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Furosmide and Bumetanide Pathophysiologic Basis Role in the Treatment of Liver Cirrhosis

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Abstract: *Liver cirrhosis is a chronic progressive disease characterized by hepatic fibrosis, architectural distortion of the liver, and the development of portal hypertension. One of the most common complications of cirrhosis is fluid retention, manifested as ascites and peripheral edema, resulting from splanchnic vasodilation, activation of the renin–angiotensin–aldosterone system (RAAS), sympathetic nervous system stimulation, and impaired renal sodium and water excretion. Diuretic therapy remains a cornerstone in the management of these complications. Loop diuretics such as furosemide and bumetanide exert their effects by inhibiting the sodium–potassium–chloride (Na^+–K^+–$2Cl^-$) cotransporter in the thick ascending limb of the loop of Henle, thereby promoting natriuresis and diuresis. Their mechanism directly counteracts the sodium retention associated with cirrhosis and helps reduce extracellular fluid accumulation.*

Furosemide is widely used in combination with aldosterone antagonists, particularly spironolactone, to achieve effective fluid control while minimizing electrolyte disturbances. Bumetanide, a more potent loop diuretic with higher oral bioavailability and predictable absorption, serves as an alternative in patients who demonstrate inadequate response or intolerance to furosemide. Both agents contribute to symptom relief, reduction of ascites, and improvement in quality of life. However, their use requires careful monitoring because excessive diuresis may lead to hypovolemia, renal dysfunction, hyponatremia, hypokalemia, and hepatic encephalopathy. Understanding the pathophysiological mechanisms underlying fluid retention in cirrhosis provides the rationale for the therapeutic use of loop diuretics. This review highlights the pharmacological properties, pathophysiologic basis, clinical applications, benefits, and limitations of furosemide and bumetanide in the management of liver cirrhosis and its associated complications..

Keywords: *Liver cirrhosis*

I. INTRODUCTION

Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury, that leads to portal hypertension and end stage liver disease. Recent advances in the understanding of the natural history and pathophysiology of cirrhosis, and in treatment of its complications, resulting in improved management, quality of life and life expectancy of cirrhotic patients.1

Patients with cirrhosis without any symptoms are termed to have compensated cirrhosis. Complications such as ascites, varietal bleeding, hepatic encephalopathy, or non-

obstructive jaundice, which can develop with cirrhosis of any origin, herald the onset of decompensated cirrhosis. In the presence of cirrhosis, superimposed hepatic injury (due

to viral, drug-induced, or alcohol-associated hepatitis) or other complications, particularly bacterial infections, can lead to hepatic and extra hepatic organ failure—a condition known as acute-on-chronic liver failure—that is associated with high short-term mortality.2

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Figure 1: liver cirrhosis

Cirrhosis is the final stage attained by various chronic liver diseases after years or decades of slow progression. There are, however, ways to prevent cirrhosis, because the diseases that most commonly lead to it progress slowly, and measures are available to prevent and treat them.3

Classification:

1. Morphological Classification (Based on liver structure) Micronodular cirrhosis

1. Nodules < 3 mm
2. Uniform appearance
3. Common in alcoholism

Macronodular cirrhosis

1. Nodules > 3 mm
2. Irregular size
3. Often seen in viral hepatitis

Mixed cirrhosis

1. Features of both micro- and macronodular
2. Etiological Classification (Based on cause)

***.Alcoholic cirrhosis**

1. Post-hepatitic cirrhosis (e.g., due to Hepatitis B, Hepatitis C)
2. Biliary cirrhosis
3. Primary (autoimmune)Secondary (bile duct obstruction)
4. Metabolic causes
5. Hemochromatosis
6. Wilson's disease

Non-alcoholic steatohepatitis (NASH)

Cardiac cirrhosis (due to chronic right heart failure) Cryptogenic cirrhosis (unknown cause)

3. Functional Classification (Severity-based)

A. Child–Pugh Classification

Used to assess severity and prognosis: Class A → Mild

Class B →Moderate Class C → Severe

Parameters include:

