for the standard care group (53 to 51; \(P>0.05\)), the educational support group (55 to 55; \(P>0.05\)), or the BFST-D group (55 to 57; \(P>0.05\)). However, among adolescents with baseline HbA1C higher than 9%, the BFST-D group demonstrated a greater change in DSMP scores than the educational support group (3.5 vs –0.5; \(P<0.05\)) and the standard care group (3.5 vs 0.3; \(P<0.05\)). Changes in HbA1C were also similar from baseline to follow-up for all 3 groups (standard care: 9.5% to 9.2%; \(P>0.05\); educational support: 9.7% to 8.9%; \(P>0.05\); BFST-D: 9.6% to 8.8%; \(P>0.05\)). Yet, among those with baseline HbA1C higher than 9.0%, the BFST-D group had a significantly greater change in HbA1C than the standard care group (–1.3% vs –0.4%; \(P<0.05\)) but was similar to the educational support group (–1.3% vs –1.1%; \(P>0.05\)).

Additional analysis of the RCT cited above followed the same 104 families for an additional 12 months to examine long-term effects of BFST-D. Mean DSMP score for the BFST-D group was significantly higher than the standard care group at 6 months (57 vs 52; \(P<0.05\)), 12 months (58 vs 52; \(P<0.05\)), and 18 months (57 vs 53; \(P<0.05\)), but was similar to the educational support group score at 6 months (57 vs 55; \(P>0.05\)), 12 months (58 vs 56; \(P>0.05\)), and 18 months (57 vs 55; \(P>0.05\)). The mean HbA1C for the BFST-D group was significantly lower than the standard care group at 6 months (8.8 vs 9.1; \(P<0.05\)), 9 months (8.7 vs 9.5; \(P<0.05\)), 12 months (8.9 vs 9.6; \(P<0.05\)), 15 months (8.6 vs 9.6; \(P<0.05\)), and 18 months (8.8 vs 9.6; \(P<0.05\)) and was significantly lower than the educational support group at 9 months (8.7 vs 9.5; \(P<0.05\)), 15 months (8.6 vs 9.6; \(P<0.05\)), and 18 months (8.8 vs 9.5; \(P<0.05\)).

Further analysis of the original RCT examined the effect of BFST-D on family communication. Families were asked to discuss and resolve a diabetes conflict for 10 minutes. Discussions were recorded and analyzed to determine the rates of positive communication (praise/affirmation) and negative communication (anger/interruption) by adolescents and their parents.

Adolescents’ rates of positive communication were similar among the BFST-D group and the standard care group at 6 months (2.1 vs 1.6; \(P>0.05\)), 12 months (2.0 vs 1.7; \(P>0.05\)), and 18 months (2.0 vs 1.4; \(P>0.05\)) as well as the educational support group at 6 months (2.1 vs 1.5; \(P>0.05\)), 12 months (2.0 vs 1.6; \(P>0.05\)), and 18 months (2.0 vs 1.7; \(P>0.05\)). The rate of adolescent negative communication was significantly lower in the BFST-D group than the standard care group at 6 months (2.1 vs 4.0; \(P<0.05\)), 12 months (2.6 vs 4.5; \(P<0.05\)), and 18 months (2.8 vs 4.2; \(P<0.05\)) as well as the educational support group at 6 months (2.1 vs 3.5; \(P<0.05\)), but not at 12 months (2.6 vs 2.9; \(P>0.05\)) or 18 months (2.8 vs 3.0; \(P>0.05\)).

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What is the incidence of acute rheumatic fever in untreated children with Streptococcus pharyngitis?

Evidence-Based Answer

Prior to the antibiotic era, the incidence of acute rheumatic fever was 28.5 cases per 100,000 children (SOR: C, retrospective study). Since the advent of antibiotics, the incidence of acute rheumatic fever has declined and is as low as 3.7 per 100,000 people in some parts of the United States (SOR: C, retrospective study).

