Hyperkalemia treatment guidelines uptodate

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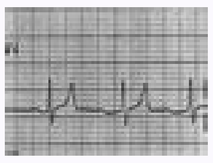
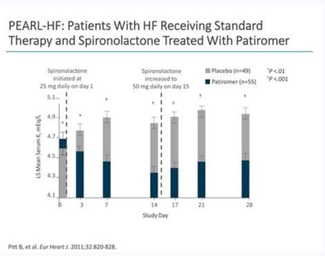


Definitions of the severity of hyperkalemia

Author	Year	Mild	Moderate	Severe
Levinsky	1966	< 6.5 mmol/l	6.5–8 mmol/l with ECG : peaked T-waves	>8 mmol/l or any level + prolongation of the QRS complex/ventricular arrhythmias/heart block
Vanden Hoek *et al.*for American Heart Association	2005	5.1–5.9 mmol/l	6.0–6.9 mmol/l	>7 mmol/l
Soar *et al.*for the European Resuscitation Council	2010	5.5–5.9 mmol/l	6.0–6.4 mmol/l	≥6.5 mmol/l
El-Sherif and Turitto	2011	5.5–7.5 mmol/l	7.5–10 mmol/l	>10 mmol/l



SCGH ED Code STEMI Protocol

Is it an acute STEMI?

Pain < 12/24

ECG changes consistent with STEMI
- ST elevation >1mm in 2 contiguous limb leads or
- ST elevation >2mm in 2 contiguous chest leads

Does it meet criteria to activate "CODE STEMI"?

Inclusion criteria
- Symptom onset < 12 hours
- <85 years
- Ongoing pain
- Mobile and independent

Exclusion criteria - for discussion
- >85 years
- LBBB
- Prior CABG
- Significant comorbidities
- Out of hospital cardiac arrest
- Pulmonary oedema
- Cardiogenic shock
- Recent major surgery (<2/52)
- Active bleeding

ED consultant / night SR discuss with on-call General Cardiologist (not cardiology registrar)

Is primary intervention appropriate?

Yes

No

Activate "CODE STEMI" where appropriate

ED consultant / night SR
DIAL 55
State "CODE STEMI ED now"

Medical Management

Patient preparation in ED

- Aspirin 300mg
- Ticagrelor 180mg or
  - *If contraindicated Prasugrel 60mg or*
  - *If contraindicated consider Clopidogrel 600mg*
- Heparin 5000 IU IV
- Prepare for transfer

Contraindications to $P2Y_{12}$ inhibitors
- Active bleeding
- Allergy

Ticagrelor:
- Heart rate <50 bpm
- Chronic dyspnoea
- History ICH / TIA / Stroke

Prasugrel:
- Weight < 60kg
- Age > 75 yrs
- History ICH / TIA / Stroke

Clopidogrel
- History ICH

Transfer to Cath Lab (CVIL)

Suitable for rapid transfer
- Transfer with ED RN and ED Dr
- Work hours 0730-1700, transfer without delay
- After hours, transfer 10 minutes after Code STEMI activation
- Record time of arrival on ECG
- **ED staff to stay with patient until appropriate CVIL team members arrive**

Not suitable for rapid transfer
- Notify cardiology registrar
- Remain in ED until appropriate CVIL team members arrive and call for patient
- Transfer to CVIL with ED RN and cardiology registrar

Hyperkalemia treatment guidelines 2020. Acute hyperkalemia treatment guidelines.

