

دانشگاه علوم پزشکی و خدمات بهداشتی،
درمانی کرمانشاه
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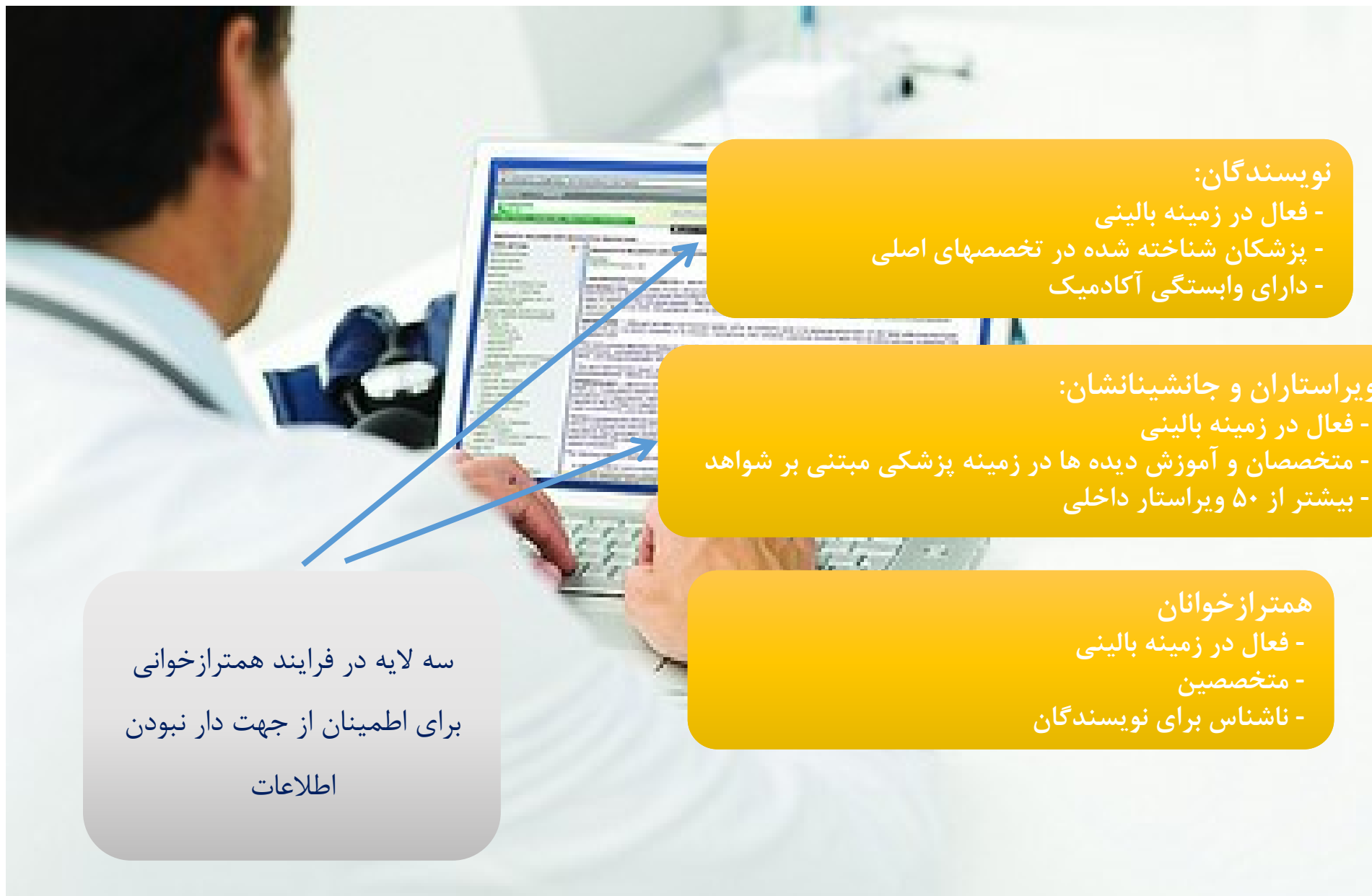
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References

21+

Specialties

هیأت تحریریه



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فرایند جستجو

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Cystic fibrosis: Treatment of acute pulmonary exacerbations

... **Cystic fibrosis** (CF) is a multisystem disorder caused by mutations in the **cystic fibrosis** transmembrane conductance regulator (CFTR) gene, located on chromosome 7 . Pulmonary disease remains the leading ...

- Incidence and consequences
- Definition
- Summary and recommendations

Cystic fibrosis: Hepatobiliary disease

...unresponsive to intensive nutritional support and treatment for **cystic fibrosis**-related diabetes, if present. Given the association of **cystic fibrosis**-related diabetes (CFRD) and advanced CFLD combined, liver-pancreas ...

- Sclerosing cholangitis in cystic fibrosis
- Summary and recommendations

Cystic fibrosis: Clinical manifestations and diagnosis

...with CRMS have been published in Europe and in the United States . **Cystic fibrosis** (CF) is caused by mutations in the **cystic fibrosis** transmembrane conductance regulator (CFTR) protein, a complex chloride ...

- Diagnosis
- Overview of clinical features
- Summary and recommendations
- Algorithm for the diagnosis of cystic fibrosis (Algorithms)

Cystic fibrosis: Overview of gastrointestinal disease

... **Cystic fibrosis** (CF) generally is thought of as a lung disease since much of the associated morbidity and mortality is related to pulmonary complications. A discussion of the pulmonary manifestations ...

