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Chris Buck

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UpToDate

Etiology, clinical manifestations, and diagnosis of nephrotic syndrome in children

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Contributor disclosures

All topics are updated as new evidence becomes available. This literature review is current through: Apr 2016. | This topic is updated as new evidence becomes available.

INTRODUCTION — The nephrotic syndrome is caused by two are used diagnostically because the last two may r

- Nephrotic range proteinuria – Urinary protein excretion
- Hypoalbuminemia – Serum albumin concentration
- Edema
- Hyperlipidemia

The etiology, clinical manifestations, and diagnosis of nephrotic syndrome that present as nephrotic syndrome in children are discussed in the [clinical features, and diagnosis of minimal change disease](#) and [glomerulosclerosis](#).)

PATHOGENESIS — Two issues are important in the pathogenesis of nephrotic syndrome: the mechanisms of glomerular injury and proteinuria.

Mechanisms of glomerular injury — A variety of different, disease-specific mechanisms have been described in the nephrotic syndrome:

the glomerular filtration barrier. It is classically character

indications and treatment of idiopathic childhood nephrotic syndrome in children" and "Treatment of idiopathic nephrotic syndrome in children" and "Epidemiology, classification, and pathogenesis of nephrotic syndrome in children".

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Azithromycin (systemic): Pediatric drug information Lexicomp®

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(For additional information [see "Azithromycin \(systemic\): Drug information"](#) and [see "Azithromycin \(systemic\): Patient drug information"](#))

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

Brand Names: US Zithromax; Zithromax Tri-Pak; Zithromax Z-Pak; Zmax

Brand Names: Canada ACT-Azithromycin; Apo-Azithromycin; Apo-Azithromycin Z; Azithromycin for Injection; Azithromycin for Azithromycin; Novo-Azithromycin; PHL-Azithromycin; PMS-Azithromycin; PRO-Azithromycin; Riva-Azithromycin; Sandoz-Azithromycin

Therapeutic Category Antibiotic, Macrolide

Dosing: Neonatal **Note:** Extended release suspension (Zmax) is not interchangeable with immediate-release formulations. All oral otherwise specified. With oral therapy, monitor for infantile hypertrophic pyloric stenosis (IHPS).

General dosing, susceptible infection (*Red Book* [AAP 2012]):

Oral: 10 to 20 mg/kg once daily

IV: 10 mg/kg once daily

Chlamydial conjunctivitis or chlamydial pneumonia: Limited data available. Oral: 20 mg/kg once daily for 3 days (CDC [Workowski 2

Pertussis, treatment and postexposure prophylaxis: Oral, IV: 10 mg/kg once daily for 5 days (*Red Book* [AAP 2012])

Dosing: Usual

(For additional information [see "Azithromycin \(systemic\): Drug information"](#))

UpToDate – Tópicos

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nephrotic syndrome children

All Topics



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Etiology, clinical manifestations, and diagnosis of nephrotic syndrome in children

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DIFFERENTIAL DIAGNOSIS

SUMMARY AND RECOMMENDATIONS

Etiology, clinical manifestations, and diagnosis of nephrotic syndrome in children

Author

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Section Editor

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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: Apr 2016. | **This topic last updated:** Jan 07, 2015.

INTRODUCTION — The nephrotic syndrome is caused by renal diseases that increase the permeability across the glomerular barrier. The first two are used diagnostically because the last two may not be seen in all patients:

- Nephrotic range proteinuria – Urinary protein excretion greater than 50 mg/kg per day
- Hypoalbuminemia – Serum albumin concentration less than 3 g/dL (30 g/L)
- Edema
- Hyperlipidemia

The etiology, clinical manifestations, and diagnosis of nephrotic syndrome in children are reviewed here. The complications and diseases that present as nephrotic syndrome in children are discussed separately. (See "[Complications of nephrotic syndrome in children](#)" and "[Etiology, clinical features, and diagnosis of minimal change disease in adults](#)" and "[Congenital and infantile nephrotic syndrome and glomerulosclerosis](#)".)

