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Continuing Education Activity

Colestipol is an antihyperlipidemic drug approved by the United States Food and Drug Administration (FDA) as an adjunctive therapy to lower elevated low-density lipoprotein cholesterol in patients with primary hypercholesterolemia unresponsive to dietary modifications alone. The drug can be used as monotherapy or in conjunction with a statin, niacin, or ezetimibe. Colestipol is a cholesterol-lowering drug that reduces the risk of coronary artery disease. This drug belongs to the class of bile acid sequestrants and is one of the earliest drugs used for this purpose. However, the current predominant application involves providing supplementary therapy, especially in cases where statins or alternative lipid-lowering agents prove inadequate in sufficiently reducing cholesterol levels.

The drug is adjunctive to dietary modifications and exercise. In addition, colestipol is used off-label to treat cholestatic pruritus and irritable bowel syndrome. This activity emphasizes the indications, mechanism of action, adverse event profile, administration, pharmacokinetics, and monitoring strategies for colestipol. This activity provides healthcare professionals with a scientific foundation for effectively treating primary hypercholesterolemia and related conditions. In addition, this activity assists healthcare providers in decision-making regarding colestipol prescription and optimizing dosage regimens to minimize adverse reactions, thereby promoting desired patient outcomes.

Objectives:

- Identify patients with primary hypercholesterolemia who may benefit from adjunctive therapy with colestipol based on their lipid profile and responsiveness to dietary modifications alone.
- Screen for potential contraindications and assess the suitability of colestipol for patients with cholestatic pruritus and irritable bowel syndrome, recognizing its off-label applications.
- Assess patient responses to colestipol therapy by regularly monitoring lipid profiles and adjusting dosage regimens as needed to achieve target cholesterol levels.
- Coordinate efforts within the interprofessional team to address knowledge gaps and facilitate ongoing colestipol education, contributing to a comprehensive and informed approach to hypercholesterolemia management.

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Indications

Colestipol is an antihyperlipidemic drug approved by the United States Food and Drug Administration (FDA) as an adjunctive therapy to lower elevated low-density lipoprotein cholesterol in patients with primary hypercholesterolemia unresponsive to dietary modifications alone. The drug can be used as monotherapy or in conjunction with a statin, niacin, or ezetimibe. The drug is adjunctive to dietary modifications and exercise.

Colestipol belongs to the class of bile acid sequestrants and is used for various indications. This drug was approved for usage in the United States back in 1977 and is one of the earliest cholesterol-lowering drugs that reduces the risk of coronary artery disease. However, the current predominant application involves providing supplementary therapy, especially in cases where statins or alternative lipid-lowering agents prove inadequate in sufficiently reducing cholesterol levels.

FDA-Approved Indications

Colestipol is FDA-approved as adjunctive therapy to reduce elevated low-density lipoprotein cholesterol (LDL-C) in patients with primary hypercholesterolemia who do not respond to diet modifications only. The drug is used as monotherapy or in combination with a statin, niacin, or ezetimibe. Doses of 4 to 16 g/d day are associated with a 12% to 24% reduction in LDL cholesterol, respectively. Higher doses may be associated with increased gastrointestinal adverse effects.

Combination therapy with simvastatin is associated with a 52% reduction of LDL cholesterol. Dual treatment with niacin demonstrated a 43% reduction in LDL cholesterol levels and reduced angiographic progression of coronary artery atherosclerotic lesions by 16.2% compared to 2.4% in individuals receiving a placebo. The National Lipid Association (NLA) guidelines endorse bile acid sequestrants like colestipol for hypercholesteremia. According to the NLA guidelines, hypercholesterolemia during pregnancy and breastfeeding, particularly in patients with familial hypercholesterolemia, may be treated with bile acid sequestrants.

Off-Label Uses

Colestipol may be used in the treatment of heterozygous familial hypercholesterolemia. Although colestipol is effective, it is poorly tolerated due to gastrointestinal adverse effects. Colestipol may be used in combination with a statin. A trial in children with familial hypercholesterolemia treated with colestipol found a 19.5% reduction in LDL-C levels after 8 weeks of therapy.