1. Bilirubin
2. Albumin
3. Prothrombin time (INR)
4. Ascites
5. Hepatic encephalopathy

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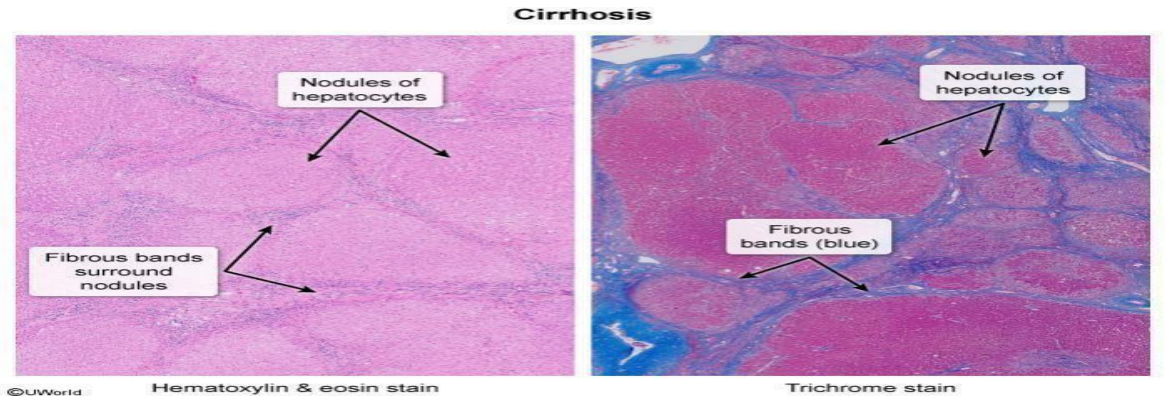

Figure; 2 marphological classification of liver

The role of zinc in liver cirrhosis

Zinc plays a key role in numerous biochemical and Physiological processes. It is an essential component of more than 300 different enzymes and owes its catalytic effect to its direct involvement in substrate conversion and the stabilization of enzyme structure. 4

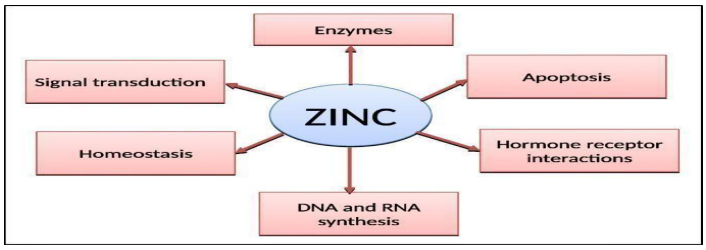

Figure 3: zinc functionin humanbody

Types of liver cirrhosis

1. Types Based on Cause (Etiology)

1. Alcoholic cirrhosis
2. Caused by chronic alcohol abuse
3. One of the most common types
4. Often preceded by fatty liver and alcoholic hepatitis

2. Viral cirrhosis

Due to chronic infection with hepatitis viruses Most commonly:

1. Hepatitis B
2. Hepatitis C

3. Biliary cirrhosis

Caused by bile duct damage or obstruction Includes:

a. Primary biliary cholangitis

b. Secondary biliary cirrhosis (due to obstruction like gallstones or tumors)

4. Cardiac cirrhosis

1. Caused by long-term right-sided heart failure
2. Leads to chronic liver congestion ("nutmeg liver")

Stages of liver cirrhosis:

Liver cirrhosis is a progressive disease in which healthy liver tissue is replaced by scar tissue, impairing liver function. The stages are commonly described in two main ways: clinical progression (compensated → decompensated) and scoring systems like Child-Pugh classification.

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Clinical Stages of Cirrhosis Stage

1: Compensated Cirrhosis

Liver is significantly scarred but still functions adequately often no symptoms or very mild ones may be detected incidentally on tests

Possible findings: fatigue, mild enlargement of liver/spleen

Many people remain in this stage for years with proper management.

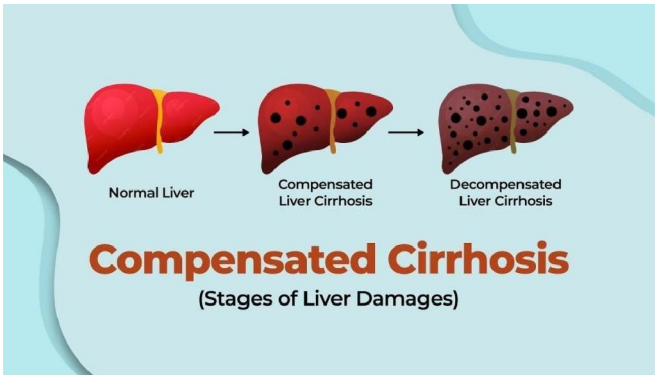

Figure: 4 compensated cirrhosis

Stage 2: Compensated Cirrhosis with Portal Hypertension

1. Increased pressure in the portal vein (portal hypertension)
2. Development of varices (swollen veins, especially in esophagus/stomach)
3. Still "compensated" (no major failure symptoms yet)
4. Risk of internal bleeding begins