- Cystic fibrosis-related liver disease (CFLD)
- Cystic fibrosis-related diabetes (CFRD)
- Summary and recommendations

Topic Outline

Show Graphics (5)

SUMMARY & RECOMMENDATIONS

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PULMONARY EXACERBATIONS IN CYSTIC FIBROSIS

- Definition
- Severity grading
- Incidence and consequences

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- Bacteria
- Noninfectious causes

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- Glucocorticoids
- Respiratory support
- Intensive care unit treatment

TREATMENT: ANTIVIRALS

TREATMENT: ANTIBIOTICS

- Rationale
- Antibiotic selection
 - Sputum cultures
 - Antibiotic susceptibility testing
 - General strategies
 - Patient-specific considerations
 - Route of administration

Topic Outline

Cystic fibrosis: Treatment of acute pulmonary exacerbations

Author: [Richard H Simon, MD](#)
Section Editor: [George B Mallory, MD](#)
Deputy Editor: [Alison G Hoppin, MD](#)

Contributor Disclosures

All topics are updated as new evidence becomes available and our [peer review process](#) is complete.

Literature review current through: Dec 2017. | **This topic last updated:** Dec 12, 2017.

INTRODUCTION — Cystic fibrosis (CF) is a multisystem disorder caused by mutations in the cystic fibrosis transmembrane conductance regulator (CFTR) gene, located on chromosome 7 [1]. (See "[Cystic fibrosis: Genetics and pathogenesis](#)".)

Pulmonary disease remains the leading cause of morbidity and mortality in patients with CF [2,3]. One of the major drivers of CF lung disease is infection [4,5]. Treatment of pulmonary exacerbations in CF is multifaceted, involving antibiotics, chest physiotherapy, inhaled medications to promote secretion clearance, and antiinflammatory agents. Improved treatment of lung disease, in conjunction with improved nutrition, are likely responsible for the increased survival that has occurred in patients with CF (figure).

The treatment of acute pulmonary exacerbations in CF will be reviewed here. Treatment of chronic pulmonary infection and other aspects of pulmonary disease in CF are discussed in separate topic reviews:

- (See "[Cystic fibrosis: Antibiotic therapy for chronic pulmonary infection](#)".)
- (See "[Cystic fibrosis: Overview of the treatment of lung disease](#)".)
- (See "[Cystic fibrosis: Clinical manifestations of pulmonary disease](#)".)
- (See "[Cystic fibrosis: Investigational therapies](#)".)

...s in other organ systems are also discussed separately. (See "[Cystic fibrosis: Clinical manifestations and complications](#)", "[Cystic fibrosis: Nutritional issues](#)" and "[Cystic fibrosis: Assessment and management of pancreatic insufficiency](#)" and "[Cystic fibrosis: Hepatobiliary disease](#)".)

...nctuated by acute episodes of worsening pulmonary status that are referred to as "pulmonary exacerbations" [7,8].

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پیشنهادات رتبه بندی شده بر اساس قدرت شواهد

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cystic fibrosis

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Cystic fibrosis: Treatment of acute pulmonary exacerbations

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 - P. aeruginosa and S. aureus
 - B. cepacia

SUMMARY AND RECOMMENDATIONS

- Cystic fibrosis (CF) lung disease is characterized by persistent bacterial infection in most age groups and are associated with accelerated loss of pulmonary function (See ['Pathogens'](#).)
- The clinical course of CF is frequently complicated by acute pulmonary exacerbations with antibiotics rather than nonantimicrobial treatment alone (See ['Severity grading'](#).) The choice of antibiotic treatment is based upon the sensitivities of the infecting bacteria, each bacterial isolate that is cultured from respiratory secretions, and two antibiotic classes: aminoglycosides and beta-lactams. (See ['Antibiotic selection'](#).)
- We typically treat *P. aeruginosa* with [piperacillin-tazobactam](#), [ceftazidime](#), or [cefepime](#), plus one of the following: [tobramycin](#), [amikacin](#), or a fluoroquinolone (eg, [ciprofloxacin](#)), guided in part by antibiotic susceptibility test results. Oral or intravenous ciprofloxacin may replace the aminoglycoside, particularly if the *Pseudomonas* is sensitive to it.
- When methicillin-sensitive *S. aureus* (MSSA) accompanies the *P. aeruginosa*, treatment options are [piperacillin-tazobactam](#), [cefepime](#), imipenem-cilastatin, [meropenem](#), or [ticarcillin-clavulanate](#) **plus** one of the following: [tobramycin](#) or [amikacin](#).
- When methicillin-resistant *S. aureus* (MRSA) accompanies the *P. aeruginosa*, we treat with [vancomycin](#) or [linezolid](#) **plus** the same antibiotic combination as for *P. aeruginosa*.

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REFERENCE

1. Rommens JM, Iannuzzi MC, Kerem B, et al. Identification of the cystic fibrosis gene: chromosome walking and jumping. *Science* 1989; 245:1059.

Topic Feedback

بر اساس شواهدی که وجود دارد و بر اساس تجربیات متخصصین، پیشنهاداتی داده میشود که درجه اعتبار آنها رتبه بندی می شود

- The pharmacokinetics of many antibiotics differs in patients with CF compared with normal individuals. Patients with CF generally require larger and/or more frequent dosing for penicillins, cephalosporins, sulfonamides, and fluoroquinolones. (See '[Antibiotic dosing](#)' above.)
- For aminoglycosides, starting doses should be larger than those recommended for individuals without CF, but dosing must be adjusted based on pharmacokinetic analysis of serum levels because of considerable interindividual variation in clearance rates. For CF patients with normal renal function, we suggest once daily dosing ("consolidated dosing") rather than conventional dosing and monitoring, with adjustments of dose and timing based on monitoring of drug levels (**Grade 2B**). Once daily dosing has comparable efficacy with conventional dosing and monitoring but has advantages of possibly reducing the risk of nephrotoxicity and simplifying administration and monitoring. (See '[Aminoglycosides](#)' above.)