Colestipol is used off-label to treat cholestatic pruritus and irritable bowel syndrome. Pruritus associated with cholestasis, particularly in primary sclerosing cholangitis, may be treated with colestipol. Colestipol may be better tolerated compared to therapy with cholestyramine. The American Association for the Study of Liver Diseases endorses bile acid sequestrants such as colestipol for cholestatic pruritus in primary biliary cholangitis. These agents are positively charged

resins that work by binding to negatively charged anions, including bile acids. Although clinical trials proving efficacy are limited, they have a long track record of clinical use. Patients may prefer colestipol and colesevelam, which are available in tablet forms.

A prospective, controlled trial with 92 patients performed by Hagag et al demonstrated the effectiveness of colestipol as an adjunct therapy to methimazole for treating hyperthyroidism. Greater efficacy was seen in severe cases of thyrotoxicosis.

In a study of 141 patients evaluating the role of bile acids in the pathogenesis of irritable bowel syndrome (IBS), treatment with colestipol improved IBS symptoms (IBS severity scoring system 220 ± 109 vs 277 ± 106). As such, colestipol may be used to treat bile acid diarrhea. However, colestipol is often discontinued due to poor tolerability.

Colestipol is successfully used to treat digoxin toxicity. Colestipol interrupts the enterohepatic circulation of digoxin and increases fecal excretion. One case report by Payne et al reported a reduced half-life of 55 hours compared to a predicted half-life of 85.[16]

The clinical trial evaluated colestipol's viability as an oral phosphate binder in hemodialysis patients and reported positive outcomes. The findings suggest the potential for a larger-scale trial to ascertain colestipol's effectiveness as a phosphate binder.

Mechanism of Action

Colestipol is a large basic anion-exchange resin copolymer that is nonabsorbable and insoluble in water. The primary mechanism of action is the formation of insoluble complexes with bile acids in the small intestine that are subsequently excreted in the feces. Bile acids emulsify and solubilize lipids in the intestinal lumen as part of normal digestion. The rate-limiting step in bile acid synthesis involves hydroxylation of the steroid nucleus of cholesterol by cholesterol 7-alpha-hydroxylase. Data show that 95% of bile acids are recycled via enterohepatic circulation to the liver, while 5% are excreted in the feces.

Increased fecal excretion of bile acids reduces the total bile acid pool available for enterohepatic circulation. As a result, upregulation of cholesterol 7-alpha-hydroxylase leads to increased conversion of cholesterol into bile acids, reducing total serum cholesterol levels. Further upregulation of hepatic low-density lipoprotein receptors (LDLR) to replenish intrahepatic cholesterol stores available for bile acid synthesis leads to increased uptake of LDL-C and an overall reduction in plasma LDL cholesterol (LDL-C) levels.

In cases of thyrotoxicosis, enterohepatic circulation of thyroid hormones, including thyroxine (T4) and triiodothyronine (T3), increases. Interaction between positively charged nitrogen atoms of colestipol with negatively charged iodothyronines leads to increased fecal excretion of iodothyronines.

According to a systematic review, colestipol is suggested for moderate to severe cholestatic pruritus. The mechanism of colestipol in cholestatic pruritus is by binding to bile acids within the small intestine, preventing reabsorption and facilitating elimination via feces.

Pharmacokinetics

Absorption: Colestipol demonstrates negligible solubility in water despite its hydrophilic nature and is not hydrolyzed by digestive enzymes. The high molecular weight polymer present in colestipol appears to render it nonabsorbable. The primary effects of bile acid sequestrants like colestipol are restricted to the intestine.

Distribution: In humans, less than 0.17% of a single dose labeled with ^14C-colestipol is excreted in the urine after 60 days of consistent administration at 20 g daily. This low percentage suggests minimal systemic distribution or absorption of colestipol hydrochloride within the body.

Metabolism: The binding of bile acids to colestipol creates an insoluble compound that cannot be reabsorbed and is excreted in the feces.