Group A Streptococcus (GAS) pharyngitis, when left untreated, triggers an autoimmune response in approximately 1% of the population, which can lead to acute rheumatic fever. In a review of a national health and morbidity survey from 1935 to 1936, an era prior to antibiotic treatment, the incidence of acute rheumatic fever in Baltimore and its surrounding communities was 28.5 cases per 100,000 people aged 5 to 19 years old.

Since the advent of antibiotics, the incidence of acute rheumatic fever in untreated children has not specifically been tracked. A 1987 retrospective trial that conducted chart reviews from western Pennsylvania and the surrounding area revealed cases of untreated GAS infections were mainly due to lack of overt symptoms of pharyngitis or failing to seek medical attention for any symptoms present. No incidence was extrapolated from this study.
A 2012 population-based, retrospective laboratory surveillance study identified invasive GAS disease among all Utah residents from 2002 to 2010. This study detected 1,514 cases of invasive GAS. The incidence of acute rheumatic fever in Utah decreased from 6.1 cases per 100,000 in 2002 to 3.7 per 100,000 in 2010. Unfortunately, this study did not distinguish between children and adults, or treated and untreated individuals when reporting incidence.

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Could procalcitonin be used as an indicator to start or stop antibiotics for acute upper and lower respiratory infections?

Evidence-Based Answer
Procalcitonin-guided algorithms for the initiation and stopping of antibiotics in acute upper and lower respiratory infections (ARIs) decreased antibiotic usage and duration in the primary care setting and was not associated with higher mortality rates or treatment failures (SOR: A, meta-analysis of RCTs).

A 2012 Cochrane review examined 14 RCTs (N=4,211) of immunocompetent adult patients (≥18 years of age) with ARIs to assess the safety and efficacy of using procalcitonin algorithms versus usual care for starting or stopping antibiotics in different clinical settings (primary care, emergency department, hospital wards, ICUs).1

Two RCTs in the Cochrane review (n=1,008) specifically focused on assessing this question in a primary care setting for ARIs (acute sinusitis, otitis media, pharyngitis, tonsillitis, laryngitis, influenza, common cold, acute bronchitis, pneumonia, COPD exacerbation, asthma exacerbation) in immunocompetent adults with no antibiotics exposure in the prior 14 to 28 days, no chronic liver disease, no active tuberculosis, no cystic fibrosis, and no autoimmune or systemic disorders.2,3 The studies considered a procalcitonin concentration of <0.25 mcg/L as an indication that bacterial infection was unlikely and recommended withholding antibiotics. The primary endpoints were all-cause mortality and treatment failure defined as death, hospitalization, ARI-specific complications, recurrent or worsening infections, or ARI-associated discomfort at 30 days post initial visit.

Procalcitonin-guided initiation of antibiotics for ARIs was not associated with higher mortality rates (pooled results; 0 deaths in the procalcitonin-guided group, 1 death in the control group) and no difference in treatment failure (pooled results; 31% in procalcitonin-guided vs 33% in control; OR 0.94; 95% CI, 0.72–1.2). In these 2 trials, fewer patients in the procalcitonin-guided group had antibiotics initiated than in the control group (23% vs 63%; adjusted OR 0.1; 95% CI, 0.07–0.14).2,3

One of the trials above obtained a repeat procalcitonin level 6 to 24 hours later and initiated antibiotics if the concentration was ≥0.25 mcg/L or had increased by >50%. All patients started on antibiotics were reassessed 3 days later and if a procalcitonin level was <0.25 mcg/L, antibiotics were recommended to be discontinued. The adjusted antibiotic duration in the procalcitonin-guided group was decreased by 1 day (95% CI, 0.4–1.7 days).2

Limitations of these studies included lack of provider blinding, underpowered data to detect small mortality differences, and lack of adherence to the algorithm (approximately 15% of providers did not adhere to the procalcitonin-guided algorithm). No consensus guidelines have been published on the use of procalcitonin to guide antibiotic administration for ARIs in the primary care setting.

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