ACE inhibitors are medications used to treat and manage hypertension, which is a significant risk factor for coronary disease, heart failure, stroke, and a host of other cardiovascular conditions. Most cases are primary and not attributable to any specific etiology. This activity reviews the indications, contraindications, activity, adverse events, and other key elements of ACE inhibitor therapy in the clinical setting related to the essential points needed by members of an interprofessional team managing the care of patients with hypertension and its related conditions and sequelae. Objectives: Identify the mechanism of action of ACE inhibitors.Summarize monitoring/follow-up for patients initiated on ACE inhibitor therapy.Review the dosing parameters adjusting or replacing ACE inhibitor therapy based on adverse events or inadequate therapeutic response.Explain the importance of ACE inhibitor therapy in hypertension management and how it affects therapeutic strategy as it pertains to improving care coordination and communication among the interprofessional team when using these agents to achieve therapeutic outcomes. Access free multiple choice questions on this topic. Angiotensin-converting enzyme inhibitors (ACEIs) are the most commonly indicated medications in the treatment of cardiovascular and renal diseases, including heart failure, acute coronary syndrome, nephrotic syndrome, diabetes, and hypertension.[1] Hypertension Angiotensin-converting enzyme inhibitors effectively lower the mean arterial blood pressure as well as systolic and diastolic blood pressure both in hypertensive and normotensive subjects.[2][3] Angiotensin-converting enzyme inhibitors have been evaluated as antihypertensive drugs in multiple randomized controlled trials.[4] In 2014, the Eighth Joint National Commission (JNC8) published evidence-based guidelines for treating high blood pressure in adults, which recommended that ACE inhibitors are one of four drug classes recommended for initial therapy for adults with elevated blood pressure.[5] The other three classes of drugs are calcium channel blockers, thiazide diuretics, and angiotensin receptor blockers, which are useful as initial therapy for the general nonblack population. Only thiazide and calcium channel blockers are recommended as initial therapy for the general black population with elevated blood pressure.[6] Recent guidelines released by the American Heart Association/American College of Cardiology (AHA/ACC) and the European Society of Cardiology (ESC) also recommend ACE inhibitors as first-line antihypertensive therapy, especially in patients with diabetes mellitus and cardiovascular diseases.[7][8] Although ACE inhibitors are generally very effective antihypertensive drugs, they have been proven to be less effective in hypertensive Black race individuals than Whites in clinical practice.[9] Heart Failure Angiotensin-converting enzyme inhibitors (ACEIs) improve heart failure by decreasing afterload, preload, and systolic wall stress, which results in increased cardiac output without any increase in heart rate.[10][11] ACE inhibitors play an important role in promoting salt excretion by augmenting the renal blood flow and reducing aldosterone and antidiuretic hormone production. Apart from decreasing the afterload, ACEIs also reduces cardiac myocyte hypertrophy. Since the 1980s, several large, prospective, randomized, placebo-controlled trials have proved that treatment with ACE inhibitors reduces overall mortality, especially in patients with heart failure with reduced ejection fraction (0HFrEF).[12][13][14] These trials demonstrated that ACE inhibitors reduce mortality even in asymptomatic patients with left ventricular dysfunction.[15] Based on the above-mentioned evidence, ACE inhibitors are strongly recommended as first-choice therapy in patients with heart failure.[16][17] Post Myocardial Infarction Over the last few decades, several prospective, randomized trials have studied the effect of ACE inhibitors on mortality after myocardial infarction (MI).[18][19] The vast majority of these trials have shown a significant decrease in mortality and a slowing of the progression to congestive heart failure after MI in patients treated with ACE inhibitors.