Evidence-based (مبنتی بر شواهد)

GRADE

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Grade 1A recommendation

A Grade 1A recommendation is a strong recommendation, and applies to most patients in most circumstances without reservation. Clinicians should follow a strong recommendation unless a clear and compelling rationale for an alternative approach is present.

Explanation:

A Grade 1 recommendation is a strong recommendation. It means that we believe that if you follow the recommendation, you will be doing more good than harm for most, if not all of your patients.

Grade A means that the best estimates of the critical benefits and risks come from consistent data from well-performed, randomized, controlled trials or overwhelming data of some other form (eg, well-executed observational studies with very large treatment effects). Further research is unlikely to have an impact on our confidence in the estimates of benefit and risk.

Recommendation grades

1. Strong recommendation: Benefits clearly outweigh the risks and burdens (or vice versa) for most, if not all, patients
2. Weak recommendation: Benefits and risks closely balanced and/or uncertain

Evidence grades

- A. High-quality evidence: Consistent evidence from randomized trials, or overwhelming evidence of some other form
- B. Moderate-quality evidence: Evidence from randomized trials with important limitations, or very strong evidence of some other form
- C. Low-quality evidence: Evidence from observational studies, unsystematic clinical observations, or from randomized trials with serious flaws

For a complete description of our use of the GRADE system, please see the UpToDate editorial policy which can be found at www.uptodate.com/home/editorial-policy.

Drug Information (اطلاعات دارویی)

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- We recommend the chronic use of [redacted] for patients 6 years and older who have clinical evidence of airway inflammation such as chronic cough or any reduction in forced expiratory volume at one minute (FEV1), regardless of the patient's P. aeruginosa infection status ([Grade 1B](#)). To avoid induction of antibiotic resistance, azithromycin should not be given to patients infected with nontuberculous mycobacteria. (See '[Chronic oral antibiotics](#)' above and "[Cystic fibrosis: Overview of the treatment of lung disease](#)", section on '[Macrolide antibiotics](#)'.)
- For patients older than [redacted] with persistent P. aeruginosa infection and moderate or severe lung disease, we recommend chronic treatment with inhaled [redacted] ([Grade 1A](#)). We also suggest this treatment for patients with mild lung disease and persistent P. aeruginosa infection ([Grade 2B](#)). Inhaled [aztreonam](#) lysine is a reasonable alternative. Either inhaled tobramycin or aztreonam lysine are given for one month, on alternate months. (See '[Inhaled antibiotics](#)' above.)



Cystic fibrosis: Treatment of acute pulmonary exacerbations

Topic Outline

SUMMARY & RECOMMENDATIONS

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PULMONARY EXACERBATIONS IN CYSTIC FIBROSIS

- Definition
- Severity grading
- Incidence and consequences

PATHOGENESIS

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 - Antibiotic susceptibility testing
 - General strategies
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 - Route of administration
 - Antibiotics for specific bacteria
 - P. aeruginosa and S. aureus
 - B. cepacia

SUMMARY AND RECOMMENDATIONS

- Cystic fibrosis (CF) lung disease is characterized by persistent bacterial infection. *Staphylococcus aureus* and *Pseudomonas aeruginosa* are the most prevalent pathogens in most age groups and are associated with accelerated loss of pulmonary function (figure 2). (See "Cystic fibrosis: Antibiotic therapy for chronic pulmonary infection", section on 'Pathogens'.)
- The clinical course of CF is frequently complicated by acute pulmonary exacerbations, superimposed on a gradual decline in pulmonary function. We recommend treating exacerbations with antibiotics rather than nonantimicrobial treatment alone (Grade 1C). The antibiotics are given either orally or intravenously, depending on the severity of the exacerbation, and selected based upon the sensitivities of the infecting bacteria (table 2) (see 'Rationale' above). Common practice is to select at least one antibiotic to cover each bacterial isolate that is cultured from respiratory secretions, and two antibiotics for *P. aeruginosa* infections, if possible. (See 'Antibiotic selection' above.)
 - We treat *P. aeruginosa* with piperacillin-tazobactam, ceftazidime, imipenem-cilastatin, meropenem (or ticarcillin-clavulanate, where available) plus one of the following: amikacin, or a fluoroquinolone (eg, ciprofloxacin), guided in part by antibiotic susceptibility test results. Oral or intravenous ciprofloxacin may replace the aminoglycoside, particularly if the *Pseudomonas* is sensitive to it.
 - When methicillin-sensitive *S. aureus* (MSSA) accompanies the *P. aeruginosa*, treatment options are piperacillin-tazobactam, cefepime, imipenem-cilastatin, meropenem, or ticarcillin-clavulanate plus one of the following: tobramycin or amikacin.
 - When methicillin-resistant *S. aureus* (MRSA) accompanies the *P. aeruginosa*, we treat with vancomycin or linezolid plus the same antibiotic combination as for *P. aeruginosa* alone (three antibiotics total).
- The pharmacokinetics of many antibiotics differs in patients with CF compared with normal individuals. Patients with CF generally require larger and/or more frequent dosing for penicillins, cephalosporins, sulfonamides, and fluoroquinolones. (See 'Antibiotic dosing' above.)
- For aminoglycosides, starting doses should be larger than those recommended for individuals without CF, but dosing must be adjusted based on pharmacokinetic analysis of serum levels because of considerable interindividual variation in clearance rates. For CF patients with normal renal function, we suggest once daily dosing ("consolidated dosing") rather than conventional dosing and monitoring, with adjustments of dose and timing based on monitoring of drug levels (Grade 2B). Once daily dosing has comparable efficacy with conventional dosing and monitoring but has advantages of possibly reducing the risk of nephrotoxicity and simplifying administration and monitoring. (See 'Aminoglycosides' above.)
- Antibiotic treatment is typically continued until the signs and symptoms that defined the pulmonary exacerbation are largely resolved. In practice, this usually entails treatment for a minimum of 10 days to as long as three weeks and occasionally more. (See 'Duration of treatment' above.)

