RESEARCH PAPER

A Comprehensive Review of the Renal Safety And Stability of Torsemide in Patients with Chronic Kidney Disease and Heart Failure

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ABSTRACT

Loop diuretics are commonly used in clinical practice for the treatment of fluid overload states associated with chronic kidney disease, congestive heart failure, and liver cirrhosis. These diuretics exert their action by inhibiting the reabsorption of sodium and chloride ions in the thick ascending limb of the loop of Henle, thus inducing diuresis and reducing extracellular fluid volume. Among all loop diuretics, torsemide and furosemide are commonly used in clinical practice. These two drugs, although belonging to the same pharmacological class, have shown significant differences in their pharmacokinetics, bioavailability, action, and clinical effects. Despite all the above-mentioned beneficial effects of torsemide, there is limited prospective clinical data on the effects of torsemide on various parameters of renal function, including GFR, serum creatinine, and BUN.

This review article aims to summarize the existing evidence of the efficacy of torsemide therapy and its effect on renal function in patients with CKD and heart failure. It also aims to present a prospective interventional study that will assess the effect of torsemide on renal function parameters, such as GFR, serum creatinine, and BUN. In addition, the study will also assess the renal safety of the drug, including acute kidney injury and electrolyte imbalance. In conclusion, this review article presents the potential role of torsemide as a safe and effective loop diuretic that may help maintain a stable renal function while effectively treating fluid overload in patients with cardiovascular and renal diseases.

Keywords: Torsemide, Loop diuretic, Chronic kidney disease, Heart failure, Glomerular filtration rate, Renal function, Diuretic therapy.

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Introduction

Fluid overload is a condition frequently encountered in patients with chronic kidney diseases, heart failure, and liver cirrhosis. Fluid overload causes complications such as respiratory edema, peripheral edema, hypertension, and heart failure in patients. Loop diuretics are considered the first-line drugs for the treatment of fluid overload because of their potent natriuretic and diuretic properties. [2,16]

Loop diuretics work by inhibiting the sodium-potassium-chloride cotransporter located in the thick ascending limb of the loop of Henle of the nephron. This mechanism of action prevents sodium and chloride reabsorption, resulting in increased sodium and chloride excretion in the urine. This makes these drugs effective in reducing fluid volume within the body and relieving symptoms of fluid retention. [1,37]

Among all the loop diuretics, furosemide has been used most commonly as a drug of choice. However, torsemide has received considerable attention in recent times because of its promising pharmacokinetic profile and potential clinical benefits. It has been observed that torsemide has a high bioavailability of 80–100%, whereas furosemide has variable bioavailability, ranging from 10% to 90%. This variable bioavailability of furosemide may lead to unpredictable clinical responses in patients. [14, 15]. Besides, torsemide has been observed to have a longer half-life and prolonged duration of action compared with furosemide, which may be advantageous for controlling fluid overload states. In addition, torsemide may have additional clinical benefits by modulating neurohormonal systems, including the renin- angiotensin-aldosterone system (RAAS). It has been observed that torsemide may inhibit myocardial fibrosis mediated by aldosterone, leading to improved clinical outcomes. [7,14]

The maintenance of renal function is of particular interest in patients who have received diuretic treatment, especially in patients who have CKD. Excessive diuresis and changes in the

hemodynamic of the kidneys have the potential to cause impairment of the kidneys or AKI.
However, the assessment of the effect of diuretic treatment on various renal functions such as GFR, creatinine, and blood urea nitrogen is crucial in the management of the patients. [12,36]
Although interest in torsemide has been rising, little prospective clinical data are available on its effects on renal function parameters, with most studies focusing on cardiovascular outcomes, including mortality and hospitalization rates. Therefore, further studies are needed to determine whether torsemide could preserve renal function and adequately manage fluid overload in patients with CKD and heart failure. [4,8]

Pharm

acological Profile of Torsemide Mechanism ofAction

Torsemide belongs to the class of diuretics known as loop diuretics. It works by inhibiting the $Na^+/K^+/2Cl^-$ co-transporter in the thick ascending limb of the loop of Henle. Inhibiting this transporter reduces the reabsorption of sodium and chloride, resulting in increased loss of these ions, along with potassium, calcium, and magnesium, in the urine, thus decreasing the volume of extracellular fluid. [1,37]

