Clinical Case Report

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Tolvaptan rescue contrast-induced acute kidney injury

A case report

Wei-Chieh Lee, MD[a,b,*], Hsiu-Yu Fang, MD[a], Chih-Yuan Fang, MD[a]

Abstract

Rationale: Contrast-induced acute kidney injury is one of the most serious adverse effects of contrast media and is related to three distinct but interacting mechanisms: medullary ischemia, formation of reactive oxygen species and direct tubular cell toxicity, especially in the patients with chronic kidney disease. The strategies of treatment, including stabilization of hemodynamic parameters and maintenance of normal fluid and electrolyte balance, were similar to the management of other types of acute kidney injury.

Patient concerns: A 58-year-old woman experienced acute oligouria after complex percutaneous coronary intervention for multiple vessel coronary artery disease.

Diagnoses: Chest radiography showed pulmonary congestion and hyponatremia was noted after fluid hydration for suspicious contrast-induced nephropathy.

Interventions: Oral tolvaptan, at 15mg per day, was used for three days.

Outcomes: Urine output increased gradually and symptoms relieved one day later after using tolvaptan. Serum creatinine also improved to baseline level one week later after this event.

Lessons: Here, we reported an interesting case about contrast-induced acute kidney injury and hypervolemic hyponatremia, where tolvaptan was used to rescue the oliguric phase. Tolvaptan could be considered to use for contrast-induced acute kidney injury and had possibility of prevention from hemodialysis. Larger studies are still needed to investigate the role of tolvaptan in rescuing the oliguric phase in contrast-induced acute kidney injury.

Abbreviations: AKI = acute kidney injury, CKD = chronic kidney disease, DES = drug-eluting stent, LAD = left anterior descending artery, LCX = left circumflex artery, mEQ/L = milliequivalents per liter, METs = metabolic equivalents of tasks, mg = milligram, mg/dL = milligram per deciliter, mL = milliliter, OM = obtuse marginal, PCI = percutaneous coronary intervention, pg/mL = picograms per milliliter, RCA = right coronary artery.

Keywords: acute kidney injury, contrast-induced nephropathy, hypervolemic hyponatremia, oliguria, tolvaptan

1. Introduction

Tolvaptan is a prototype antidiuretic hormone receptor antagonist that acts at the level of renal tubules to increase free water excretion, without inducing major systemic electrolyte abnormalities.[1] In large, randomized studies, tolvaptan was used for different etiologies of induced hyponatremia including heart failure, liver cirrhosis, syndrome of inappropriate antidiuretic hormone secretion, and malignancy.[2] Few studies and case reports focus on acute kidney injury and contrast-induced nephropathy. We present case on the use of tolvaptan for contrast-induced acute kidney injury and hypervolemic hyponatremia.

2. Case report

This 58-year-old woman experienced intermittent palpitation, and chest discomfort with sensation of compression during exercise. A 24-hour Holter monitor did not detect any arrhythmia and conduction block. Treadmill exercise test was positive for ischemic change with exercise loading of 4.3 metabolic equivalents of tasks (METs). She had a medical history of hypertension, diabetes mellitus type 2, and stage 3 chronic kidney disease. Her serum creatinine was 1.62 mg/dL (milligram per decilitre) (normal range in female: 0.5–1.2 mg/dL),

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[a] Division of Cardiology, Department of Internal Medicine, [b] Division of Cardiology, Cardiac Care Unit, Kaohsiung Chang Gung Memorial Hospital, Chang Gung University College of Medicine, Kaohsiung, Taiwan, Republic of China.

[*] Correspondence: Wei-Chieh Lee, Division of Cardiology, Department of Internal Medicine, Kaohsiung Chang Gung Memorial Hospital, No. 123, Ta Pei Road, Niao-Sung District, Kaohsiung City, 83301, Taiwan, Republic of China (e-mail: leeweichieh@yahoo.com.tw).