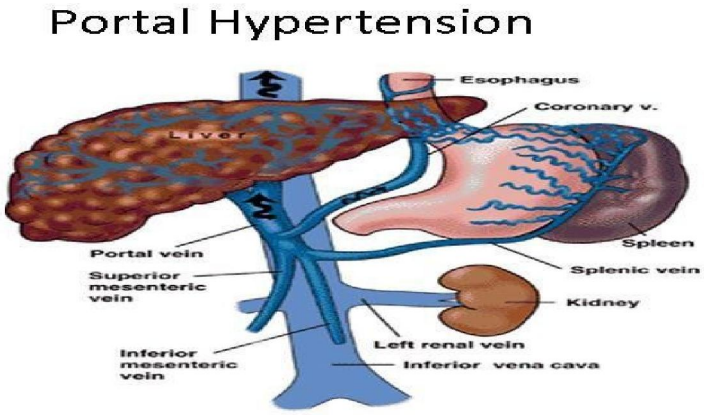

Figure :5 Portal Hypertension Stage

3: Decompensated Cirrhosis

Liver can no longer perform its functions effectively Major complications appear:

1. Ascites (fluid in abdomen)
2. Jaundice (yellowing of skin/eyes)
3. Easybruising/bleeding
4. Frequent hospitalizations may begin Figure : 6 decomplication cirrhosis

Stage 4: Advanced Decompensated Cirrhosis(End-Stage) Severe, life-threatening complications:

1. Hepatic encephalopathy (confusion, coma)

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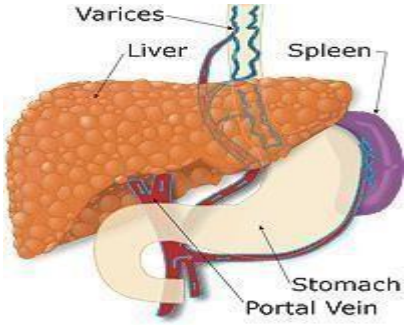
2. Severe bleeding from varices
3. Kidney dysfunction (hepatorenal syndrome)
4. Often requires liver transplant



2. Child-Pugh Classification (Severity Scoring)
1. Doctors also classify cirrhosis using the Child-Pugh score, which predicts prognosis:
2. Class A (mild) – well-compensated
3. Class B (moderate) – significant functional compromise
4. Class C (severe) – advanced disease, poor prognosis5

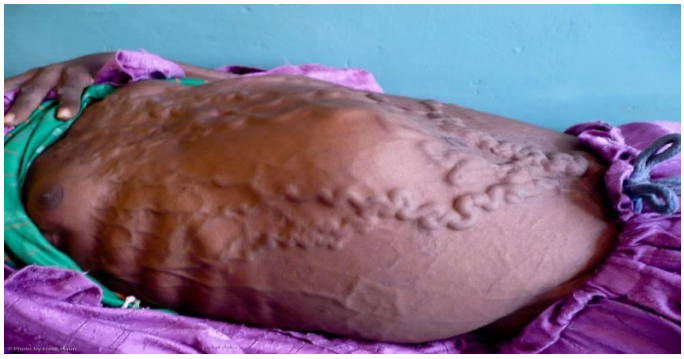


Figure 7: advanced decomplication cirrhosis

Malnutrition in Patients with Liver Cirrhosis

Cirrhosis is a.systemic disease and malnutrition is a key feature as well as an important complication of the disease. This implicates that malnutrition diagnosis is not only relevant as one of the cclinica Characteristics of cirrhosis, but also needs to be considered as an important complication, that. Warrants timely and appropriate therapy to improve prognosis.

Pathogenesis

The etiology of malnutrition in liver cirrhosis is multifactorial Decreased energy and protein intake, inflammation, malabsorption, altered nutrient metabolism, hormonal disturbances, hypermetabolism and gut microbiome dysbiosis can contribute to Malnutrition.