Elimination: Bile acids ordinarily undergo extensive (>95%) enterohepatic recirculation, are secreted in bile, act as fat-solubilizing compounds in the upper intestine, and are reabsorbed in the distal small bowel. However, colestipol disrupts this cycle, resulting in the excretion of colestipol-bound bile acids in the feces. Colestipol is minimally excreted in the urine and predominantly in feces due to its nonabsorbable nature.

Administration

Available Dosage Forms and Strengths

Colestipol is available to patients in 2 forms—oral tablet or granular form as a powdered suspension. The powdered suspension is also available in a flavored form to improve palatability. However, the flavored suspension is taken cautiously in patients with phenylketonuria, as it contains phenylalanine.

Colestipol is available in various formulations and doses, including granules in a bottle containing 5 g of the medication and flavored granules in packets, each containing 5 g of the substance. Furthermore, the oral form is available in tablets with a dosage of 1 g per tablet.

Adult Dosage

Patients with phenylketonuria must consult their clinician before initiating therapy with a flavored colestipol suspension. Powdered suspensions of colestipol should be mixed with water or other fluids before ingestion to prevent inhalation or esophageal distress. Other medications, such as fat-soluble vitamins, should be ingested 1 hour before or 4 hours after the administration of colestipol.

Colestipol tablets are recommended in 2 to 16 g daily. Therapy should be initiated at a dosage of 2 g/d. Colestipol powdered suspension is recommended in a dosage of 1 to 6 packets per day. One packet contains 5 g of colestipol.

Specific Patient Populations

Hepatic impairment: There is no dosage adjustment information for hepatic impairment for colestipol in the product labeling.

Renal impairment: There is no dosage adjustment information for renal impairment for colestipol in the product labeling.

Pregnancy considerations: Colestipol is safe since it is not systemically absorbed. However, colestipol may induce the malabsorption of fat-soluble vitamins (A, D, E, and K), which is hazardous to the mother and child. Therefore, the benefits of drug therapy should be weighed against the risks before the initiation of treatment.

As per the NLA guidelines, most women diagnosed with hypercholesterolemia are advised against drug therapy during pregnancy. However, those with atherosclerotic cardiovascular disease or homozygous familial hypercholesterolemia may require pharmacotherapy. Bile acid sequestrants are considered reasonable therapeutic options during pregnancy.

Breastfeeding considerations: Colestipol, a nonabsorbable resin, does not reach the maternal bloodstream and consequently does not transfer to the infant through breast milk. Accordingly, colestipol is considered safe during lactation.

Pediatric patients: The adverse event profile of colestipol in patients under 18 has not been established. However, colestipol has been used safely in pediatric patients with familial hypercholesterolemia. According to the NLA guidelines, statins and bile acid sequestrants can be considered for children and adolescents.

Older patients: The gastrointestinal adverse effects of colestipol, such as constipation, may be more pronounced in patients over the age of 60.[25]

Adverse Effects

Adverse Drug Reactions

> Most adverse effects caused by colestipol therapy are related to the gastrointestinal system. Colestipol can cause a wide range of adverse effects, including constipation, bloating, dyspepsia, and nausea.[26]

Constipation is the most common adverse drug reaction and ranges in severity from mild cases to severe cases. The reaction is often a cause of poor compliance and discontinuation of therapy.[27] Patients aged 65 or older and patients using a larger dose of colestipol are more prone to constipation.[25] Constipation may be alleviated with increased fluid and fiber intake, as well as the use of stool softeners. Colestipol may aggravate preexisting hemorrhoids.

The usage of colestipol in powdered suspension form may cause decreased palatability. Flavored suspensions of colestipol are used to improve palatability.

Non-gastrointestinal adverse effects are infrequently reported. These include infrequent occurrences of cardiovascular symptoms such as angina, musculoskeletal symptoms such as joint pains and arthritis, and neurologic symptoms such as migraine headaches.

Colestipol may cause transient elevations in alkaline phosphatase levels, although the clinical significance is unknown.[25]

Colestipol therapy is reported to cause asymptomatic hepatotoxicity with elevated serum transaminases. However, the mechanism by which colestipol causes hepatotoxicity is unknown.[28]

Colestipol may cause hyperchloremic metabolic acidosis.