PATHOGENESIS — Two issues are important in the pathogenesis of nephrotic syndrome: the mechanisms of glomerular injury

Mechanisms of glomerular injury — A variety of different, disease-specific mechanisms have been described in the nephrotic syndrome:

- Circulating factors in minimal change disease and primary focal segmental glomerulosclerosis. (See "[Idiopathic nephrotic syndrome](#)".)
- Circulating immune factors in disorders such as membranoproliferative glomerulonephritis, poststreptococcal glomerulonephritis, and membranous glomerulonephritis. (See "[Membranoproliferative glomerulonephritis](#)" and "[Membranous glomerulonephritis](#)".)

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Causes of edema in childhood

Generalized edema

Increased capillary hydrostatic pressure

Increased plasma volume from sodium and water retention

Heart failure

(Localization can be seen in some cardiac disease)

Primary renal disease

Acute glomerulonephritis

Renal failure (acute/chronic)

Nephrotic syndrome

Drug induced

Vasodilators (eg, minoxidil)

Dihydropyridine calcium channel blockers (eg, nifedepine and amlodipine)

Venous obstruction

Hepatic cirrhosis

Decreased capillary oncotic pressure (Hypoalbuminemia)

Nephrotic syndrome

Liver failure

Protein-losing enteropathy

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Bronchodilators for bronchiolitis (Review)

Gadomski AM, Scribani MB

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ABSTRACT

Background

Bronchiolitis is an acute, viral lower respiratory tract infection affecting infants and is sometimes treated with bronchodilators.

Objectives

To assess the effects of bronchodilators on clinical outcomes in infants (0 to 12 months) with acute bronchiolitis.

Search methods

We searched CENTRAL 2013, Issue 12, MEDLINE (1966 to January Week 2, 2014) and EMBASE (1998 to January 2014).

Selection criteria

Randomized controlled trials (RCTs) comparing bronchodilators (other than epinephrine) with placebo for bronchiolitis.

Data collection and analysis

Two authors assessed trial quality and extracted data. We obtained unpublished data from trial authors.

Main results

We included 30 trials (35 data sets) representing 1992 infants with bronchiolitis. In 11 inpatient and 10 outpatient studies, oxygen saturation did not improve with bronchodilators (mean difference (MD) -0.43, 95% confidence interval (CI) -0.92 to 0.06, n = 1242). Outpatient bronchodilator treatment did not reduce the rate of hospitalization (11.9% in bronchodilator group versus 15.9% in placebo group, odds ratio (OR) 0.75, 95% CI 0.46 to 1.21, n = 710). Inpatient bronchodilator treatment did not reduce the duration of hospitalization (MD 0.06, 95% CI -0.27 to 0.39, n = 349).

Effect estimates for inpatients (MD -0.62, 95% CI -1.40 to 0.16) were slightly larger than for outpatients (MD -0.25, 95% CI -0.61 to 0.11) for oximetry. Oximetry outcomes showed significant heterogeneity (I^2 statistic = 81%). Including only studies with low risk of bias had little impact on the overall effect size of oximetry (MD -0.38, 95% CI -0.75 to 0.00) but results were close to statistical significance.

In eight inpatient studies, there was no change in average clinical score (standardized MD (SMD) -0.14, 95% CI -0.41 to 0.12) with bronchodilators. In nine outpatient studies, the average clinical score decreased slightly with bronchodilators (SMD -0.42, 95% CI -0.79 to -0.06), a statistically significant finding of questionable clinical importance. The clinical score outcome showed significant

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Authors' conclusions

Bronchodilators such as albuterol or salbutamol do not improve oxygen saturation, do not reduce hospital admission after outpatient treatment, do not shorten the duration of hospitalization and do not reduce the time to resolution of illness at home. Given the adverse side effects and the expense associated with these treatments, bronchodilators are not effective in the routine management of bronchiolitis. This meta-analysis continues to be limited by the small sample sizes and the lack of standardized study design and validated outcomes across the studies. Future trials with large sample sizes, standardized methodology across clinical sites and consistent assessment methods are needed to answer completely the question of efficacy.



