000 ml of normal saline and dopamine (10 mic/kg/min), the patient's blood pressure temporarily rose to 100/80 mm Hg and he suddenly experienced nausea and vomiting, which was relieved by an intravenous injection of metoclopramide and discontinuation of dopamine. Additionally, due to suspicion of anaphylactoid reactions, hydrocortisone 200 mg was injected intravenously.

The patient was transferred to the Catheterization Laboratory in order to measure hemodynamic indices for better diagnosis and management. Meanwhile, the patient's brother revealed that the patient was on specific drugs for polyuria and familial diabetes insipidus. Therefore, the management strategy was immediately changed: serum electrolyte level was requested, volume replacement was initiated, and intranasal Minirin 20 µg (desmopressin acetate) was administered. After a few minutes, the patient's blood pressure increased, his heart rate decreased demonstrably, and his hemodynamic conditions showed complete improvement.

The patient was closely observed for forty-eight hours. Laboratory tests, including troponin I and CK-MB, were normal and serial ECGs showed no changes. At the discharge, the patient was asymptomatic and well oriented.

nd planning.

DISCUSSION

Following PCI, hypotension and shock, which is a form of severe hypotension accompanied by the hypoperfusion of vital organs, has a broad diagnosis. Prompt diagnosis and immediate management of this clinical syndrome has a vital importance. There is also a distinct possibility of stent thrombosis resulting from hypotension [4]. Prolonged hypotension can beget an increase in anaerobic metabolism in order to maintain ATP production, and this metabolic shift could in turn give rise to lactic

acidosis. The degree of hyperlactemia and metabolic acidosis is a sign of organ failure and poor patient prognosis [5-7]. The causes of hypotension complicating PCI and cardiac catheterization are related to drugs, anaphylactoid reactions, dehydration, vasovagal reactions, bleeding, cardiac tamponade, iatrogenic injury to the left main coronary artery or right coronary artery, coronary artery dissection, acute stent thrombosis, severe left ventricular systolic dysfunction, cardiac arrhythmias; in addition, it can rarely occur due to air embolism, cerebrovascular accident, aortic dissection, or pulmonary embolism [3,8]. Our patient did not have the aforementioned problems and was only a case of familial diabetes insipidus.

Central diabetes insipidus manifests with polydipsia and polyuria. Diagnosis is based on low serum levels of antidiuretic hormone and low osmolar urine (<300 mosm). The well known etiologies of central diabetes insipidus are injury to the central nervous system due to neoplastic or autoimmune disease, hypothalamic or pituitary surgery or ischemia, radiation to the brain, infections (e.g. meningitis and encephalitis), cerebral edema, and intracranial hemorrhage [9]. Nevertheless, 30-50% of cases are considered idiopathic. Hereditary forms account for less than 10% of diabetes insipidus patients which has been observed in clinical practice, and mutations in the neurophysin II coding region of the antidiuretic hormone gene which have been discovered [10,11].

The patient had clinical signs of shock in the presence of excessive urine production, which was immediately relieved via intranasal desmopressin and volume replacement. In this patient, precipitating factors of severe hypotension were long-lasting deprivation of fluids before the procedure and overdiuresis following the use of the contrast agent during PCI. The patient informed us regarding his underlying disease in the first place; we could have prevented the periprocedural hypotension and shock by the infusion of copious volumes of isotonic saline and use of desmopressin. The present rare case underscores the significance of accurate history taking. Indeed, if our patient's underlying disease had not been disclosed, he could have been subjected to unnecessary diagnostic tests and ineffective management strategies and eventually might have experienced a life-threatening outcome.

CONCLUSION

It was ultimately attained that periprocedural shock following PCI requires immediate diagnosis and treatment, and the salient point to be taken into consideration is that unexplained hypotension or shock may be triggered by rare causes. Attention to clinical findings and accurate history taking are, therefore, fundamental to a clear diagnosis and treatment.

ACKNOWLEDGMENT

This case report was supported by Tehran Heart Center, Tehran University of Medical Sciences. The authors sincerely thank the nursing staff of the Catheterization Laboratory and Post-Catheterization Coronary Care Unit of Tehran Heart Center.

CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

REFERENCES

1. S. Valente, C. Lazzeri, S. Vecchio, C. Giglioli, M. Margheri, P. Bernardo, et al. Predictors of in-hospital mortality after percutaneous coronary intervention for cardiogenic shock. *International journal of cardiology*. 2007;114(2):176-82.
2. Eng MH, Moses JW, Teirstein PS. Complications of Percutaneous Coronary Intervention. In: Topol EJ. *Textbook of Interventional Cardiology*. Elsevier. 6th edition. pp 357-371, 2012.
3. G. Dangas, I. Iakovou, E. Nikolsky, E. D. Aymong, G. S. Mintz, N. N. Kipshidze, et al. Contrast-induced nephropathy after percutaneous coronary interventions in relation to chronic kidney disease and hemodynamic variables. *The American journal of cardiology*. 2005;95(1):13-19.
4. Ragoša M. Retroperitoneal bleeding. In: *Cases in Interventional Cardiology*. Elsevier – Saunders, pp 135-8, 2011.
5. J. Bakker, P. Gris, M. Coffernils, R. J. Kahn and J. L. Vincent. Serial blood lactate levels can predict the development of multiple organ failure following septic shock. *The American journal of surgery*. 1996;171(2):221-26.
6. M. H. Weil, J. Leavy and E. C. Rackow. Prognosis in shock. *Anaesthesia*. 1986;41(1):80-2.
7. A. Meregalli, R. P. Oliveira and G. Friedman. Occult hypoperfusion is associated with increased mortality in hemodynamically stable, high-risk, surgical patients. *Crit Care*. 2004;8(2):R60-5.
8. Ragoša M. Complications of cardiac catheterization. In: *Cardiac Catheterization*. Elsevier – Saunders, pp 21-31, 2010.
9. A. N. Makaryus and S. I. McFarlane. Diabetes insipidus: diagnosis and treatment of a complex disease. *Cleveland Clinic journal of medicine*. 2006;73(1):65-71.
10. F. Rauch, C. Lenzner, P. Nürnberg, C. Frömmel and U. Vetter. A novel mutation in the coding region for neurophysin II is associated with autosomal dominant neurohypophyseal diabetes insipidus. *Clinical endocrinology*. 1996;44(1):45-51.
11. D. R. Repaske, R. Medlej, E. K. Gültekin, M. Krishnamani, G. Halaby, J. W. Findling, et al. Heterogeneity in clinical manifestation of autosomal dominant neurohypophyseal diabetes insipidus caused by a mutation encoding Ala-1 → Val in the signal peptide of the arginine vasopressin/neurophysin II/copeptin precursor. *Journal of Clinical Endocrinology & Metabolism*. 1997;82(1):51-56.