Drug-Drug Interactions

Colestipol may induce malabsorption of fat-soluble vitamins (vitamins A, D, E, and K). Patients with preexisting deficiencies of these vitamins should consult their clinician before commencing therapy with colestipol. These vitamins should be taken 4 hours after colestipol.[22]

Colestipol can reduce the absorption of beta blockers, fibrates, and ezetimibe.[21]

Colestipol may reduce the absorption of many drugs if they are given concomitantly with colestipol. Anionic drugs such as warfarin, thiazide diuretics, propanolol, levothyroxine, and digitalis should be given 1 hour before or 4 hours after colestipol.[22]

Contraindications

Colestipol should not be used in patients with known hypersensitivity to the drug or its components.

Warning and Precautions

Colestipol should be avoided for use in patients with disorders that cause decreased intestinal motility, in patients with a recent history of abdominal surgery, and patients with a history of recent episodes of intestinal obstruction.[22]

Colestipol is contraindicated in patients undergoing therapy with mycophenolic acid. As colestipol binds to bile acids, it reduces the absorption of mycophenolic acid and may reduce the efficacy.

Colestipol causes elevated triglyceride levels. Lyons et al demonstrated an 8.9% rise in triglyceride levels in patients treated with colestipol.[28] Colestipol should be avoided in patients with triglyceride levels above 400 mg/dL. It may be used in patients with serum levels below 200 mg/dL of triglyceride.[22] Therefore, as a precaution, clinicians should reserve this drug for use in patients with hypercholesterolemia without associated hypertriglyceridemia.[18]

Colestipol is a bile acid sequestrant. As a large anion exchange resin, it may bind numerous intestine molecules. Of note, colestipol may bind to free triiodothyronine (T3), resulting in increased fecal excretion of T3 and reduced enterohepatic circulation.[29] As a result, colestipol therapy may worsen symptoms in patients with preexisting hypothyroidism. Therefore, patients on colestipol therapy should consider consulting their clinician before commencing treatment.

Colestipol should be avoided in patients with complete biliary obstruction.[22]

Chronic use of colestipol may cause an increased risk of bleeding due to hypoprothrombinemia resulting from vitamin K deficiency. However, this condition can be promptly treated with parenteral vitamin K1 and prevented from recurring by administering oral vitamin K1.

Monitoring

Periodic monitoring of serum lipoproteins is recommended in patients taking colestipol to assess treatment response and dosage modifications. As colestipol is known to cause constipation of varying severity and may cause intestinal obstruction, monitoring of constipation at 4- to 6-week intervals is recommended. Worsening constipation or failure to meet therapeutic goals may require combination therapy or replacement with alternate therapy.

Clinicians should regularly monitor patients with preexisting fat-soluble vitamin deficiencies (A, D, E, and K) when on colestipol therapy. Colestipol may impede the absorption of fat-soluble vitamins by acting as a bile acid sequestrant. If any of these vitamins are deficient, signs should be recognized, and treatment should be promptly started. As colestipol may cause hyperchloremic metabolic acidosis, signs of hyperchloremic acidosis may ensue. These include headaches,

seizures, increased respiratory rate, and arrhythmias. Patients on warfarin therapy should have their International Normalized Ratio (INR) monitored regularly as the absorption is affected by colestipol.

Toxicity

Signs and Symptoms of Overdose

No reports of toxicity associated with colestipol therapy have been reported other than the adverse events described above. Colestipol is relatively safe as it is not absorbed systemically and does not cause severe adverse effects. In the event of an overdose, the primary concern is the potential obstruction of the gastrointestinal tract.

Management of Overdose

Treatment decisions in overdose are influenced by the location and severity of any potential obstruction, along with the assessment of normal gut motility.

Recommendations

Patients often report poor tolerability to colestipol due to the many gastrointestinal adverse effects and poor palatability. Therefore, healthcare professionals should consider this and modify treatment by offering a different drug formulation.