[20] The clinical practice guidelines in the contemporary era recommend that patients with left ventricular dysfunction or heart failure be treated with ACE inhibitors without delay after infarction. It is also recommended that all patients should be treated with ACE inhibitors initially, with a review of the need for continuation later based on left ventricular function assessment.[21] Diabetes The Renin-Angiotensin-Aldosterone system and increased glomerular capillary pressure have been reported to increase the progression of renal dysfunction due to diabetes mellitus related nephropathy.[22] A large, prospective, randomized, placebo-controlled has demonstrated that CE inhibitors slow down the progression of nephropathy in patients with insulin-dependent diabetes mellitus and significantly reduce the combined endpoints of dialysis, transplantation, and death.[23] Current recommendations are using ACEi or ARB as first-line therapy for hypertension in patients with a history of diabetes. Also, the use of ACEi in diabetic hypertensive patients with no history of coronary heart disease has been shown to decrease the incidence of myocardial infarction and improved heart function.[24] Nephrotic Syndrome or Proteinuria Angiotensin-converting enzyme inhibitors have been reported to decrease glomerular capillary pressure by decreasing arterial pressure and selectively dilating efferent arterioles.[25] It has been shown that the use of ACE inhibitors prevents the progression of microalbuminuria to overt proteinuria.[26] Angiotensin-converting enzyme inhibition provides long-term protection against the development and progression of proteinuria and stabilizes renal function in previously untreated patients with impaired renal function.[26] Chronic Kidney Disease ACE inhibitors or ARBs are the first-line drugs in managing chronic kidney disease (CKD) patients. The use of ACEI or ARB has been proven to have a superior effect compared to placebo treatment on decreasing proteinuria and slowing kidney disease progression. The efficacy of ACEI and ARB is comparable.[27]Glomerular Disease and Post-transplant Glomerulonephritis\The use of ACE inhibitors or ARB is the mainstay of treatment in patients with glomerular diseases. It slows down the decline in glomerular filtration rate (GFR) and proteinuria.[28] The use of renin-angiotensin-aldosterone inhibitors prolongs graft survival in patients with post-transplant glomerulonephritis.[29]Angiotensin II causes direct vasoconstriction of precapillary arterioles and postcapillary venules, inhibits the reuptake of norepinephrine, stimulates the release of catecholamines from the adrenal medulla, reduces urinary excretion of sodium and water, stimulates synthesis and release of aldosterone, and stimulates hypertrophy of both vascular smooth muscle cells and cardiac myocytes.[30][31] The exact mechanism of ACE inhibitors is not fully known. They interfere with the renin-angiotensin-aldosterone system, but their effect is not directly related to renin levels in the blood. As the name implies, ACE inhibitors block an angiotensin-converting enzyme that converts angiotensin I to angiotensin II. Decreased production of angiotensin II enhances natriuresis, lowers blood pressure, and prevents remodeling of smooth muscle and cardiac myocytes. Lowered arterial and venous pressure reduces preload and afterload. Also, the hypothesis is that ACE inhibitors interfere with the degradation of bradykinin, a peptide that causes vasodilation.[32]Angiotensin-converting enzyme regulates the balance between the vasodilatory and natriuretic properties of bradykinin and the vasoconstrictive and salt-retentive properties of Angiotensin II. ACE inhibitors alter this balance by decreasing the formation of Angiotensin II and the degradation of bradykinin. ACE inhibitors also alter the formation and degradation of several other vasoactive substances, such as substance P, but the contribution of these compounds to the therapeutic or adverse effects of ACE inhibitors is uncertain.