Additionally, fasting periods and external factors such as alcohol consumption have impact on malnutrition 6

-Chemical Name: 4-Chloro-N-furfuryl-5-sulfamoylanthranilic acid.

The structure of Furosemide can be visualized as a benzene ring with a carboxylic acid group, with a choro group and a sulfonamide group para to each other, and a furfurylamino group ortho to the carboxylic acid. This specific arrangement allows it to act as a loop diuretic.

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Indications

The Food and Drug Administration (FDA) has approved furosemide to treat conditions with volume overload and edema secondary to congestive heart failure exacerbation, liver failure, or renal failure, including the nephrotic syndrome.

For patients with acutely decompensated heart failure (ADHF) with volume overload who have not received diuretics previously, the Initial dose of furosemide should be 20 to 40 mg intravenously. Later, titrate the furosemide dose according to the clinical response of the patients. However, for those patients with ADHF with a normal kidney function on chronic diuretic therapy, the initial dose of furosemide can be equivalent to or greater than the total oral maintenance dose of furosemide the patient takes daily. Subsequently, the diuretic dose adjustments are according to the patient's clinical response. Nevertheless, starting with higher doses of furosemide, at a dose of 2.5 times the total daily oral dose of furosemide per day, has shown a significant trend toward a rapid improvement in the global assessment of patient symptoms.10Although the FDA approved the use of loop diuretics alone or in combination with other anti-hypertensive medications as an alternative to thiazide diuretics to treat hypertension, however, the clinical guidelines panel report of the Eighth Joint National Committee (JNC-8) published in 2014 and the American College of Cardiology/American Heart Association (ACC/AHA) Task Force Panel Guidelines on hypertension treatment published in 2017 do not recommend the use of loop diuretic as a first-line medication to treat hypertension.11-12 Nevertheless, Furosemide can be a second-line agent in heart failure patients with symptoms and advanced kidney disease with an estimated glomerular filtration rate of less than 30 ml per minute; loop diuretics (furosemide) are preferred over thiazide diuretics to treat hypertension.13 Diuretic therapy is recommended in patients with liver cirrhosis and ascites, accompanied by dietary sodium restriction.

Furosemide is indicated for the treatment of edema associated with congestive heart failure, cirrhosis of the liver, and renal disease, including the nephrotic syndrome, in adults and pediatric patients.

Oral furosemide is indicated alone for the management of mild to moderate hypertension or severe hypertension in combination with other antihypertensive medications.

Intravenous furosemide is indicated as adjunctive therapy in acute pulmonary edema when a rapid onset of diuresis is desired.

Subcutaneous furosemide is indicated for the treatment of congestion due to fluid overload in adults with NYHA Class II/III chronic heart failure. This drug formulation is not indicated for emergency situations or in patients with acute pulmonary edema.

Subcutaneous furosemide is also indicated for the treatment of edema in pediatric patients weighing 43 kg and above.

Mechanism of Action

Furosemide inhibits tubular reabsorption of sodium and chloride in the proximal and distal tubules and the thick ascending loop of Henle by inhibiting the sodium-chloride cotransport system resulting in excessive excretion of water along with sodium, chloride, magnesium, and calcium.8

Pharmacokinetics

Absorption: The onset of action of furosemide is usually within the first hour of oral furosemide intake, and it takes the first 1 to 2 hours to achieve a peak effect. The mean bioavailability of oral furosemide is 51% compared with the bioavailability of intravenously administered furosemide. Bioavailability: The furosemide absorption is slower than normal in patients with edema, particularly in patients with decompensated heart failure; however, the amount of loop diuretic absorbed is normal.14 Oral and sublingual administration of furosemide achieves a peak concentration slower than the iv route. Although furosemide is more avidly absorbed with a bioavailability of 59% via the sublingual route compared with the oral route of administration, i.e., 47%, the half-life and time to peak concentration were not different between the oral and sublingual route of drug delivery. Also, the urinary excretion rate of furosemide and sodium and cumulative urine excretion rate was not different between the oral and sublingual administration of

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furosemide.[10].The bioavailability of furosemide is variable and relatively lesser than that of torsemide in patients with compensated congestive heart failure.15

Distribution: In healthy individuals, greater than 95% of furosemide is bound to plasma protein, mainly albumin. Only 2.3% to 4.1% of furosemide is existent in an unbound form in therapeutic concentrations.