Pharmacokinetics

Torsemide demonstrates several pharmacokinetic advantages over other loop diuretics:

- **High oral bioavailability (80–100%)**
- **Longer half-life (approximately 3–6 hours)**
- **Longer duration**
- **of action (6–8 hours)**
- **Predictable dose–response relationship**
- **Pharmacodynamic Effects**

Torsemide has a potent effect of increasing sodium and water excretion in urine. It also has the effect of decreasing venous pressure and symptoms of congestion in patients with heart failure. Other research has indicated that torsemide has the potential effect of inhibiting aldosterone- induced fibrosis, which could be beneficial for patients with cardiovascular and kidney diseases. [2,7]

Literature Review

Study by Mohammad Al Mamun et al., 2025

The real-world observational study by Mohammad Al Mamun et al. examined the clinical outcomes of heart failure patients who are taking loop diuretics such as torsemide, furosemide, and bumetanide. The study has a large number of patients and examined mortality rates, hospitalization rates, and renal function parameters. [4,9]
The results showed that patients treated with torsemide had slightly better clinical outcomes compared to those treated with other loop diuretics. The mortality rate was lower among patients treated with torsemide, and no worsening of renal function was observed. This implies that torsemide could be effective in fluid management without impairing renal function.. [4,8]

TRANSFORM-HF Trial, 2023

The TRANSFORM-HF trial is a randomized clinical trial aimed at evaluating the effectiveness of torsemide and furosemide in patients who were hospitalized due to heart failure. The main endpoint of the trial was all-cause mortality. [1,8]

Discussion

The management of fluid overload in patients with CKD and heart failure demands the selection of diuretic therapy that balances fluid removal with renal function preservation. Torsemide has a number of pharmacological advantages that may contribute to better clinical outcomes compared with traditional loop diuretics. [14,15]Torsemide has a higher bioavailability and longer duration of action, resulting in more consistent diuretic responses that may reduce the need for dose adjustments. Furthermore, the potential for torsemide to exert anti-fibrotic effects may also contribute to better outcomes in patients with heart failure. [7]

Impact on Glomerular Filtration Rate

One of the most important parameters for assessing kidney function is the glomerular filtration rate (GFR). Diuretic therapy affects the glomerular filtration rate because it affects renal blood flow and intraglomerular pressure. Excessive diuresis caused by diuretic therapy results in hypovolemia, which affects the glomerular filtration rate. [22]
Torsemide, when used in the right dosage, is effective in maintaining stable GFR in patients with heart failure or mild to moderate renal impairment. Torsemide is effective in increasing cardiac output and relieving venous congestion; these effects improve perfusion of the kidneys

and GFR. [21] In practice, serum creatinine levels may rise transiently in patients receiving diuretic therapy due to alterations in intravascular volume; however, these effects may not always be permanent and may be reversible. Monitoring of these parameters ensures that the benefits of diuretic therapy outweigh the risks to the kidneys. [31]Torsemide in Heart Failure Management Role in Managing Fluid Overload

Fluid retention is one of the major characteristics of heart failure and is responsible for the occurrence of the symptoms. Fluid retention in the lungs leads to pulmonary congestion and breathlessness, while retention of excess fluids in the peripheral tissues leads to peripheral edema. Torsemide facilitates the process of diuresis and natriuresis, helping to get rid of excess fluids from the body. [2]

By reducing the volume of circulating blood, torsemide reduces the workload of the failing heart. This results in a relief of symptoms such as shortness of breath and fatigue. In most cases, diuretics such as torsemide are used for the relief of symptoms in patients with heart failure who show signs of fluid retention. [16]

Use in Patients with Coexisting Kidney Disease

patients with heart failure also have underlying compromised renal function. This situation is termed cardiorenal syndrome. The management of fluid balance in patients with cardiorenal syndrome may be problematic. Excessive diuretic use may compromise renal function further, and inadequate diuretic use may exacerbate heart failure symptoms. [21]