Enhancing Healthcare Team Outcomes

Colestipol is a bile acid sequestrant that may be offered to patients to treat various conditions, such as hypercholesterolemia or pruritus associated with chronic liver disease. Colestipol is less commonly prescribed in the current age due to poor compliance with use and poor tolerability. Still, colestipol is highly effective in managing hypercholesterolemia and pruritus. Therefore, healthcare professionals treating these conditions should be familiar with the properties. This familiarity may enable them to offer patients an improved quality of life.

Clinicians initiating colestipol for hypercholesterolemia should review and consider atherosclerotic cardiovascular disease risk factors. As per the NLA, it is advisable to consult or refer children with familial hypercholesterolemia to a lipid specialist. Nurses should be aware of colestipol's most commonly encountered adverse effects to develop strategies to monitor their occurrence and report them to clinicians. The pharmacist should serve as the main point of contact for those members of the interprofessional team seeking advice on the dosage and administration of colestipol, checking for drug interactions, risk mitigation of adverse effects, and giving clear instructions to patients on how to use the drug.

A study investigated the effectiveness of the multidisciplinary lipid clinic in enhancing guideline-based care for lipid disorders within the healthcare system. Though a modest referral rate from active cardiology and primary care providers, the clinic attended to 83 patients, significantly improving the diagnosis and management of conditions such as familial hypercholesterolemia and dyslipidemia. The impact included increased use of evidence-based therapies, decreased lipid levels, and adaptive measures like transitioning to telehealth for sustained capacity. These results underscore the potential of the multidisciplinary lipid clinic to enhance patient outcomes. Coordination between all interprofessional healthcare team members, including clinicians, specialists, pharmacists, and nurses, is crucial in enhancing outcomes associated with colestipol therapy.

Review Questions

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References

1.
LiverTox: Clinical and Research Information on Drug-Induced Liver Injury [Internet]. National Institute of Diabetes and Digestive and Kidney Diseases; Bethesda (MD): Sep 28, 2017. Colestipol. [PubMed: 31643751]

2.
Gaw A, Packard CJ, Lindsay GM, Murray EF, Griffin BA, Caslake MJ, Colquhoun I, Wheatley DJ, Lorimer AR, Shepherd J. Effects of colestipol alone and in combination with simvastatin on apolipoprotein B metabolism. Arterioscler Thromb Vasc Biol. 1996 Feb;16(2):236-49. [PubMed: 8620338]

3.
Insull W, Davidson MH, Demke DM, Dujovne CA, Eckert SM, Ginsberg D, Goldberg AC, Hodis HN, Hughes TA, Kane JP. The effects of colestipol tablets compared with colestipol granules on plasma cholesterol and other lipids in moderately hypercholesterolemic patients. Atherosclerosis. 1995 Jan 20;112(2):223-35. [PubMed: 7772081]

4.
McCrindle BW, Helden E, Cullen-Dean G, Conner WT. A randomized crossover trial of combination pharmacologic therapy in children with familial hyperlipidemia. Pediatr Res. 2002 Jun;51(6):715-21. [PubMed: 12032266]

5.
Blankenhorn DH, Nessim SA, Johnson RL, Sanmarco ME, Azen SP, Cashin-Hemphill L. Beneficial effects of combined colestipol-niacin therapy on coronary atherosclerosis and coronary venous bypass grafts. JAMA. 1987 Jun 19;257(23):3233-40. [PubMed: 3295315]

6.
Jacobson TA, Ito MK, Maki KC, Orringer CE, Bays HE, Jones PH, McKenney JM, Grundy SM, Gill EA, Wild RA, Wilson DP, Brown WV. National lipid association recommendations for patient-centered management of dyslipidemia: part 1--full report. J Clin Lipidol. 2015 Mar-Apr;9(2):129-69. [PubMed: 25911072]