Metabolism: Furosemide glucuronide is a major biotransformation active product of furosemide, having an active diuretic effect. Contemporary evidence indicates that furosemide is minimally metabolized in the liver.16

Excretion: The terminal half-life of furosemide is approximately 2 hours, and the total time of therapeutic effect is 6 to 8 hours. However, the half-life of furosemide will prolong in patients with chronic renal disease.8 Although more furosemide gets excreted in the urine after IV administration, there is no difference in the amount of unchanged furosemide excretion in urine between the two formulations. After intravenous administration, furosemide achieves an early and high serum peak concentration and a higher peak excretion rate. A greater extent of furosemide is excreted in urine following the parenteral administration than oral administration.

* Bumetanide -Bumetanide1 is a potent 'loop' diuretic for the treatment of oedema associated with congestive heart failure, hepatic and renal diseases, acute pulmonary congestion and premenstrual syndrome and in forced diuresis during and after surgery. Bumetanide may be given orally, intravenously or intramuscularly and produces a rapid and marked diuresis, and increased urinary excretion of sodium, chloride and other electrolytes (within 30 minutes) which persists for 3 to 6 hours. Its principal site of action is on the ascending limb of the loop of Henle, with a secondary action on the proximal tubule.17

Pharmacologically, bumetanide is about 40- fold more potent thanfrusemide (furosemide), with the exception of its effects on urinary potassium excretion where its potency is lower. Studies in patients with oedema due to congestive heart failure, pulmonary oedema or hepatic disease show that oral or intravenous bumetanide 0.5 to 2 mg/day produces results comparable to those with frusemide 20 to 80 mg/day. In acute pulmonary oedema, intravenous bumetanide produces a very rapid diuresis. Higher doses of bumetanide may be required (up to 15 mg/day) in patients with chronic renal failure or nephrotic syndrome. In these patients muscle cramps are not uncommon with bumetanide, but glomerular filtration rates are unaffected. In most studies, diuretic effects were accompanied by decreased bodyweight, abdominal girth and improvements in a variety of haemodynamic parameters.parameters.17

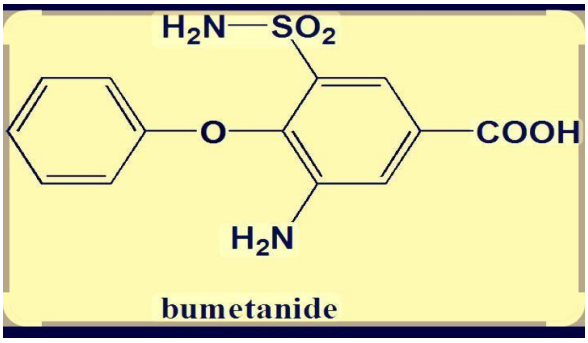

Figure ; 9 Bumetanide

• Structureal features

amino-5-sulfamoylbenzoic acid derivative.

Its chemical formula is \(C_{17}H_{20}N_{2}O_{5}S\), molecular weight of approximately 364.42 g/mol. (15)

• Indication

Bumetanide has United States Food and Drug Administration (FDA) approval for managing various edematous conditions secondary to cardiac failure with or without ascites or hepatic/renal disease, including nephrotic syndrome.18 It may also be indicated for refractory edema resistant to other loop diuretics. Bumetanide may be used alone or in conjunction with other antihypertensive agents in treating hypertension, although this is not an FDA-approved indication. Treating acute hypercalcemia is also an off-label indication for the drug.18Bumetanide may be an

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appropriate option for patients who have an allergic reaction to furosemide, another loop diuretic. Recent studies show seizures and behavioral problems in patients with tuberous sclerosis may be treated by agents that enhance GABA-anergic transmission by influencing chloride regulation.18 Bumetanide is also not FDA-approved for this purpose.