In this method, torsemide may be beneficial due to its predictable absorption and long duration of action. This enables the practitioner to achieve diuretic effects while limiting fluctuations in drug levels. Torsemide may be useful in maintaining a stable fluid balance in patients with both heart failure and renal impairment. [14]

Combination Therapy in Heart Failure

Torsemide is not often employed as a single agent for heart failure treatment. It is normally employed as part of a multi-faceted approach that also involves other agents like angiotensin- converting enzyme inhibitors, beta blockers, mineralocorticoid receptor antagonists, and sodium-glucose cotransporter-2 inhibitors. Although the first three agents are thought toinfluence the pathophysiology of heart failure, torsemide has a more symptomatic effect by managing fluid overload. [34, 35]

Monitoring Parameters in Patients Receiving Torsemide

The monitoring parameters include the assessment of renal function, electrolytes, fluid balance, cardiovascular parameters, metabolic changes, and possible interactions of the drug. These monitoring parameters play a crucial role in the optimization of treatment outcomes.

1. Renal Function Monitoring

Monitoring renal function is one of the most important parameters in the management of torsemide. As the diuretic action of torsemide involves the renal system, there is a potential effect on the renal system, especially in those with underlying renal impairment. [12] Parameters that need to be checked for the assessment of renal function include creatinine, blood urea nitrogen, and the estimated glomerular filtration rate. The assessment of these parameters is essential in determining whether the kidneys are filtering the metabolic waste products appropriately. In the case of chronic kidney diseases, the renal function should be checked before the administration of the drug. [36]

Rise in serum creatinine and BUN levels may be a sign of deteriorating renal function or over- diuresis resulting in dehydration. eGFR levels need to be monitored to assess the progression of kidney disease and titrate the dosage of Torsemide accordingly. In patients with advanced stages of CKD, close monitoring is required as the drug response is altered due to decreased clearance of drugs by the kidneys. [46]

2. Electrolyte Monitoring

Electrolyte disturbances are among the most common complications associated with loop diuretic therapy. Torsemide promotes the excretion of sodium, potassium, chloride, calcium, and magnesium through urine. Therefore, regular monitoring of electrolyte levels is essential.[1]Serum potassium levels are particularly important because hypokalemia is a frequent adverse effect of loop diuretics. Low potassium levels can lead to muscle weakness, cardiac arrhythmias, and fatigue. [11] Magnesium and calcium levels should also be assessed because long-term diuretic therapy may lead to deficiencies that contribute to neuromuscular and cardiovascular complications. [37]Fluid Balance and Hydration Status

Monitoring fluid balance is essential to evaluate the therapeutic effectiveness of torsemide and to prevent complications associated with excessive diuresis. Accurate measurement of fluid intake and urine output helps clinicians determine whether the diuretic effect is appropriate.

[1]Assessment of clinical signs such as edema, pulmonary congestion, and jugular venous distention also helps determine the effectiveness of torsemide therapy[2]. Signs of dehydration, including dry mucous membranes, decreased skin turgor, and hypotension, should be evaluated to avoid complications from excessive fluid loss.

3. Blood Pressure Monitoring

The drug can have a significant impact on blood pressure as it is a diuretic and natriuretic. This is in the sense that the drug can reduce the volume of blood in the body. This can lead to a reduction in the pressure of the blood. Blood pressure needs to be monitored regularly, especially in patients with hypertension. [1, 41]

Hypotension is a possible side effect of over-diuresis and may cause symptoms such as dizziness, fainting, or weakness. Monitoring both systolic and diastolic blood pressure will ensure that the drug is effective while at the same time preventing a fall in blood pressure. [1] Orthostatic measurements of blood pressure may also be needed in the elderly or patients with autonomic dysfunction. A fall in blood pressure when a patient stands up may show volume depletion and require adjustment of the dosage of torsemide. [28]

