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DRUGS USED IN HEART FAILURE

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Abstract: Heart failure is the most commoncardiovascular reason for hospital admission for people older than 60years of age.Few areasin medicine have progressed as remarkably as heart failure treatment over the past three decades. However, progress has been consistent only for chronic heart failure with reducedejection fraction, none of the treatments tested to dars have been definitively proven to improve survival.Delaying or preventing heart failure has become increasingly important in patients who are prone to heart failure.The prevention of worsening chronic heart failure and hospitalisations for acute decompensation is also of great importance.

Keywords: Therapeutic strategies, cytostatics, antiarrhythmics, positive inotropic drugs, vasodilators, cardiac glycosides, beta adrenoceptor antagonists, toxic responses, anesthetics.

INTRODUCTION

The effects of cardiac glycosides is more pronounced than other drugs used in heart failure. This article is intended to provide more detailed information the physiological mechanisms of the superiority of cardiac glycosides in heart failure over other drugs attempted.

Heart failure is a clinicalthat ispredominantly causes by cardiovascular disordes such as coronary heart disease and hypertension.This articleaims to deeply study and analyze each groups of drugs used in heart failure. Today, heart failure is one of the most common heart pathologies, and it is necessary to organize the treatment of this disease early and carry out excellent treatment.

METHODS

This review article synthesizes current literature and clinical guidelines to elucidate the pharmacological interventions employed in heart failure management. The information presented here is structured according to the IMRAD (Introduction, Methods, Results, and Discussion) format to facilitate a systematic exploration of the topic.We studied the structure of cardiac glycosides using, for example of sugar and analyzed its parts. We analyzed the pharmacokinetics and pharmacodynamics of its parts. We studied the pharmacokinetics of the glycone part and the pharmacodynamics of aglycon part. The pharmacodynamic effects of the algycon part are the main functional effects of the heart.

RESULTS

1.Angiotensin-Converting Enzyme (ACE) Inhibitors:

Mechanism of Action: ACE inhibitors block the conversion of angiotensin I to angiotensin II, thereby dilating blood vessels and reducing blood pressure.

Examples: Enalapril, lisinopril, ramipril.

Clinical Efficacy: ACE inhibitors have demonstrated efficacy in improving symptoms, reducing hospitalizations, and prolonging survival in heart failure patients.

2.Angiotensin II Receptor Blockers (ARBs):

Mechanism of Action: ARBs antagonize the action of angiotensin II, leading to vasodilation and decreased aldosterone secretion.

Examples: Losartan, valsartan, candesartan.

Indications: ARBs are indicated in patients intolerant to ACE inhibitors or as an alternative therapy.

3.Beta-Blockers:

Mechanism of Action: Beta-blockers inhibit the effects of catecholamines on the heart, reducing heart rate and myocardial contractility.

Examples: Metoprolol, carvedilol, bisoprolol**.**

Clinical Benefits: Beta-blockers improve left ventricular function, reduce hospitalizations, and enhance survival rates in heart failure patients.

4.Diuretics:

Mechanism of Action: Diuretics promote the excretion of sodium and water, alleviating fluid retention and symptoms of congestion.

Examples: Furosemide, hydrochlorothiazide, spironolactone.

Clinical Considerations: Diuretics are essential for managing volume overload in heart failure, although long-term use may be associated with electrolyte imbalances and renal dysfunction**.**

5.Aldosterone Antagonists:

Mechanism of Action: Aldosterone antagonists inhibit the effects of aldosterone, reducing sodium retention and myocardial fibrosis.

Examples: Spironolactone, eplerenone**.**

Indications: Aldosterone antagonists are recommended in patients with moderate to severe heart failure and reduced ejection fraction, particularly those at risk of fluid retention and potassium imbalance.

6.Digoxin:

Mechanism of Action: Digoxin increases myocardial contractility and reduces heart rate through its effects on intracellular calcium levels.

Clinical Utility: Digoxin is reserved for patients with heart failure and atrial fibrillation or those with persistent symptoms despite optimal medical therapy.

7.Other Agents:

Sodium-Glucose Cotransporter-2 (SGLT2) Inhibitors: Emerging evidence supports the use of SGLT2 inhibitors in heart failure patients with or without diabetes, offering benefits in terms of reducing heart failure hospitalizations and cardiovascular mortality.

Sacubitril/Valsartan (Entresto): This combination drug, comprising a neprilysin inhibitor and an angiotensin receptor blocker, has shown superiority over ACE inhibitors in reducing cardiovascular death and heart failure hospitalizations.

DISCUSSION

The pharmacological management of heart failure necessitates a multifaceted approach tailored to individual patient characteristics and disease severity. While numerous drugs have demonstrated efficacy in improving outcomes and alleviating symptoms, their judicious use requires careful consideration of potential adverse effects, drug interactions, and patient comorbidities. Furthermore, ongoing research efforts seek to **uncover novel** therapeutic targets

and refine existing treatment strategies to enhance the prognosis of individuals afflicted by this debilitating condition

Cardiac effects of cardiac glycosides include:

- Positive inotrope
- Positive tonotrope
- Negative chromotrope
- Negative dromotropic
- Positive botmotrope.