• Mechanism of action –

Diuretics play a crucial role in treating edema and hypertension by causing the induction of a negative balance of solute and water. Loop diuretics are physiologically the most potent family of diuretics, as demonstrated by possessing natriuretic and chloriuretic potency of the diuretic drug class. These agents potentially increase Na and Cl excretion to over 25% of the filtered load. Although they have no direct epithelial effect on segments such as the thin descending limb of Henle and the thick ascending limb of Henle, many diuretics decrease fluid reabsorption by abolishing the papillary osmotic gradient. Most loop diuretics have a direct inhibitory effect on the cotransport process, specifically by interfering with active chloride transport secondary to the presence of sodium. This is located on the luminal membrane of the segment. Loop diuretics (furosemide, bumetanide, torsemide, ethacrynic acid) inhibit the concentrating mechanisms in the medullary segment. In contrast, diuretics such as thiazides are effective primarily in the cortical segment and inhibit the urinary diluting mechanism.19

Bumetanide inhibits the reabsorption of sodium and chloride in the ascending loop of Henle and proximal renal tubule, which interferes with the chloride-binding cotransport system. This mechanism increases the excretion of water, magnesium phosphate, sodium chloride, and calcium. It decreases free water clearance and solute-free water reabsorption and increases sodium chloride excretion to the distal tubule (natriuresis), calciuria, phosphaturia, and minimal bicarbonaturia. Studies have shown that diuretic action onset occurs 0 to 30 minutes following intravenous use and 30 to 60 minutes following oral administration. The diuretic effect and the total duration of action last for 3 to 4 hours (270 min) with similar net urine output between intravenous and oral administration. The peak of the drug's action occurs at 90 minutes after oral administration.

• Administration –

Bumetanide is available in oral and injectable administrations (intravenous, intramuscular). Bumetanide is rapidly absorbed after oral and intravenous formulations. 95% of the drug extensively binds to plasma proteins and is eliminated by the metabolism of the butyl side chain and partially removed through urine excretion. The apparent half-life is 1.0 to 1.5 hours, and the volume of distribution is about 25 liters. Plasma clearance is 225 to 228 ml/min. Bumetanide has high bioavailability, between 80% and 100%; this means that oral and intravenous bioavailability are roughly equivalent, constructed to furosemide, where oral doses should be roughly double intravenous doses.

The different modes of administration of bumetanide with dosages are the following:

• Bumetanide oral tablet: 0.5 mg, 1 mg, 2 mg

• Bumetanide intramuscular/intravenous injection solution: 0.25 mg/1 mL Dosing regimens based on the condition are as follows:

• Edema

o Oral route of administration:

 0.5 to 10 mg per day orally divided once or twice daily. Initiate treatment of 0.5 to 2.0 mg daily, repeating every 4 to 5 hours until the appropriate response is obtained. The maximum dosage is 10 mg per day.

o Intramuscular/intravenous route of administration:

 0.5 to 10 mg per day intramuscularly/intravenously divided once or twice daily. Initiate treatment 0.5 to 1.0 mg per day over 1 to 2 minutes, repeating every 2 to 3 hours until obtaining the appropriate response. The maximum dosage is 10 mg per day.

 An alternate day schedule or schedule of 3 to 4 days on with a 1 to 2-day rest period may increase tolerability and effectiveness in continued edema control. Intravenous administration should be reserved for patients unable to take oral medications.

 Bumetanide solution is not Y-site compatible with midazolam and is admixed incompatible with dobutamine and milrinone.

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• Hypercalcemia (off-label):

o 1 to 4 mg intravenously every 1 to 4 hours; administer with saline to maintain the patient's urine output at 200 to 250 mL/hour.

• Hypertension (off-label):

o 0.5 to 2.0 mg daily by mouth divided into 1 or 2 doses.

Renal dosing: in patients with anuria, bumetanide is contraindicated. In patients with hepatic failure, dosing should remain at a minimum level and, if necessary, increased very carefully.

Data is lacking for the use of bumetanide in pregnancy and breastfeeding. No teratogenicity is expected based on data from other loop diuretics, although there is a potential risk for decreased placental perfusion based on the drug's mechanism of action. Clinicians should consider alternative therapy in patients breastfeeding high-risk infants.20