4. Cardiac Monitoring

Cardiac monitoring plays a crucial role in patients with heart failure who are undergoing treatment with torsemide. Alterations in the levels of certain electrolytes, e.g., low levels of potassium (hypokalemia) and magnesium (hypomagnesemia), may predispose a patient to the occurrence of cardiac arrhythmias. In this case, electrocardiography may be used to detect certain abnormalities, e.g., QT interval prolongation, ventricular arrhythmias, and conduction abnormalities. Patients with existing cardiovascular diseases and those on cardiac glycosides should be monitored closely to prevent any complications that may arise from arrhythmias. The heart rate and rhythm are constantly monitored to detect any imbalances or drug interactions that may affect the heart. [27]

5. Metabolic Parameter Monitoring

The metabolic parameters are also altered during torsemide treatment. Therefore, it is imperative to monitor them. It is known that loop diuretics can cause alterations in glucose metabolism, uric acid, and lipid levels. [11] Uric acid level in the blood is also to be regularly monitored during torsemide treatment. This is because torsemide can cause hyperuricemia by reducing uric acid excretion in the urine. Gout attacks may also be precipitated. Therefore, in patients with a history of gout, it is imperative to monitor uric acid levels and take appropriate measures to prevent gout attacks. [28] Glucose level in the blood is also to be monitored during torsemide treatment. This is particularly applicable in diabetic patients. In diabetic patients, it is known that loop diuretics can cause hyperglycemia. In addition, it can also cause impaired glucose tolerance. Therefore, it is imperative to monitor blood glucose levels in diabetic patients. In addition, it is known that loop diuretics can cause alterations in lipid levels. However, these alterations are less significant compared to other antihypertensives. [1]

Conclusion

Torsemide is a well-established loop diuretic that is effective in the treatment of fluid overload conditions in patients with chronic kidney disease and heart failure. Its favorable pharmacokinetic profile, which includes high bioavailability and longer duration of action, enables the drug to provide sustained diuretic activity. Evidence of the effectiveness of torsemide in the maintenance of stable renal function has been established, and its potential benefits are demonstrated in comparison to other loop diuretics. However, the available body of research is limited, and more prospective studies are required to fully elucidate its potential benefits in the maintenance of renal function. The proposed study aims to provide more evidence regarding the impact of torsemide on the most important renal function parameters, namely glomerular filtration rate, serum creatinine, and blood urea nitrogen. This would be helpful in making appropriate clinical decisions regarding the choice of diuretics in the treatment of CKD and heart failure, which would improve patient outcomes.

Reference

1. Brater DC. Diuretic therapy. N Engl J Med. 1998 Aug 6;339(6):387–395.
2. Ellison DH, Felker GM. Diuretic treatment in heart failure. N Engl J Med. 2017 Nov 16;377(20):1964–1975.
3. Felker GM, Lee KL, Bull DA, Redfield MM, Stevenson LW, Goldsmith SR, et al. Diuretic strategies in patients with acute decompensated heart failure. N Engl J Med. 2011 Mar 3;364(9):797–805.
4. Mentz RJ, Stevens SR, DeVore AD, Lala A, Vader JM, AbouEzzeddine OF, et al. Torsemide versus furosemide in patients with heart failure: insights from the ASCEND- HF trial. J Am Coll Cardiol. 2015 Jan 27;65(3):235–242.
5. Murray MD, Deer MM, Ferguson JA, Dexter PR, Bennett SJ, Perkins SM, et al. Open- label randomized trial of torsemide compared with furosemide therapy for patients with heart failure. Am J Med. 2001 Nov;111(7):513–520.
6. Dormans TPJ, Gerlag PGG. Combination of high-dose furosemide and torasemide in refractory heart failure. Eur Heart J. 1996 Dec;17(12):1867–1874.
7. Cosín J, Díez J. Torasemide in chronic heart failure: results of the TORIC study.

Eur J Heart Fail. 2002 Aug;4(4):507–513.