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1. **Temporal Trends in Patient Characteristics and Outcomes Among Children Enrolled in Mozambique's National Antiretroviral Therapy Program.**
[Auld AF¹](#), [Alfredo C](#), [Macassa E](#), [Jobarteh K](#), [Shiraishi RW](#), [Rivadeneira ED](#), [Houston J](#), [Spira TJ](#), [Ellerbrock TV](#), [Vaz P](#).

Author information

Abstract
BACKGROUND: During 2004-2009, >12,000 children (<15 years old) initiated antiretroviral therapy (ART) in Mozambique. Nationally representative outcomes and temporal trends in outcomes were investigated.
METHODS: Rates of death, loss to follow-up (LTFU) and attrition (death or LTFU) were evaluated in a nationally representative sample of 1054 children, who initiated ART during 2004-2009 at 25 facilities randomly selected using probability-proportional-to-size sampling.
RESULTS: At ART initiation during 2004-2009, 50% were male; median age was 3.3 years; median CD4% was 13%; median CD4 count was 375 cells/ μ L; median weight-for-age Z score was -2.1. During 2004-2009, median time from HIV diagnosis to care initiation declined from 33 to 0 days ($P = 0.001$); median time from care to ART declined from 93 to 62 days ($P = 0.004$); the percentage aged <2 at ART initiation increased from 16% to 48% ($P = 0.021$); the percentage of patients with prior tuberculosis declined from 50% to 10% ($P = 0.009$); and the percentage with prior lymphocytic interstitial pneumonia declined from 16% to 1% ($P < 0.001$). Over 2652 person-years of ART, 183 children became LTFU and 26 died. Twelve-month attrition was 11% overall but increased from 3% to 22% during 2004-2009, mainly because of increases in 12-month LTFU (from 3% to 18%).
CONCLUSION: Declines in the prevalence of markers of advanced HIV disease at ART initiation probably reflect increasing ART access. However, 12-month LTFU increased during program expansion, and this

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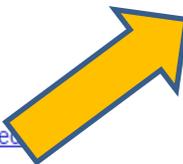
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Surveillance of HIV Drug Resistance in Children Receiving Antiretroviral Therapy: A Pilot Study of the World Health Organization's Generic Protocol in Maputo, Mozambique

P. Vaz,¹ O. Augusto,¹ D. Bila,² E. Macassa,¹ A. Vubil,² I. V. Jani,² R. Pillon,³ P. Sandstrom,³ D. Sutherland,⁴ C. Giaquinto,⁵ M. R. Jordan,^{6,7} and S. Bertagnolio⁶

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Between 2007 and 2008, the Mozambique Ministry of Health conducted an assessment of human immunodeficiency virus drug resistance (HIVDR) using World Health Organization (WHO) methods in a cohort of children initiating antiretroviral therapy (ART) at the main pediatric ART referral center in Mozambique. It was shown that prior to ART initiation 5.4% of children had HIVDR that was associated with nevirapine perinatal exposure ($P < .001$). Twelve months after ART initiation, 77% had viral load suppression (<1000 copies/mL), exceeding the WHO target of $\geq 70\%$; 10.3% had HIVDR at 12 months. Baseline HIVDR ($P = .04$), maternal prevention of mother-to-child transmission ($P = .02$), and estimated days of missed medication ($P = .03$) predicted HIVDR at 12 months. As efforts to eliminate pediatric AIDS are intensified, implementation of ritonavir-boosted protease inhibitor regimens in children with prevention of mother-to-child transmission exposure may reduce risk of virological failure in our setting.

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