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and glomerular filtration rate index was 34 mL/min (milliliter per minute) per 1.73 m^2, and serum sodium level was 142 mmol/L (millimole per liter) (normal range: 135–145 mmol/L). According to her risk factors as diabetes mellitus and impaired renal function, we well explained the risk of contrast-induced nephropathy and the risk of hemodialysis. The patient and her family understood and agree the procedure. Before procedure, normal saline solution hydration as 60 mL/h was used for 12 hours to prevent contrast-induced nephropathy.

Coronary angiography showed severe triple vessel coronary artery disease, including the proximal left anterior descending artery (LAD), which had 90% long tubular stenosis; the first obtuse marginal (OM) branch of left circumflex artery (LCX), which had 80% to 90% stenosis; and the mid right coronary artery (RCA), which had 70% to 80% stenosis with distal segment subtotal occlusion. The family and the patient refused coronary artery bypass graft surgical intervention because they wanted a quick recovery time. After discussions, we performed percutaneous coronary intervention (PCI) for total revascularization. Initially, the possibility of contrast nephropathy was well explained due to renal insufficiency and diabetes mellitus and multiple vessel approach. According to her body weight (50 kg) and serum creatinine (1.62 mg/dL), the maximal contrast volume was estimated 154 mL by Cigarroa formula.[3] One long drug-eluting stent (DES) was deployed near the ostial LAD after pre-dilatation. However, proximal LCX delay flow due to plague shifting after stenting occurred. Thus, another DES was deployed at the ostial LCX for plague shifting. Another short DES was deployed to the first OM branch, and another 2 DESs were deployed to the proximal and mid-RCA after adequate pre-dilatation. After stenting and adequate post-dilatation with different sizes of high-pressure balloons, thrombolysis in myocardial infarction flow 3 was achieved. A contrast volume of 280 mL (Omnipaque 350, GE Healthcare) was used due to multiple vessel approach, plaque shifting at ostial LCX, and subtotal lesion at RCA. Omnipaque 350 was a kind of low-osmolar nonionic contrast medium iohexol and was an only 1 contrast available in our hospital. Hydration with normal saline solution was maintained as 60 mL/h owing to the volume of contrast used.

Unfortunately, serum creatinine increased from 2.0 to 3.17 mg/dL, and urine output also gradually decreased to 220 mL 2 days later. Serum potassium was 4.6 mEq/L (milliequivalents per liter) (normal range: 3.5–4.9 mEq/L). The patient experienced transient bradycardia with symptomatic dizziness for several times, and transvenous temporary pacemaker was inserted. Acute kidney injury (AKI) with oliguria was noted. We favored contrast-induced nephropathy due to no other nephron-toxic drug was used and no urinary tract infection. We kept hydration with normal saline, and her body weight also increased 3 kg. High dose furosemide at 60 mg per day was administrated, but urine output remained <1000 mL/d. Serum creatinine increased to 3.69 mg/dL, and serum sodium decreased to 122 mEq/L. Orthopnea and impending respiratory failure was noted. Chest radiography showed pulmonary congestion. High brain-type natriuretic peptide 1169 pg/mL (picograms per milliliter) (normal range: <100 pg/mL) was noted. Oral tolvaptan, at 15 mg per day, was used because of hyponatremia and oliguria. One day later, urine output increased to 1000 to 1500 mL every 8 hours. Symptoms abated, and serum sodium gradually returned within normal range. In total, oral tolvaptan, at 15 mg per day, was used for 3 days. Finally, serum creatinine returned to 2.05 mg/dL 6 days later, and was 1.38 mg/dL 12 days later (post-PCI laboratory data are listed in Table 1). Her symptoms gradually improved, and she was discharged 20 days later. During 1-year follow-up period, her renal function revealed chronic kidney disease stage 2 to 3, and she presented with adequate urine output.