8. Felker GM, Anstrom KJ, Adams KF, Ezekowitz JA, Fiuzat M, Houston-Miller N, et al. Effect of torsemide vs furosemide after discharge on all-cause mortality in patients hospitalized with heart failure: the TRANSFORM-HF randomized clinical trial. JAMA. 2023 Jan 17;329(3):214–223.
9. Peacock WF, Costanzo MR, De Marco T, Lopatin M, Wynne J, Mills RM, et al. Impact of intravenous loop diuretics on outcomes of patients hospitalized with acute decompensated heart failure. Am J Cardiol. 2009 Apr 15;103(8):1219–1224.
10. Ellison DH. Diuretic resistance: physiology and therapeutics. Semin Nephrol. 1999 Nov;19(6):581–597.
11. Wilcox CS. Metabolic and adverse effects of diuretics. Semin Nephrol. 1999 Nov;19(6):557–568.
12. Sica DA. Diuretic use in renal disease. Nat Rev Nephrol. 2012 Feb;8(2):100–109.
13. Fliser D, Schroter M, Neubeck M, Ritz E. Coadministration of thiazides increases the efficacy of loop diuretics even in patients with advanced renal failure. Kidney Int. 1994 Aug;46(2):482–488.
14. Knauf H, Mutschler E. Clinical pharmacokinetics and pharmacodynamics of torasemide. Clin Pharmacokinet. 1998 Jan;34(1):1–24.
15. Vargo DL, Kramer WG, Black PK, Smith WB, Serpas T, Brater DC. Bioavailability, pharmacokinetics, and pharmacodynamics of torsemide and furosemide in patients with congestive heart failure. J Clin Pharmacol. 1995 Mar;35(5):458–463.
16. Faris R, Flather MD, Purcell H, Poole-Wilson PA, Coats AJ. Diuretics for heart failure. Cochrane Database Syst Rev. 2012 Feb 15;2:CD003838.
17. Gupta S, Neyses L. Diuretic resistance in chronic congestive heart failure. Cardiovasc Drugs Ther. 2005 Aug;19(4):297–302.
18. O'Connor CM, Hasselblad V, Mehta RH, Tasissa G, Califf RM, Fiuzat M, et al. Triage after hospitalization with advanced heart failure. J Am Coll Cardiol. 2011 Nov 15;58(21):2141–2148.
19. Ahmed A, Husain A, Love TE, Gambassi G, Dell'Italia LJ, Francis GS, et al. Heart failure, chronic diuretic use, and increase in mortality and hospitalization. Am Heart J. 2006 Jan;152(1):67–73.
20. Testani JM, Chen J, McCauley BD, Kimmel SE, Shannon RP. Potential effects of aggressive decongestion during treatment of decompensated heart failure on renal function and survival. Circulation. 2010 Jul 20;122(3):265–272.
21. Ronco C, McCullough P, Anker SD, Anand I, Aspromonte N, Bagshaw SM, et al. Cardiorenal syndrome. J Am Coll Cardiol. 2010 Aug 24;56(9):742–748.
22. Damman K, Valente MAE, Voors AA, O'Connor CM, van Veldhuisen DJ, Hillege HL. Diuretic therapy and renal function in heart failure. Eur Heart J. 2014 Feb;35(7):455– 469.
23. Costanzo MR, Ronco C. Cardiorenal syndrome in heart failure. J Am Coll Cardiol. 2012 Sep 18;60(12):1031–1040.
24. Verbrugge FH, Dupont M, Steels P, Grieten L, Malbrain M, Tang WHW, et al. The kidney in congestive heart failure. Eur Heart J. 2013 Jul;34(28):2191–2200.Neuberg GW, Miller AB, O'Connor CM, Belkin RN, Carson PE, Cropp AB, et al. Diuretic resistance predicts mortality in patients with advanced heart failure. Am Heart
J. 2002 Jan;144(1):31–38.
25. Francis GS, Siegel RM, Goldsmith SR, Olivari MT, Levine TB, Cohn JN. Acute vasoconstrictor response to intravenous furosemide in patients with chronic congestive heart failure. Ann Intern Med. 1985 Jan;103(1):1–6.
26. Felker GM, O'Connor CM. Diuretics and outcomes in heart failure. Circulation. 2012 Jan 31;125(4):558–560.
27. Ahmed A. Use of diuretics in elderly patients with heart failure. Drugs Aging. 2002 Oct;19(10):705–718.
28. Sica DA, Gehr TW. Diuretic therapy in heart failure and renal disease. Heart Fail Rev. 2003 Jul;8(3):231–239.
29. Brater DC, Day B, Burdette A, Anderson S. Bumetanide and torsemide pharmacology and clinical use. Clin Pharmacol Ther. 1984 Aug;36(2):183–189.
30. Shah RV, McNulty S, O'Connor CM, Felker GM, Braunwald E, Givertz MM. Renal function changes during aggressive diuresis in heart failure. Am Heart J. 2011 Aug;162(2):325–331.
31. Forman DE, Butler J, Wang Y, Abraham WT, O'Connor CM, Gottlieb SS, et al. Incidence, predictors, and outcomes of acute renal failure among hospitalized patients with heart failure. J Am Coll Cardiol. 2004 Jan 7;43(1):61–67.
32. Adams KF, Fonarow GC, Emerman CL,