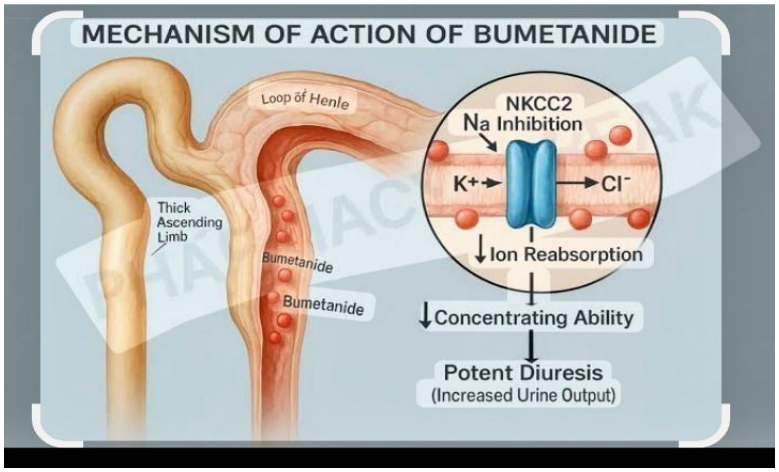

Figure : 10 Mechanism of Action of Bumetanide

• Bumetanide and furosemide drug compression drugs Reaction.

Furosemide (Lasix) and bumetanide (Bumex) are potent loop diuretics used to treat edema and high blood pressure by aiding the body in removing excess fluid. Bumetanide is generally 40 times more potent than furosemide, with higher bioavailability (\(80\%\) vs \(\sim40-50\%\)), making it often preferred for severe heart failure or diuretic resistance.

Furosemide and bumetanide are rarely prescribed together because they are both loop diuretics with similar mechanisms, making combined use generally redundant and increasing risks of dehydration and electrolyte imbalances. They act on the same part of the kidney (loop of Henle) to treat fluid retention, though bumetanide is about 40 times more potent.

We assessed the response to and handling of furosemide and bumetanide in 30 experiments with the former and 46 with the latter in normal subjects. Oral doses of furosemide (20, 40, and 80 mg) were used, and subjects received oral doses

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of 0.5, 1, and 2 mg bumetanide and intravenous doses of 0.5 and 1 mg bumetanide. Both drugs were quickly absorbed and peak urinary amounts were reached at 75 min (median). Approximately 30% of an oral dose of each drug was excreted unchanged in the urine with no evidence of dose-dependent elimination. After intravenous injection, 36% of the bumetanide was excreted unchanged. Consequently, bumetanide has an estimated bioavilability of 80% (approximately 40% for furosemide). The relationship between the logarithm of the urinary bumetanide excretion rate and the logarithm of the sodium excretion rate was described by a sigmoid-shaped dose-response curve, with a dose inducing half-maximal response of 1 ± 0.04 µg/min; it was 69.8 µg/min for furosemide. Overall, the distinguishing features between the two drugs are the 200% greater bioavailability and the much greater potency of bumetanide.

Figure : 11 Furosemide and Bumetanide

Future Scope –

Liver Cirrhosis is a progressive and irreversible liver disorder characterized by fibrosis, nodular regeneration, and liver dysfunction.

Despite current therapies, complete reversal is limited—so future research focuses on prevention, early detection, and reversal of fibrosis.

Development of Antifibrotic Drugs

Major future direction is targeted antifibrotic therapy Research focuses on:

1. Blocking hepatic stellate cell activation
2. Inhibiting collagen deposition
3. Modulating TGF-β signaling pathways

Goal: Reverse or halt liver fibrosis progression

Stem Cell Therapy and Regenerative Medicine

- Use of mesenchymal stem cells (MSCs)
- Liver regeneration through:
- Cell replacement
- Tissue repair

Anti-inflammatory effects

- Potential benefit:

1) Partial restoration of liver function without transplant

2) Gene Therapy and Molecular Targets

Identification of genes involved in fibrosis progression

Techniques:

1. Gene silencing (siRNA)
2. CRISPR-based correction

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3. Targeting:
4. Fibrotic pathways
5. Inflammatory cytokines

Artificial Intelligence & Early Diagnosis

1. AI-based imaging for early cirrhosis detection
2. Machine learning models using:
3. Blood biomarkers
4. Ultrasound/CT scans
5. Patient history.5-8

II. CONCLUSION

Both drugs increase urine output by blocking sodium and chloride reabsorption in the loop of Henle.In liver cirrhosis, they help remove excess fluid accumulation such as ascites and peripheral edema.Furosemide is commonly preferred as first-line therapy because it is inexpensive and widely available.

Bumetanide is more potent and may be useful in patients who do not respond adequately to furosemide or who have poor gastrointestinal absorption.

Both drugs can cause adverse effects such as:

- Hypokalemia
- Dehydration
- Hypotension
- Renal impairment 5-8

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