Angiotensin converting enzyme inhibitors

ACEi, ARBs effect on kidney:

Angiotensin-converting enzyme inhibitors (ACE inhibitors, ACEI) belong to the group of cardiopharmaceuticals. Their administration leads to a **decrease in the production of angiotensin II** (AGII) and to **accumulation of bradykinin**. The result is dilatation of arterioles and venules. In the kidneys, the excretion of Na + and water increases, and the excretion of K + decreases. By a mechanism not yet fully elucidated, ACEIs are able to **prevent myocardial remodeling** after acute myocardial infarction. In addition, long-term administration regresses already developed hypertrophic changes in blood vessels and the heart.

They are well absorbed from the GIT and, except for captopril, their absorption is not affected by the current presence of food. They are administered either in **active** form (captopril, lisinopril) or in the vast majority as **prodrugs** and are metabolized in the liver to the active substance (eg enalapril enalaprilat, perindopril perindoprilat, spirapril spiraprilate). ACEIs are **excreted** predominantly by the **kidneys**. Therefore, in renal impairment, the dose should be reduced.

To optimize dosing, it is important to divide the ACEI according to the biological half-life into short-acting, medium-acting, and long-acting (see table).

Representatives and their properties

Distribution of ACE inhibitors by duration of action and dosage (according to Widimsky, 2000)[1]

Drug	onset time (h)	duration of effect (h)	daily starting dose	daily maintenance dose
Short term				
captopril	0,25	6–8	3 × 6,25 mg	2–3 × 50 mg
Medium term				
enalapril	4	12	2,5 mg	2 × 10 mg
quinapril	2	12	2,5–5 mg	2 × 5–10 mg
Long term				
perindopril	3	24	2 mg	1 × 4 mg
ramipril	2	24	1,25–2,5 mg	2 × 5 mg
spirapril	1–3	24	3 mg	1 × 6 mg
trandolapril	3	24	0,5 mg	1 × 4 mg
lisinopril	6–8	24	2,5 mg	1 × 30–32,5 mg
fosinopril	1	24	5 mg	1 × 10–20 mg
moexipril	1–2	24	7,5 mg	1 × 7,5–15 mg

Indication

The basic indications for ACE inhibitors include:

- **arterial hypertension**;

- **heart failure**;
- **acute myocardial infarction**;
- **diabetic nephropathy**.

Contraindication

- **Renal artery stenosis** – bilateral or unilateral in solitary kidney.
- **Pregnancy** , **lactation** and **fertile age** in a woman not using reliable contraception.
- **Cardiogenic shock**.
- Occurrence of **angioneurotic edema** after another ACE inhibitor.

Side effects

- **Hypotension** after the first dose - occurs mainly in patients treated with higher doses of diuretics for a long time. Therefore, it is advisable to reduce the dose of diuretics before starting treatment with ACEI or to discontinue them for a short time (24-48 hours) and start treatment with lower doses of ACEI.
- **Impaired renal function** - creatinine and urea levels should be monitored regularly. In the case of progressive deterioration, the dose of the diuretic may first be reduced. If the decline in renal function continues, ACEI should be discontinued.
- **Hyperkalemia**.
- **Cough** - unpleasant, dry, irritating. The cause is the accumulation of bradykinin in the tissues. The solution is to exchange ACEIs for blockers of AT 1 -receptors for angiotensin II.
- **Angioneurotic edema** – a cause similar to cough.

Interaction

- Administration of potassium-sparring diuretics or potassium supplementation may lead to hyperkalaemia in patients treated wit hACEIs.
- Patients taking diuretics may be particularly sensitive to the hypotensive effect of ACE inhibitors. The risk of hypotension is further increased by antihypertensives, anesthetics, sedatives, nneuroleptics and antidepressants.

Links

Related articles

- Renin-angiotensin-aldosterone system
- Angiotensin II receptor blockers
- Treatment of ischemic heart disease
- Hypertension
- Hypertensive crisis

Source

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Reference

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