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REFERENCES

1. Rommens JM, Iannuzzi MC, Kerem B, et al. Identification of the cystic fibrosis gene: chromosome walking and jumping. *Science* 1989; 245:1059.

اطلاعات بیش از ۵۱۰۰ دارو از طریق Lexicomp

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cystic fibrosis

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Tobramycin (systemic): Drug information

cystic fibrosis Find Print

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- Pharmacologic Category
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- Dosing: Geriatric
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- Contraindications
- Warnings/Precautions
- Metabolism/Transport Effects

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(For additional information [see "Tobramycin \(systemic\): Patient drug information."](#) and [see "Tobramycin \(systemic\): Pediatric drug information."](#))

For abbreviations and symbols that may be used in Lexicomp ([show table](#))

ALERT: US Boxed Warning

Ototoxicity:

Neurotoxicity, manifested as both auditory and vestibular ototoxicity, can occur. The auditory changes are irreversible, are usually bilateral, and may be partial or total. Eighth nerve impairment and nephrotoxicity may develop, primarily in patients having preexisting renal damage and in those with healthy renal function to whom aminoglycosides are administered for longer periods or in higher doses than those recommended. Other manifestations of neurotoxicity may include numbness, skin tingling, muscle twitching, and convulsions. The risk of aminoglycoside-induced hearing loss increases with the degree of exposure to either high peak or high trough serum concentrations. Patients who develop cochlear damage may not have symptoms during therapy to warn them of eighth-nerve toxicity, and partial or total irreversible bilateral deafness may continue to develop after the drug has been discontinued. Keep patients treated with tobramycin injection and other aminoglycosides under close clinical observation because these drugs have an inherent potential for causing ototoxicity.

Nephrotoxicity:

Rarely, nephrotoxicity may not become apparent until the first few days after therapy is initiated. Nephrotoxicity is reversible. Keep patients treated with tobramycin injection and other aminoglycosides under close clinical observation for causing nephrotoxicity.

Monitoring:

Closely monitor renal and eighth nerve function in patients with known or suspected renal impairment and also in those whose renal function is initially normal but who develop signs of renal dysfunction during therapy. Periodically monitor peak and trough serum concentrations of aminoglycosides during therapy to assure adequate levels and to avoid potentially toxic levels. Prolonged serum concentrations above 12 mcg/mL should be avoided. Rising trough levels (above 2 mcg/mL) may indicate tissue accumulation. Such

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تداخلات دارویی

Topic Feedback

cystic fibrosis



Tobramycin (systemic): Drug information

cystic fibrosis Find Print

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- Pregnancy Risk Factor
- Pregnancy Implications
- Breast-Feeding Considerations
- Dietary Considerations
- Monitoring Parameters
- Reference Range
- Mechanism of Action

Other warnings/precautions:

- Long-term use: Systemic therapy is not intended for long-term therapy due to toxic hazards associated with extended administration.

Metabolism/Transport Effects None known.

Drug Interactions

(For additional information, see the following interactions.)

- AbobotulinumtoxinA: Aminoglycosides may enhance the neuromuscular-blocking effect of AbobotulinumtoxinA. *Risk C: Monitor therapy*
- Amphotericin B: May enhance the nephrotoxic effect of Aminoglycosides. *Risk C: Monitor therapy*
- Arbekacin: May enhance the nephrotoxic effect of Aminoglycosides. Arbekacin may enhance the ototoxic effect of Aminoglycosides. *Risk C: Monitor therapy*
- Ataluren: May enhance the adverse/toxic effect of Aminoglycosides. Specifically, an increased risk of nephrotoxicity may occur with the concomitant use of ataluren and aminoglycosides. *Risk X: Avoid combination*
- BCG (Intravesical): Antibiotics may diminish the therapeutic effect of BCG (Intravesical). *Risk X: Avoid combination*
- BCG Vaccine (Immunization): Antibiotics may diminish the therapeutic effect of BCG Vaccine (Immunization). *Risk C: Monitor therapy*
- Bisphosphonate Derivatives: Aminoglycosides may enhance the hypocalcemic effect of Bisphosphonate Derivatives. *Risk C: Monitor therapy*
- Capreomycin: May enhance the neuromuscular-blocking effect of Aminoglycosides. *Risk C: Monitor therapy*
- CARBOplatin: Aminoglycosides may enhance the ototoxic effect of CARBOplatin. Especially with higher doses of carboplatin. *Risk C: Monitor therapy*
- Cefazedone: May enhance the nephrotoxic effect of Aminoglycosides. *Risk C: Monitor therapy*
- Cephalosporins (2nd Generation): May enhance the nephrotoxic effect of Aminoglycosides. *Risk C: Monitor therapy*
- Cephalosporins (3rd Generation): May enhance the nephrotoxic effect of Aminoglycosides. *Risk C: Monitor therapy*
- Cephalosporins (4th Generation): May enhance the nephrotoxic effect of Aminoglycosides. *Risk C: Monitor therapy*
- Cephalothin: May enhance the nephrotoxic effect of Aminoglycosides. *Risk C: Monitor therapy*
- Cephradine: May enhance the nephrotoxic effect of Aminoglycosides. *Risk C: Monitor therapy*
- Cholera Vaccine: Antibiotics may diminish the therapeutic effect of Cholera Vaccine. *Risk X: Avoid combination*

Lexicomp® Drug Interactions

Add items to your list by searching below.

ITEM LIST

Clear List

Analyze

– [Tobramycin \(Systemic\)](#)

– [Typhoid Vaccine](#)

– [Vitamin A](#)

Display complete list of interactions for an individual item by clicking item name.