3. Discussion

Contrast-induced AKI is one of the serious adverse effects of contrast media and is the third most common cause after renal hypoperfusion and postoperative renal injury, especially in the patients with chronic kidney disease (CKD).[4,5] A history of contrast-induced AKI may be associated with the development of CKD and subsequent progression to end-stage renal disease in the long-term period.[6] Both short-term and long-term mortality rates have been found to be significantly higher in patients with contrast-induced AKI compared with patients without contrast-induced AKI.[7] The reported incidence of contrast-induced AKI after PCI varies between 10% and 37%, depending on the prevalence of associated risk factors including preexisting renal insufficiency, diuretics use, diabetes mellitus, and old age, with the higher incidence being reported after emergency PCI.[8–10] Currently, there is no specific treatment for contrast-induced AKI, and no evidence is available on helpful preventive strategies once the contrast-induced AKI develops.[11] Intravascular hydration seems to be the best preventive measure against contrast-induced AKI.[12] When contrast-induced AKI happened, treatment strategies, including stabilization of hemodynamic parameters and maintenance of normal fluid and electrolyte balance, are similar to the management for other types of AKI.[12]

Tolvaptan is an orally active, selective, non-peptide antagonist which blocks arginine vasopressin from binding to V2 receptors of the distal nephron, and induces the excretion of electrolyte-free water without changing the total level of electrolyte excre-

Table 1

Post-PCI laboratory data.

Laboratory values	Pre-PCI	Post-PCI D1	Post-PCI D2	Post-PCI D3	Post-PCI D4	Post-PCI D6	Post-PCI D8	Post-PCI D12	3 months later at OPD
Oral tolvaptan, at 15 mg per day									
BUN, mg/dL	37		51	50	33	35	35	38	36
Creatinine, mg/dL	1.59	2	3.17	3.69	3.42	2.05	1.66	1.38	1.84
Sodium, mmol/L	142		122	124	134	141	142	145	139
Potassium, mEq/L	4.3		4.6	3.9	3.6	4	3.6	3.1	4.2
Urine output, mL		540	220	960	4630	1840	1850	1450	1200
Body weight, kg	53		56	57	56	55	54	53	53

BUN = blood urea nitrogen, D = day, kg = kilogram, mEQ/L = milliequivalents per liter, mg/dL = milligram per deciliter, mg = milligram, mL = milliliter, mmol/L = millimole per liter, OPD = out-patient department, PCI = percutaneous coronary intervention.

tion.[13,14] Several studies stated that tolvaptan appears to decrease both body weight and edema and increases serum sodium concentrations without adversely affecting serum electrolyte levels, vital signs, or renal function in patients with heart failure.[15–17] Tolvaptan also reduces the risk of worsening renal function in patients with acute decompensated heart failure,[18] and reduces volume overloading in the patients with CKD.[19]

In our case, the patient experienced contrast-induced AKI after complex PCI for multiple vessel coronary artery approach, and hyponatremia after hydration. Chest radiography also showed pulmonary congestion. Her body weight also increased, and she was poorly responsive to high-dose diuretics. After the administration of tolvaptan, her renal function remained at baseline level during follow-up period. No previous case report focused on the use of tolvaptan for contrast-induced AKI. One case report mentioned that tolvaptan was used in the treatment of hypervolemic hyponatremia and oliguric acute tubular necrosis after surgery.[20] In our case, we shared our favorable experience with tolvaptan, highlighting its positive effect on contrast-induced AKI and associated hypervolemic hyponatremia.

4. Conclusion

We reported an interesting case about tolvaptan use for contrast-induced AKI and associated hypervolemic hyponatremia, which prevented the patient from hemodialysis. Larger studies are still needed to investigate the role of tolvaptan in rescuing the oliguric phase in contrast-induced AKI.

Author contributions

Conceptualization: Wei-Chieh Lee.
Data curation: Wei-Chieh Lee.
Formal analysis: Wei-Chieh Lee.
Investigation: Wei-Chieh Lee.
Methodology: Wei-Chieh Lee.
Project administration: Wei-Chieh Lee.
Software: Wei-Chieh Lee.
Supervision: Wei-Chieh Lee.
Validation: Wei-Chieh Lee.
Visualization: Wei-Chieh Lee.
Writing – original draft: Wei-Chieh Lee.
Writing – review and editing: Hsiu-Yu Fang, Chih-Yuan Fang.

