Heart failure in questions and answers

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Is the use of combination therapy with furosemide and torasemide justified in patients with heart failure? Which diuretics can be combined to achieve a higher diuretic effect? Is this a reasonable option in patients with edema? In the treatment of resistant edema, 2 loop diuretics may be used in combination. The recommendation on the combination treatment using different loop diuretics despite their synergic effect is a result of the observed differences in bioavailability of these drugs, a longer duration of effect of torasemide compared with furosemide, and additional aldosterone-lowering properties of torasemide.

In patients with high blood pressure and fluid retention, diuretics from different classes may also be combined, for example, a loop diuretic plus a thiazide diuretic plus an aldosterone antagonist. Such therapy uses different mechanisms to increase the diuretic effect.

In patients with renal failure with glomerular filtration rates of 35 ml/min/1.73 m² or less, the efficacy of thiazide diuretics and aldosterone antagonists is low, and these drugs are therefore not recommended.

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Is immunization against influenza and pneumococcal infections recommended in patients with heart failure? Patients with heart failure should be vaccinated to reduce incidence of influenza and pneumococcal disease and/or to alleviate the infections. Infection can dramatically worsen the course of heart failure and may lead to death and hospital admissions.

Choosing between a less and a more cardioselective β-blocker (carvedilol/nebivolol versus bisoprolol) in the treatment of heart failure: what to choose when? In patients with heart failure with impaired contractile function, all β-blockers allowed by guidelines may be used. In addition to the drugs listed in the title, metoprolol may also be used. These drugs can be used even in patients with asthma or chronic obstructive pulmonary disease. In this group of patients, starting from low doses of a β-blocker and titration up to optimal or maximum tolerated doses is recommended. Reduced mortality and exacerbations of these chronic respiratory diseases were shown in patients treated with β-blockers.

However, β-blockers show some differences, including in their electrophysiological effects, which makes metoprolol and bisoprolol more effective than carvedilol and nebivolol in patients with arrhythmias.

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Is it reasonable to use a low maintenance dose of a β-blocker (eg, carvedilol, 6.25 mg/d) in a patient with heart failure in whom the dose cannot be increased because of bradycardia or atrioventricular block? What should be the target dose? Based on the results of clinical trials, so called optimal doses of β-blockers, associated with optimal effects on mortality and exacerbation rates, were defined. The doses are as follows: for metoprolol, 20–200 mg/d; for carvedilol, 50–100 mg/d; for nebivolol, 10 mg/d; and for bisoprolol, 10 mg/d. In patients treated with suboptimal doses of β-blockers, beneficial effects were also seen but they were markedly lower compared with the optimal doses. Therefore, it is reasonable to use even low doses of β-blockers.
A patient with severe edema of the lower extremities, ascites, tachycardia, low blood pressure, and previously detected markedly reduced ejection fraction refuses hospitalization. Which drugs are the safest to start outpatient treatment? The most important thing is patience and repeated offer of hospital admission. In 95% of cases, professional, calm, and clear argumentation results in the patient’s consent to hospitalization. However, if the patient does not give such consent, I suggest obtaining (in a reassuring and clear manner) a written (by a patient or by his or her guardian) refusal to admission. If this is not possible, an annotation of refusal in the presence of a witness (eg, a nurse) is sufficient. Such situation is not included in the European Society of Cardiology (ESC) guidelines, but logically we have to reduce a number of hypotensive drugs in the treatment of heart failure. In such circumstances, I suggest starting treatment with:

1. a digitalis glycoside (an inotropic drug to improve myocardial contractility) combined with a loop diuretic and a low-dose aldosterone antagonist (eg, spironolactone, 25 mg/d, or eplerenone, 25 mg/d);
2. ivabradine (only in patients with sinus rhythm of 75 bpm or higher) combined with a loop diuretic and a low-dose aldosterone antagonist (eg, spironolactone, 25 mg/d, or eplerenone, 25 mg/d).

In patients with persistent heart rate of 75 bpm or higher despite the therapy with a digitalis glycoside and ivabradine in doses recommended by the guidelines, I suggest using a combination therapy with ivabradine and digoxin, and add a loop diuretic and a low-dose aldosterone antagonist. In patients with contraindications to β-blockers, the ESC guidelines recommend the use of digoxin and ivabradine in the treatment of heart failure. Such patients require follow-up visits even once a week. When systolic blood pressure exceeds 100 mmHg (this effect is usually achieved after using at least 2 weeks of the treatment described above), adding a low-dose angiotensin-converting enzyme inhibitor under blood pressure monitoring should be considered.

In the case of a lack of therapeutic effect, I suggest reducing the dose of metoprolol to 25 mg/d and start a loop diuretic. Furthermore, when the sinus rhythm is 75 bpm or higher, it is necessary to add ivabradine in a dose sufficient to achieve a heart rate of 50 to 65 bpm. Another alternative for the patient with persistent heart rate of 75 bpm or higher (sinus rhythm or atrial fibrillation) is addition of a digitalis glycoside, which has inotropic effects and will make it possible to treat hypertension.