NOTE: This tool does not address chemical compatibility related to I.V. drug preparation or administration.

X Avoid combination	C Monitor therapy	A No known interaction
D Consider therapy modification	B No action needed	<i>More about Risk Ratings</i>



1 Result

D Typhoid Vaccine
Tobramycin (Systemic) (Antibiotics)

DISCLAIMER: Readers are advised that decisions regarding drug therapy must be based on current product information, and changing medical practices.

X	Avoid Combination Data demonstrate that the specified agents may interact with each other in a clinically significant manner. The risks associated with concomitant use of these agents usually outweigh the benefits. These agents are generally considered contraindicated.
D	Consider Therapy Modification Data demonstrate that the two medications may interact with each other in a clinically significant manner. A patient-specific assessment must be conducted to determine whether the benefits of concomitant therapy outweigh the risks. Specific actions must be taken in order to realize the benefits and/or minimize the toxicity resulting from concomitant use of the agents. These actions may include aggressive monitoring, empiric dosage changes, choosing alternative agents.
C	Monitor Therapy Data demonstrate that the specified agents may interact with each other in a clinically significant manner. The benefits of concomitant use of these two medications usually outweigh the risks. An appropriate monitoring plan should be implemented to identify potential negative effects. Dosage adjustments of one or both agents may be needed in a minority of patients.
B	No Action Needed Data demonstrate that the specified agents may interact with each other, but there is little to no evidence of clinical concern resulting from their concomitant use.
A	No Known Interaction Data have not demonstrated either pharmacodynamic or pharmacokinetic interactions between the specified agents

Results by Item Print

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Lexicomp® Drug Interactions

Add items to your list by searching below.

Enter item name

ITEM LIST

Clear List

Analyze

– [Tobramycin \(Systemic\)](#)

– [Typhoid Vaccine](#)

– [Vitamin A](#)

Display complete list of interactions for an individual item by clicking item name.

NOTE: This tool does not address chemical compatibility related to I.V. drug preparation or administration.

Title Typhoid Vaccine / Antibiotics

Print

Dependencies

- **Route** (oral): Only the live typhoid vaccine (oral product) is subject to this potential interaction.

Risk Rating D: Consider therapy modification

Summary Antibiotics may diminish the therapeutic effect of Typhoid Vaccine. Only the live attenuated Ty21a strain is affected. **Severity** Major **Reliability Rating** Fair

Patient Management Vaccination with live attenuated typhoid vaccine (Ty21a) should be avoided in patients being treated with systemic antibacterial agents. Use of this vaccine should be postponed until at least 3 days after cessation of antibacterial agents, and when possible, antibacterials should not be started within 3 days of the last vaccine dose.

Antibiotics Interacting Members Amdinocillin, Amikacin, Amoxicillin, Ampicillin, Arbekacin, Azithromycin (Systemic), Aztreonam (Systemic), Bacampicillin, Bedaquiline, Cefaclor, Cefadroxil, Cefazedone, CeFAZolin, Cefcapene, Cefdinir, Cefepime, Cefixime, Cefminox, Cefoperazone, Cefotaxime, CefoTETan, Cefotiam, CefOXitin, Cefpodoxime, Cefprozil, Ceftaroline Fosamil, CeFTAZidime, Ceftibuten, Ceftizoxime, Ceftolozane, CeFTRIAXone, Cefuroxime, Cephalexin, Cephalothin, Cephradine, Chloramphenicol (Ophthalmic), Chloramphenicol (Otic), Chloramphenicol (Systemic), Ciprofloxacin (Systemic), Clarithromycin, Clindamycin (Systemic), Clofazimine, Cloxacillin, Colistimethate, CycloSERINE, Dalbavancin, Dapsone (Systemic), Delafloxacin, Delamanid, Demeclocycline, Dicloxacillin, Doripenem, Doxycycline, Ertapenem, Erythromycin (Systemic), Ethambutol, Ethionamide, Flomoxef, Flucloxacillin, Fosfomycin, Fusidic Acid (Systemic), Gemifloxacin, Gentamicin (Systemic), Imipenem, Isepamicin, Isoniazid, Ivermectin (Systemic), Kanamycin, LevoFLOXacin (Oral Inhalation), LevoFLOXacin (Systemic), Lincomycin, Linezolid, Lomefloxacin, Lymecycline, Meropenem, Methenamine, MetroNIDAZOLE (Systemic), Minocycline, Moxifloxacin (Systemic), Nafcillin, Nalidixic Acid, Nifuroxazide, Nitrofurantoin, Norfloxacin, Ofloxacin (Systemic), Oritavancin, Oxacillin, Oxytetracycline, Pefloxacin, Penicillin G (Parenteral/Aqueous), Penicillin G Benzathine, Penicillin G Procaine, Penicillin V Benzathine, Penicillin V Potassium, Pentamidine (Oral Inhalation), Pentamidine (Systemic), Pipemidic Acid, Piperacillin, Prothionamide, Pyrazinamide, Rifabutin, RifAMPin, Rifapentine, Roxithromycin, Secnidazole, Sparfloxacin, Spiramycin, Streptomycin, SulfADIAZINE, Sulfadoxine, Sulfamethoxazole, SulfiSOXAZOLE, Tedizolid, Teicoplanin, Telavancin, Telithromycin, Temocillin, Tetracycline, Ticarcillin, Tinidazole, Tobramycin (Systemic), Trimethoprim, Vancomycin