7.
Jacobson TA, Maki KC, Orringer CE, Jones PH, Kris-Etherton P, Sikand G, La Forge R, Daniels SR, Wilson DP, Morris PB, Wild RA, Grundy SM, Daviglus M, Ferdinand KC, Vijayaraghavan K, Deedwania PC, Aberg JA, Liao KP, McKenney JM, Ross JL, Braun LT, Ito MK, Bays HE, Brown WV, Underberg JA., NLA Expert Panel. National Lipid Association Recommendations for Patient-Centered Management of Dyslipidemia: Part 2. J Clin Lipidol. 2015 Nov-Dec;9(6 Suppl):S1-122.e1. [PubMed: 26699442]

8.
Vuorio A, Kuoppala J, Kovanen PT, Humphries SE, Tonstad S, Wiegman A, Drogari E, Ramaswami U. Statins for children with familial hypercholesterolemia. Cochrane Database Syst Rev. 2019 Nov 07;2019(11) [PMC free article: PMC6836374] [PubMed: 31696945]

9.
Lozano P, Henrikson NB, Dunn J, Morrison CC, Nguyen M, Blasi PR, Anderson ML, Whitlock E. Lipid Screening in Childhood and Adolescence for Detection of Familial Hypercholesterolemia: A Systematic Evidence Review for the U.S. Preventive Services Task Force [Internet]. Agency for Healthcare Research and Quality (US); Rockville (MD): Aug, 2016. [PubMed: 27559556]

10.
Scaldaferri F, Pizzoferrato M, Ponziani FR, Gasbarrini G, Gasbarrini A. Use and indications of cholestyramine and bile acid sequestrants. Intern Emerg Med. 2013 Apr;8(3):205-10. [PubMed: 21739227]

11.
Bunchorntavakul C, Reddy KR. Pruritus in chronic cholestatic liver disease. Clin Liver Dis. 2012 May;16(2):331-46. [PubMed: 22541702]

12.
Lindor KD, Bowlus CL, Boyer J, Levy C, Mayo M. Primary Biliary Cholangitis: 2018 Practice Guidance from the American Association for the Study of Liver Diseases. Hepatology. 2019 Jan;69(1):394-419. [PubMed: 30070375]

13.
Hagag P, Nissenbaum H, Weiss M. Role of colestipol in the treatment of hyperthyroidism. J Endocrinol Invest. 1998 Dec;21(11):725-31. [PubMed: 9972670]

14.
Bajor A, Törnblom H, Rudling M, Ung KA, Simrén M. Increased colonic bile acid exposure: a relevant factor for symptoms and treatment in IBS. Gut. 2015 Jan;64(1):84-92. [PubMed: 24727487]

15.
Farrugia A, Arasaradnam R. Bile acid diarrhoea: pathophysiology, diagnosis and management. Frontline Gastroenterol. 2021;12(6):500-507. [PMC free article: PMC8515273] [PubMed: 34712468]

16.
Payne VW, Secter RA, Noback RK. Use of colestipol in a patient with digoxin intoxication. Drug Intell Clin Pharm. 1981 Nov;15(11):902-3. [PubMed: 7297421]

17.
Hood CJ, Wolley MJ, Kam AL, Kendrik-Jones JC, Marshall MR. Feasibility study of colestipol as an oral phosphate binder in hemodialysis patients. Nephrology (Carlton). 2015 Apr;20(4):250-6. [PubMed: 25557531]

18.
Heel RC, Brogden RN, Pakes GE, Speight TM, Avery GS. Colestipol: a review of its pharmacological properties and therapeutic efficacy in patients with hypercholesterolaemia. Drugs. 1980 Mar;19(3):161-80. [PubMed: 6988203]

19.
McGlone ER, Bloom SR. Bile acids and the metabolic syndrome. Ann Clin Biochem. 2019 May;56(3):326-337. [PubMed: 30453753]

20.
Ebhohon E, Chung RT. Systematic review: efficacy of therapies for cholestatic pruritus. Therap Adv Gastroenterol. 2023;16:17562848231172829. [PMC free article: PMC10226044] [PubMed: 37255856]