Pharmacologic therapies for heart failure include the removel of retained salt and water with diuretics; reduction of afterload and salt and water retention by means of angiotensin-converting enzyme(ACE) inhibitors; reduction of excessive sympathetic stimulation by means of beta blockers; reduction of preloador afterload with vasodilators; and systolic failure, direct augmentation of depressed cardiac contractility with positive inotropic drugs such as digitalis glycosides.

Cytostatics. Anthracyclines. The anthracyclines are effective antineoplastic agents which are used in the treatment of many types of malignancy. The antracyclines are antibiotic agents which antineoplastic action results from the inhibition of the nucleicacid synthesis by binding to both strands of the deoxyribonucleic acid (DNA) helix. Cyclophosphamide is an alkylating agent that causes cytotoxicity by its biologically active metabolites.Mitoxantrone is structurally related to doxorubicin and has also been associated with the development of left ventricular impairment. Furthermore, cumulative mitoxantrone dose was associated with an increasing risk of cardiotoxicity.

Antiarrhythmics. The cardiodepressant adverse effects antiaarrhythmicdrugs can mainly be attributed to their negative inotropic properties. Particularly, in patients with preexistent left ventricular impairment, antiarrhythmics can be induce or exacerbate congestive heart failure (CHF).The degree of negative inotropy may vary from drug to drug.

Positive inotropic drugs. Dobutamine and dopamine are often useful in acute failure in which systolic function is markedly depressed. However, they are not appropriate for chronic failure because of tolerance, lack of oral efficacy, and significant arrhythmogenic effects. Digoxin antibodies are extremely effective and should always be used if other therapies appear to be failing. They are effective for poisoning with several cardiac glycosides am addition to digoxin and may save patients who would otherwise die.

Vasodilators. These agents have been shown to reduce morbidity and mortality and chronic heart failure. Although they have no direct positive inotropic action, angiotensin antagonists reduce aldosterone secretion, salt and water retention and vascular resistance. They are now considered, along with diuretics, to be first-line drugs for chronic heart failure. The ANG II receptor blockers (ARBs, eg,**losartan**) appear to have the same benefits as ACE inhibitors (eg, **captopril**), although experience with ARBs is not as extensive.

Cardiac glycosides. Digitalis glycosides are no longer considered first-line drugs in the treatment of heart failure. However, because they are not discussed elsewhere in this book, we begin our discussion with this group. All cardiac glycosides are cardenolides (they include a steroid nucleus and a lactone ring); most also have one or more sugar residues, justifying the glycoside designation. The cardiac glycosides are often calles digitalis (foxglove) plant. Current clinical evidence suggests that acute heart failure should be treated with a loop diuretics.

Beta adrenoceptor antagonists. Several beta blockers (**carvedilol, labetalol, metoprolol**) have been shown in long-term studies to slow professionof chronic heart failure. The benefit of

beta blockers had long been recognized in patients with hypertrophic cardiomiopathy but has also been shown to occur in patients without cardiomiopathy. Nebivolol a beta blocker with vasodilator effects approved for the treatment of hypertension, is investigitionalin heart failure. Beta blockers are of no value in acute and may be detrimental id systolic dysfunction marked.

Toxic responses. Increased automaticity, caused by intracellular calcium overload, is the most important manifestation of digitalis toxicity. Intracellular calcium overload results in delayed afterdepolarizations, which may evoke extrasystoles, tachycardia or fibrillation in Abu part of the heart.

Anesthetics. During general anesthesia, cardiovascular homeostassis will be subject to several influences. Patients characteristics, such as age and concomitant comorbidity, intravenous fluid administration, surgical procedures and medication used during general anesthesia may all affect cardiovascular homeostasis. Therefore, the occurrence or exacerbationof signs and symptoms of CHF in relationship with general anesthesia cannot always easily be attributed to a spesific agent used during general anesthesia. Nevertheless, a number of agents have negative effects on myocardial contractility. The halogenated volatile anesthetics halothane and enflurane both have mild negative inotropic effects. In particular, halothane has been associated with cardiodepressant effects. Negative inotropic effects, however, have been found to be more pronounced in patients with preexisting left ventricular impairment. Cardiodepressant effects seem to be less frequent when using newer agents such as isoflurane and desflurane, although it has been suggested that these differences may apply only to young patients. The intravenous barbiturate anesthetics thiopental and methohexital may also depress myocardial contractility. In general, the hemodynamicconsequences of usual plazma levels of these agents are limited.

CONCLUSION

In conclusion, the pharmacotherapy of heart failure represents a dynamic field characterized by continuous advancements and refinements. The judicious selection and optimization of medications, in conjunction with lifestyle modifications and device-based therapies, constitute integral components of a comprehensive management strategy for heart failure patients. By leveraging the collective insights gleaned from clinical trials, real-world data, and translational research endeavors, healthcare providers can strive to optimize outcomes and improve the quality of life for individuals grappling with this challenging cardiovascular disorder.

Medicines used in heart failure have spesific tasks for each group. In the process of studying them, we analyzed the characteristics of each group more deeply. Knowing the side effects of drugs used in heart failure, it is necessary to use them correctly. For this, we need to better study the types of each drug according to the specific stage of the disease and which receptors it affects. Knowing their pharmocology, we can organize the treatment correctly.

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