Exceptions Acetic Acid (Otic), Acetic Acid (Topical), Aluminum Acetate, Azithromycin (Ophthalmic), Aztreonam (Oral Inhalation), Bacitracin (Ophthalmic), Bacitracin (Systemic), Bacitracin (Topical), Benzoin, Capreomycin, Chlortetracycline, Ciprofloxacin (Ophthalmic), Clindamycin (Topical), Dapsone (Topical), Dibrompropamide (Ophthalmic), Dibrompropamide (Topical), Erythromycin (Ophthalmic), Erythromycin (Topical), Fidaxomicin, Framycetin, Fusidic Acid (Ophthalmic), Fusidic Acid (Topical), Gatifloxacin, Gentamicin (Ophthalmic), Gentamicin (Topical), Gentian Violet, Hexachlorophene, Mafenide, MetroNIDAZOLE (Topical), Mupirocin, Neomycin, Nitrofurazone, Oxychlorosene, Ozenoxacin, Polymyxin B, Povidone-Iodine (Topical), RifAXIMin, Silver Nitrate, Silver Sulfadiazine, Sulfacetamide (Ophthalmic), Sulfacetamide (Topical), Taurolidine, Tobramycin (Ophthalmic)

Discussion The prescribing information for the live attenuated typhoid vaccine (Ty21a) warns that it should not be administered to individuals who are being treated with antibacterial agents.¹ A report from the Advisory Committee on Immunization Practices (ACIP) advises that use of this vaccine should be postponed until at least 3 days after cessation of antibacterial agents, and when possible, antibacterials should not be started within 3 days of the last vaccine dose.² These recommendations are consistent with the concern regarding the potential for some antibacterial agents to interfere with the replication of and resultant immune response to the live bacterial strain used in the live vaccine.^{1,3}

Lexicomp® Drug Interactions

Add items to your list by searching below.

ITEM LIST

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Analyze

[Tobramycin \(Systemic\)](#)

[Typhoid Vaccine](#)

[Vitamin A](#)

Display complete list of interactions for an individual item by clicking item name.

X Avoid combination	C Monitor therapy	A No known interaction
D Consider therapy modification	B No action needed	<i>More about Risk Ratings</i> ▼

39 Results

Print

X	Tobramycin (Systemic) (Aminoglycosides) Ataluren
X	Tobramycin (Systemic) (Antibiotics) BCG (Intravesical)
X	Tobramycin (Systemic) (Antibiotics) Cholera Vaccine
X	Tobramycin (Systemic) (Aminoglycosides) Foscarnet
X	Tobramycin (Systemic) (Aminoglycosides) Mannitol (Systemic)
X	Tobramycin (Systemic) (Aminoglycosides) Mecamylamine
X	Tobramycin (Systemic) (Aminoglycosides) Methoxyflurane
D	Tobramycin (Systemic) (Aminoglycosides) Colistimethate
D	Tobramycin (Systemic) (Aminoglycosides) Penicillins
D	Tobramycin (Systemic) (Antibiotics) Sodium Picosulfate
D	Tobramycin (Systemic) (Antibiotics) Typhoid Vaccine

NOTE: This tool does not address chemical compatibility related to I.V. drug preparation or administration.

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Cystic fibrosis: Treatment of acute pulmonary exacerbations

Topic Outline

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 - Definition
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 - Continuation of the chronic treatment regimen
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 - Rationale
 - Antibiotic selection
 - Sputum cultures
 - Antibiotic susceptibility testing
 - General strategies
 - Patient-specific considerations

Cystic fibrosis: Treatment of acute pulmonary exacerbations

Contributor Disclosures

All topics are updated as new evidence becomes available and clinical practice changes. **Literature review current through: Jan 2018. | This topic last updated: Jan 2018.**

INTRODUCTION — Cystic fibrosis (CF) is a multisystem disorder caused by mutations in the cystic fibrosis transmembrane conductance regulator (CFTR) gene, located on chromosome 7 [1]. (See "[Cystic fibrosis: Genetics and pathogenesis](#)".)

Pulmonary disease remains the leading cause of morbidity and mortality in CF. The treatment of acute pulmonary exacerbations in CF is multifaceted, involving antibiotic therapy, inhaled corticosteroids, and respiratory support. Improved treatment of lung disease, in conjunction with improved management of other organ systems, has led to a significant increase in life expectancy in CF.

The treatment of acute pulmonary exacerbations in CF will be reviewed in separate topic reviews:

- (See "[Cystic fibrosis: Antibiotic therapy for chronic pulmonary disease](#)".)
- (See "[Cystic fibrosis: Management of acute pulmonary exacerbations](#)".)

PULMONARY EXACERBATIONS IN CYSTIC FIBROSIS

Definition — The clinical course of most patients with CF is punctuated by acute pulmonary exacerbations (APEs), which are defined as "pulmonary exacerbations" [7,8].

Contributor disclosure

Topic Feedback

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Pulmonary disease remains the leading cause of morbidity and mortality in patients with CF [2,3]. One of the major drivers of CF lung disease is infection [4,5]. Treatment of acute pulmonary exacerbations in CF is multifaceted, involving antibiotics, chest physiotherapy, inhaled medications to promote secretion clearance, and antiinflammatory agents. Improved treatment of lung disease, in conjunction with improved nutrition, are likely responsible for the increased survival that has occurred in patients with CF ([figure 1](#)) [4,6].

The treatment of acute pulmonary exacerbations in CF will be reviewed here. Treatment of chronic pulmonary infection and other aspects of pulmonary disease in CF are discussed in separate topic reviews:

- (See "[Cystic fibrosis: Antibiotic therapy for chronic pulmonary infection](#)".)
- (See "[Cystic fibrosis: Overview of the treatment of lung disease](#)".)
- (See "[Cystic fibrosis: Clinical manifestations of pulmonary disease](#)".)
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Topic Feedback

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Nat Rev Dis Primers. 2015 May 14;1:15010. doi: 10.1038/nrdp.2015.10.

Cystic fibrosis.