21.
Feingold KR. Cholesterol Lowering Drugs. In: Feingold KR, Ahmed SF, Anawalt B, Blackman MR, Boyce A, Chrousos G, Corpas E, de Herder WW, Dhatariya K, Dungan K, Hofland J, Kalra S, Kaltsas G, Kapoor N, Koch C, Kopp P, Korbonits M, Kovacs CS, Kuohung W, Laferrère B, Levy M, McGee EA, McLachlan R, Muzumdar R, Purnell J, Rey R, Sahay R, Shah AS, Singer F, Sperling MA, Stratakis CA, Trence DL, Wilson DP, editors. Endotext [Internet]. MDText.com, Inc.; South Dartmouth (MA): Feb 12, 2024. [PubMed: 27809434]

22.
Jacobson TA, Armani A, McKenney JM, Guyton JR. Safety considerations with gastrointestinally active lipid-lowering drugs. Am J Cardiol. 2007 Mar 19;99(6A):47C-55C. [PubMed: 17368279]

23.
Halpern DG, Weinberg CR, Pinnelas R, Mehta-Lee S, Economy KE, Valente AM. Use of Medication for Cardiovascular Disease During Pregnancy: JACC State-of-the-Art Review. J Am Coll Cardiol. 2019 Feb 05;73(4):457-476. [PubMed: 30704579]

24.

Drugs and Lactation Database (LactMed®) [Internet]. National Institute of Child Health and Human Development; Bethesda (MD): Sep 19, 2022. Colestipol. [PubMed: 30000452]

25.
Steinmetz KL. Colesevelam hydrochloride. Am J Health Syst Pharm. 2002 May 15;59(10):932-9. [PubMed: 12040732]

26.
Arnold MA, Swanson BJ, Crowder CD, Frankel WL, Lam-Himlin D, Singhi AD, Stanich PP, Arnold CA. Colesevelam and colestipol: novel medication resins in the gastrointestinal tract. Am J Surg Pathol. 2014 Nov;38(11):1530-7. [PubMed: 24921636]

27.
Lin S, Sanders DS, Gleeson JT, Osborne C, Messham L, Kurien M. Long-term outcomes in patients diagnosed with bile-acid diarrhoea. Eur J Gastroenterol Hepatol. 2016 Feb;28(2):240-5. [PubMed: 26636406]

28.
Sirmans SM, Beck JK, Banh HL, Freeman DA. Colestipol-induced hepatotoxicity. Pharmacotherapy. 2001 Apr;21(4):513-6. [PubMed: 11310528]

29.
Witztum JL, Jacobs LS, Schonfeld G. Thyroid hormone and thyrotropin levels in patients placed on colestipol hydrochloride. J Clin Endocrinol Metab. 1978 May;46(5):838-40. [PubMed: 262768]

30.
Schwarz KB, Goldstein PD, Witztum JL, Schonfeld G. Fat-soluble vitamin concentrations in hypercholesterolemic children treated with colestipol. Pediatrics. 1980 Feb;65(2):243-50. [PubMed: 7354970]

31.
Camilleri M. Bile Acid diarrhea: prevalence, pathogenesis, and therapy. Gut Liver. 2015 May 23;9(3):332-9. [PMC free article: PMC4413966] [PubMed: 25918262]

32.
Goldberg AC, Hopkins PN, Toth PP, Ballantyne CM, Rader DJ, Robinson JG, Daniels SR, Gidding SS, de Ferranti SD, Ito MK, McGowan MP, Moriarty PM, Cromwell WC, Ross JL, Ziajka PE. Familial hypercholesterolemia: screening, diagnosis and management of pediatric and adult patients: clinical guidance from the National Lipid Association Expert Panel on Familial Hypercholesterolemia. J Clin Lipidol. 2011 May-Jun;5(3):133-140. [PubMed: 21600517]

33.
Jones LK, McMinn M, Kann D, Lesko M, Sturm AC, Walters N, Chen N, Fry K, Brownson RC, Gidding SS, Williams MS, Rahm AK. Evaluation of a multidisciplinary lipid clinic to improve the care of individuals with severe lipid conditions: a RE-AIM framework analysis. Implement Sci Commun. 2021 Mar 19;2(1):32. [PMC free article: PMC7977494] [PubMed: 33741054]

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