[33]ACE inhibitors differ in their chemical structure, potency, bioavailability, plasma half-life, route of elimination, as well as their distribution and affinity for tissue-bound angiotensin-converting-enzyme.Depending on the chemical structure, ACE inhibitors are classified into three groups.[34][35]Sulfhydryl-containing ACE inhibitorsCaptopril – Hypertension therapy is 25 mg, either BID or TID, with a maximum of 450 mg. Heart failure therapy is 6.25 mg TID, with a maximum of 450 mg.Dicarboxylic-containing ACE inhibitors: see table.Phosphorus-containing ACE inhibitor. Fosinopril – Hypertension therapy dosing is 10 mg, increasing to a maximum dose of 80 mg. May split into two equal doses during the day to control blood pressure. Heart failure therapy is 5 to 10 mg daily to a maximum dose of 40 mg. General Dosing Information All of the ACE inhibitors are prescribed orally, except for enalapril, which can be given intravenously. Enalapril's IV dosage is initially 0.625 to 1.25 mg every 6 hours. Dosage titration up can be to 5 mg IV every 6 hours. Geriatric dosing should definitely initiate at the lower end of the adult dosing regimen.There should be a dosage decrease in patients with heart failure, salt-depleted patients, and/or renal impairment. Lisinopril and captopril are the only ACE inhibitors that do not have to be activated in the body to be effective. All the other ACE inhibitors are prodrugs and require activation. Most reach peak serum levels within 1 hour after ingestion. Since most of the activation occurs in the liver, a non-prodrug form is preferable in patients with underlying liver issues.[36]About 1 to 10% will develop a dry, nonproductive paroxysmal cough, and there is no treatment for the cough.[37][38] Experimental studies have shown that using non-steroidal anti-inflammatory agents (NSAIDs) and intermediate-dose aspirin (500 mg) can help with ACE inhibitors induced cough.[39] ACE inhibitor-induced cough is reported more frequently among women than men.[40] The cough is usually dry, and it often requires cessation of therapy.Angioedema is the most significant adverse effect of ACEi. It can affect any part of the body, including the intestine, but the most concerning is edema of the tongue, glottis, and/or larynx, causing airway obstruction.[41] Angioedema has a higher rate of incidence in the African-American population. When airway compromise is present, the treatment always secures the airway with an endotracheal tube that allows ventilation until the edema resolves. Multiple treatments have been tried, such as diphenhydramine, methylprednisolone, and epinephrine. Also, fresh frozen plasma and the newer agents, bradykinin blocking agents, have been tried. There are case reports that these bradykinin blocking agents might improve the angioedema, but no literature exists proving that they are better than the other agents.[42] There is an ongoing phase III trial at this time.Life-threatening anaphylactoid reactions have occurred in patients receiving Hymenoptera venom desensitization treatment and patients receiving dialysis with high-flux membranes. Treatment includes diphenhydramine, epinephrine, and blood pressure support with fluids and catecholamines.[43][44]Angiotensin-converting enzyme inhibitors have been reported to cause a reversible decline in renal function. Those with heart failure who depend on the renin-angiotensin-aldosterone system may develop changes in renal function with the use of ACE inhibitors.[45] Also, about one-fifth of the patients with renal artery stenosis develop increases in blood urea nitrogen and creatinine.[46] Any patient who already has a renal insufficiency is susceptible to a worsening of renal function. The renal function requires monitoring during treatment for susceptible groups.[47]As with any medication that lowers blood pressure, hypotension is an adverse reaction. Those at risk for this side effect: heart failure with systolic blood pressure below 100 mmHg, ischemic heart disease, cerebrovascular disease, hyponatremia, high dose diuretic therapy, renal dialysis, or severe volume and/or sodium depletion.[48]ACE inhibitors may cause hyperkalemia. Those at risk for this side