References

[1] Thajudeen B, Salahudeen AK. Role of tolvaptan in the management of hyponatremia in patients with lung and other cancers: current data and future perspectives. Cancer Manag Res 2016;8:105–14.
[2] Laville M, Burst V, Peri A, et al. Hyponatremia secondary to the syndrome of inappropriate secretion of antidiuretic hormone (SIADH): therapeutic decision-making in real-life cases. Clin Kidney J 2013;6 (suppl):i1–20.
[3] Cigarroa RG, Lange RA, Williams RH, et al. Dosing of contrast material to prevent contrast nephropathy in patients with renal disease. Am J Med 1989;86(6 Pt 1):649–52.
[4] Mohammed NM, Mahfouz A, Achkar K, et al. Contrast-induced nephropathy. Heart Views 2013;14:106–16.
[5] Ozkok S, Ozkok A. Contrast-induced acute kidney injury: a review of practical points. World J Nephrol 2017;6:86–99.
[6] Nemoto N, Iwasaki M, Nakanishi M, et al. Impact of continuous deterioration of kidney function 6 to 8 months after percutaneous coronary intervention for acute coronary syndrome. Am J Cardiol 2014;113:1647–51.
[7] Rudnick M, Feldman H. Contrast-induced nephropathy: what are the true clinical consequences? Clin J Am Soc Nephrol 2008;3:263–72.
[8] Evola S, Lunetta M, Macaione F, et al. Risk factors for contrast induced nephropathy: a study among Italian patients. Indian Heart J 2012;64: 484–91.
[9] Bolognese L, Falsini G, Schwenke C, et al. Impact of iso-osmolar versus low-osmolar contrast agents on contrast-induced nephropathy and tissue reperfusion in unselected patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention (from the Contrast Media and Nephrotoxicity Following Primary Angioplasty for Acute Myocardial Infarction [CONTRAST-AMI] Trial). Am J Cardiol 2012;109:67–74.
[10] Chen SL, Zhang J, Yei F, et al. Clinical outcomes of contrast-induced nephropathy in patients undergoing percutaneous coronary intervention: a prospective, multicenter, randomized study to analyze the effect of hydration and acetylcysteine. Int J Cardiol 2008;126:407–13.
[11] Weisbord SD, Mor MK, Resnick AL, et al. Incidence and outcomes of contrast-induced AKI following computed tomography. Clin J Am Soc Nephrol 2008;3:1274–81.
[12] Mueller C, Buerkle G, Buettner HJ, et al. Prevention of contrast media-associated nephropathy: randomized comparison of 2 hydration regimens in 1620 patients undergoing coronary angioplasty. Arch Intern Med 2002;162:329–36.
[13] Decaux G, Soupart A, Vassart G. Non-peptide arginine-vasopressin antagonists: the vaptans. Lancet 2008;371:1624–32.
[14] Doggrell SA. Tolvaptan (Otsuka). Curr Opin Investig Drugs 2004;5: 977–83.
[15] Gheorghiade M, Niazi I, Ouyang J, et al. Vasopressin V2-receptor blockade with tolvaptan in patients with chronic heart failure: results from a double-blind, ran-domized trial. Circulation 2003;107:2690–6.
[16] Patra S, Kumar B, Harlalka KK, et al. Short term efficacy and safety of low dose Tolvaptan in patients with acute decompensated heart failure with hyponatremia: a prospective observational pilot study from a single center in South India. Heart Views 2014;15:1–5.
[17] Gheorghiade M, Gattis WA, O'Connor CM, et al. Effects of tolvaptan, a vasopressin antagonist, in patients hospitalized with worsening heart failure: a randomized controlled trial. JAMA 2004;291:1963–71.
[18] Matsue Y, Suzuki M, Seya M, et al. Tolvaptan reduces the risk of worsening renal function in patients with acute decompensated heart failure in high-risk population. J Cardiol 2013;61:169–74.
[19] Tanaka A, Katsuno T, Ozaki T, et al. The efficacy of tolvaptan as a diuretic for chronic kidney disease patients. Acta Cardiol 2015;70: 217–23.
[20] Gopinath S, Janga KC, Greenberg S, et al. Tolvaptan in the treatment of acute hyponatremia associated with acute kidney injury. Case Rep Nephrol 2013;2013:801575.