LeJemtel TH, Costanzo MR, Abraham WT, et al. Characteristics and outcomes of patients hospitalized for heart failure in the United States. Am Heart J. 2005 Feb;149(2):209–216.

33. Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Drazner MH, et al. ACC/AHA guideline for the management of heart failure. Circulation. 2013 Oct 15;128(16):e240– e327.
34. McMurray JJ, Adamopoulos S, Anker SD, Auricchio A, Böhm M, Dickstein K, et al. ESC guidelines for diagnosis and treatment of acute and chronic heart failure. Eur Heart J. 2012 Jul;33(14):1787–1847.
35. KDIGO Clinical Practice Guideline for the evaluation and management of chronic kidney disease. Kidney Int Suppl. 2013 Jan;3(1):1–150.
36. Rang HP, Ritter JM, Flower RJ, Henderson G. Rang and Dale's Pharmacology. 9th ed. London: Elsevier; 2020.
37. Brunton LL, Hilal-Dandan R, Knollmann BC. Goodman & Gilman's The Pharmacological Basis of Therapeutics. 13th ed. New York: McGraw Hill; 2018.
38. Katzung BG, Trevor AJ. Basic and Clinical Pharmacology. 15th ed. New York: McGraw Hill; 2021.
39. DiPiro JT, Yee GC, Posey LM, Haines ST, Nolin TD, Ellingrod VL. Pharmacotherapy: A Pathophysiologic Approach. 11th ed. New York: McGraw Hill; 2020.
40. Sica DA. Loop diuretic therapy in hypertension. J Clin Hypertens. 2003 Jan;5(1):64– 71.
41. Felker GM, Mentz RJ. Diuretics and ultrafiltration in acute heart failure. J Am Coll Cardiol. 2012 Jun 12;59(24):2145–2153.
42. Ellison DH. Clinical pharmacology in diuretic use. Clin J Am Soc Nephrol. 2019 Aug 7;14(8):1248–1257.
43. Murray MD, Brater DC. Renal toxicity of diuretics. Clin Pharmacokinet. 1993 Jul;25(1):35–44.
44. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, et al. Acute kidney injury network report. Crit Care. 2007;11(2):R31.
45. Kellum JA, Lameire N. Diagnosis and management of acute kidney injury. Kidney Int Suppl. 2012 Mar;2(1):1–138.
46. Ronco C, Haapio M, House AA, Anavekar N, Bellomo R. Cardiorenal syndrome. J Am Coll Cardiol. 2008 Nov 4;52(19):1527–1539.
47. Testani JM, Brisco MA, Turner JM, Spatz ES, Bellumkonda L, Parikh CR, et al. Loop diuretic efficiency and outcomes in heart failure. J Am Coll Cardiol. 2014 Mar 4;63(9):999–1007.
48. McMurray JJV, Pfeffer MA. Heart failure pathophysiology and treatment. Lancet. 2005 May 14;365(9474):1877–1889.
49. House AA, Wanner C, Sarnak MJ, Piña IL, McIntyre CW, Komenda P, et al. Heart failure in chronic kidney disease. Kidney Int Suppl. 2017 Mar;7(2):132–143.