effect: prior history of renal impairment and/or diabetes, simultaneous use of potassium-sparing diuretics, and/or potassium supplements.[49] Treatment depends upon the potassium level, EKG changes, and whether the patient still has kidney function and produces urine.[47] There has been one report of increased sudden death in patients taking ACE inhibitors and co-trimoxazole simultaneously. The mechanism is believed to be hyperkalemia.[50]Cholestatic jaundice or hepatitis is another rare but serious adverse effect that can progress to hepatic necrosis and sometimes death. The clinician needs to discontinue the drug, and the patient managed appropriately.[51]ACE inhibitors are contraindicated in patients with a history of angioedema or hypersensitivity related to treatment with an ACE inhibitor and those with hereditary or idiopathic angioedema.[52] These drugs should not be given to patients already taking a direct renin inhibitor such as aliskiren.ACE inhibitors should not be given in pregnancy. They were Category D in pregnancy under the old FDA system because it is known to cause skull hypoplasia, anuria, hypotension, renal failure, lung hypoplasia, skeletal deformations, oligohydramnios, and death.[53]The combination therapy of ACEi and ARBs does not reduce mortality in patients with heart failure compared to monotherapy. In fact, the combination therapy can worsen renal indices and cause life-threatening hyperkalemia.[54]Renal function and electrolytes need to be checked regularly due to the effects of the drug on the renin-angiotensin-aldosterone system. For patients with increasing potassium, drop-in GFR, or increasing creatinine, the drug needs to be adjusted accordingly or discontinued.[55]Excessive doses of ACE inhibitors are usually well-tolerated, but they can cause hypotension, drop-in GFR, and electrolyte derangements. In addition, since ACE inhibitors block aldosterone, they can cause hyperkalemia and hyponatremia.[56][57]If the patient presents within 1 hour of ingestion and is awake and able to protect their airway, activated charcoal can be given. If they remain asymptomatic for 6 hours of observation, they can be considered medically stable for either discharge or referral to psychiatry.[58]For those with hypotension, fluids should be the first line of therapy. There is no antidote for ACE inhibitor poisoning. Naloxone has been used in captopril overdoses in the past with some success and can be a choice if intravenous fluids are not successful. Also, vasopressors are an option for the treatment of hypotension.[59]ACE inhibitors are one of the most widely used drugs for hypertension and heart failure, but their popularity does not mean they do not require the management of an interprofessional team. Besides nephrologists and cardiologists, these drugs are widely prescribed by nurse practitioners and primary care providers. While ACE inhibitors are relatively safe, a pharmacist should examine the patient's medication record to verify dosing and check for drug-drug interactions. Nursing can provide patient counsel, monitor for interactions and adverse events, and report any issues to the prescriber. It is important to monitor renal function and levels of electrolytes regularly.[60] Because many ACE inhibitors are currently available, it is important to keep up with the guidelines and recommendations, and the pharmacist can help the prescriber in this area.[61] An interprofessional team approach will optimize ACE inhibitor therapy resulting in improved patient outcomes. [Level 5]Review QuestionsDicarboxyl-containing ACE inhibitors and doses. Contributed by Linda L Herman 1.Nasution SA. The use of ACE inhibitor in cardiovascular disease. Acta Med Indones. 2006 Jan-Mar;38(1):60-4. [PubMed: 16479034]2.Vidt DG, Bravo EL, Fouad FM. Medical intelligence drug therapy: captopril. N Engl J Med. 1982 Jan 28;306(4):214-9. [PubMed: 7033784]3.Todd PA, Heel RC. Enalapril. A review of its pharmacodynamic and pharmacokinetic properties, and therapeutic use in hypertension and congestive heart failure. Drugs. 1986 Mar;31(3):198-248. 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22/07/2022 · ACE inhibitors is a class of drugs prescribed to control high blood pressure; and for the treatment and prevention of heart attacks, heart failure, and prevent kidney disease. Common side effects are headache, cough, rash, dizziness, and chest pain. Drug interactions, uses, dosage, and pregnancy safety information are provided. 11/12/2021 · Losartan (Cozaar) is a prescription medication used to treat high blood pressure, reduce the risk of stroke in those with hypertension, type 2 diabetes, diabetic neuropathy, and kidney disease. Side effects include chest pain, diarrhea, dizziness, fatigue, nasal congestion, UTIs, and weakness. Losartan belongs to the angiotensin receptor blockers (ARBs) drug class. 120 to 360 mg PO once daily. Clinical practice guidelines recommend a nondihydropyridine calcium channel blocker to slow the ventricular heart rate in patients with paroxysmal, persistent, or permanent atrial fibrillation. A nondihydropyridine calcium channel blocker is the preferred agent in patients with chronic obstructive pulmonary disease. 07/05/2020 · Metabolic alkalosis is a type of alkalosis that occurs when your blood becomes overly alkaline. It's most often caused by excessive vomiting. We'll tell you how it's treated. 120 to 360 mg PO once daily. Clinical practice guidelines recommend a nondihydropyridine calcium channel blocker to slow the ventricular heart rate in patients with paroxysmal, persistent, or permanent atrial fibrillation. A nondihydropyridine calcium channel blocker is the preferred agent in patients with chronic obstructive pulmonary disease. Hyperkalemia is an elevated level of potassium (K +) in the blood. Normal potassium levels are between 3.5 and 5.0 mmol/L (3.5 and 5.0 mEq/L) with levels above 5.5 mmol/L defined as hyperkalemia. Typically hyperkalemia does not cause symptoms. Occasionally when severe it can cause palpitations, muscle pain, muscle weakness, or numbness. ... Dual blockade of renin-angiotensin system increases risks of hypotension, hyperkalemia, and renal impairment. captopril. losartan, captopril. Either increases toxicity of the other by pharmacodynamic synergism. Avoid or Use Alternate Drug. Dual blockade of renin-angiotensin system increases risks of hypotension, hyperkalemia, and renal impairment.

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