Ratjen F¹, Bell SC², Rowe SM³, Goss CH⁴, Quittner AL⁵, Bush A⁶.

Author information

Abstract

Cystic fibrosis is an autosomal recessive, monogenetic disorder caused by mutations in the cystic fibrosis transmembrane conductance regulator (CFTR) gene. The gene defect was first described 25 years ago and much progress has been made since then in our understanding of how CFTR mutations cause disease and how this can be addressed therapeutically. CFTR is a transmembrane protein that transports ions across the surface of epithelial cells. CFTR dysfunction affects many organs; however, lung disease is responsible for the vast majority of morbidity and mortality in patients with cystic fibrosis. Prenatal diagnostics, newborn screening and new treatment algorithms are changing the incidence and the prevalence of the disease. Until recently, the standard of care in cystic fibrosis treatment focused on preventing and treating complications of the disease; now, novel treatment strategies directly targeting the ion channel abnormality are becoming available and it will be important to evaluate how these treatments affect disease progression and the quality of life of patients. In this Primer, we summarize the current knowledge, and provide an outlook on how cystic fibrosis clinical care and research will be affected by new knowledge and therapeutic options in the near future. For an illustrated summary of this Primer, visit: <http://go.nature.com/4VrefN>.

PMID: 27189798 DOI: [10.1038/nrdp.2015.10](https://doi.org/10.1038/nrdp.2015.10)

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Topic Feedback

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Cystic fibrosis: Treatment of acute pulmonary exacerbations

Author: Richard H Simon, MD
 Section Editor: George B Mallory, MD
 Deputy Editor: Alison G Hoppin, MD

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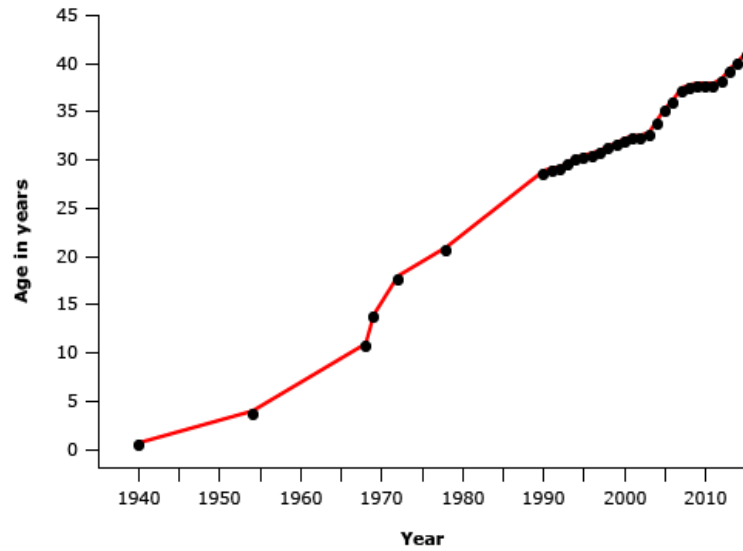


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Median predicted survival of patients with cystic fibrosis



Data from:

1. 1940 - 1978. Davis PB, Drumm M, Konstan MW. Cystic fibrosis. *Am J Respir Crit Care Med* 1996; 154:1229.
2. 1990 - 2015. Cystic Fibrosis Foundation Patient Registry, 2015 Annual Data Report, Bethesda, MD. (Each point represents a rolling 5 year average).

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Survival_of_CF_patients [Compatibility Mode] - PowerPoint (Product Acti... ? [Icons] - [Icons] X

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Median predicted survival of patients with cystic fibrosis

Year	Age in years
1940	0
1950	4
1960	10
1970	17
1980	21
1990	28
2000	31
2010	37
2015	40

Data from:

1. 1940 - 1978. Davis PB, Drumm M, Konstan MW. Cystic fibrosis. *Am J Respir Crit Care Med* 1996; 154:1229.
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Cystic fibrosis: Treatment of acute pulmonary exacerbations

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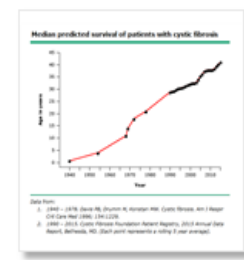
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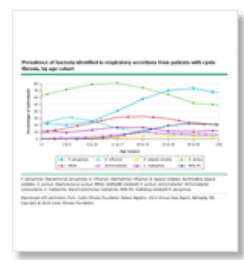
Graphics for: Cystic fibrosis: Treatment of acute pulmonary exacerbations



Survival of CF patients

Organism	% with positive respiratory cultures
Pseudomonas aeruginosa	74.1
Methicillin-resistant S. aureus (MRSA)	63.2
Respiratory moraxella	47.3
Staphylococcus aureus	42.1
Stenotrophomonas maltophilia	33.8
Haemophilus influenzae	22.2
A. baumannii	13.9
S. pneumoniae	11.8
Other	9.1
Acinetobacter baumannii	8.2
Legionella pneumophila	6.7
Stenotrophomonas maltophilia	5.6

CF micro-organisms



CF colonization by age

Cystic fibrosis antibiotics

Maintenance dose aminoglycoside

(See "[Cystic fibrosis: Investigational therapies](#)".)

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PULMONARY EXACERBATIONS IN CYSTIC FIBROSIS

Definition — The clinical course of most patients with CF is punctuated by acute episodes of worsening pulmonary status that are referred to as "pulmonary exacerbations" [7,8]. Although the CF field has not settled on a single set of criteria to define a pulmonary exacerbation [9-11], symptoms that are commonly present include:

- New or increased cough
- New or increased sputum production or chest congestion

regulator (CFTR) gene, located on chromosome 7. The disease is infection [4,5]. Treatment of acute pulmonary exacerbations includes antibiotics, mucolytics, and antiinflammatory agents. Exacerbations are more frequent in patients with CF (figure 1) [4,6]. The clinical course of pulmonary disease in CF are discussed

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می توانید این اطلاعات را برای بیمار ایمیل کنید یا پرینت بگیرید

Patient education: Cystic fibrosis (The Basics)

[Written by the doctors and editors at UpToDate](#)

What is cystic fibrosis? — Cystic fibrosis is a disease that some children are born with. It causes thick mucus and other fluids to build up and clog different parts of the body, including the lungs, pancreas, liver, and intestine ([figure 1](#)).

The thick mucus in the lungs causes people with cystic fibrosis to get frequent lung infections. Over time, these infections damage the lungs. The thick fluids in the pancreas and liver keep the intestine from absorbing certain nutrients from food. This affects a child's growth and causes abnormal bowel movements.

Cystic fibrosis is caused by an abnormal gene. To get the disease, people need to get the abnormal gene from both their mother and father. If people get the abnormal gene from only 1 parent, they will not have cystic fibrosis. But they will have a chance of passing on the abnormal gene to their children.

Cystic fibrosis is a lifelong condition. As of now, doctors can't cure the disease, but they can use different treatments to help with symptoms.

People with cystic fibrosis don't live as long as people without the disease. But better treatments are helping people with cystic fibrosis live longer. To help manage your child's disease for as long as possible, it's important to work closely with his or her doctor.

What are the symptoms of cystic fibrosis? — People can have different symptoms at different times. Most people start having symptoms as a baby or young child. A few people start having symptoms as teens or adults. A person's symptoms usually get worse over time.

Common symptoms of cystic fibrosis include:

- Not growing or gaining weight normally
- A long-lasting cough – The cough usually brings up mucus (it sounds "wet"). Some people cough up blood.
- Trouble breathing or breathing that sounds like whistling (wheezing)
- Frequent infections of the lungs or sinuses – The sinuses are hollow areas in the bones of the face.
- Skin that tastes salty (for example, you might taste salt when you kiss your child)
- Belly pain, diarrhea, or constipation (trouble having bowel movements)
- Bowel movements that are oily, bad-smelling, and float in the toilet bowl

Topic Outline

- What is cystic fibrosis?
- What are the symptoms of cystic fibrosis?
- Is there a test for cystic fibrosis?
- How is cystic fibrosis treated?
- How can I help my child stay as healthy as possible?
- How can I learn more about cystic fibrosis?
- More on this topic

GRAPHICS [View All](#)

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Patient Education

Patient Education

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INFECTIOUS DISEASES; OBSTETRICS, GYNECOLOGY AND WOMEN'S HEALTH (November 2017)

Elvitegravir-cobicistat use during pregnancy

NEPHROLOGY AND HYPERTENSION (November 2017)

Acetylcysteine does not prevent contrast nephropathy

HEMATOLOGY, ADULT PRIMARY CARE, FAMILY MEDICINE AND GENERAL PRACTICE, GASTROENTEROLOGY AND HEPATOLOGY (November 2017)

Frequency for dosing of oral iron

GASTROENTEROLOGY AND HEPATOLOGY, ADULT PRIMARY CARE, FAMILY MEDICINE

Practice Changing UpDates

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Contributor Disclosures

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: Jan 2018. | This topic last updated: Feb 21, 2018.

INTRODUCTION — This section highlights selected specific new recommendations and/or updates the UpDates focus on changes that may have significant and broad impact on practice, and therefore do not reflect important changes to UpToDate over the past year, are presented chronologically, a

CARDIOVASCULAR MEDICINE (February 2018)

Catheter ablation for atrial fibrillation with heart failure

- For selected patients with symptomatic atrial fibrillation (AF) and heart failure with reduced ejection fraction, catheter ablation of AF rather than continued attempts with antiarrhythmic drug therapy or no antiarrhythmic therapy is preferred.

For many patients with heart failure (HF) and atrial fibrillation (AF), we prefer a rhythm to a rate control strategy for the management of AF. Catheter ablation (CA) is an alternative to medical therapy (eg, antiarrhythmic drugs) or catheter ablation (CA). The strongest evidence supporting the use of CA in many of these patients comes from the CASTLE-AF randomized trial that compared CA with medical therapy (rate or rhythm control) in 363 patients with symptomatic paroxysmal or persistent AF; New York Heart Association class II, III, or IV HF; a left ventricular ejection fraction of ≤ 35 percent; failure or unwillingness to take antiarrhythmic drug therapy; and an implanted cardioverter-defibrillator [1]. CA significantly reduced the primary composite end point of death from any cause or hospitalization for worsening HF. Based on the results of CASTLE-AF, we now suggest CA as an appropriate treatment for selected patients with AF and HF for whom initial attempts at antiarrhythmic therapy were ineffective. (See "The management of atrial fibrillation in patients with heart failure", section on 'Catheter ablation'.)

ONCOLOGY (January 2018)

Adjuvant therapy for cutaneous melanoma

- In most patients with *BRAF* wild type or unknown stage III cutaneous melanoma or stage IV disease who have undergone definitive resection of all sites of disease, we recommend adjuvant nivolumab rather than ipilimumab (Grade 1B). In patients whose tumor contains a *BRAF* V600 mutation, we suggest adjuvant nivolumab rather than targeted therapy (Grade 2C).



Practice Changing Updates
 حاوی اطلاعات مهم و ضروری است که
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Conventional (gravimetric, imperial, US) unit to SI unit conversions: Immunology lab values

SI unit to conventional (gravimetric, imperial, US) unit conversions: Chemistry and endocrine tests

SI unit to conventional (gravimetric, imperial, US) unit conversions: Immunology lab values

ANESTHESIOLOGY CALCULATORS

Clinical Criteria



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