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V Mundada and N Brown

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Correspondence to: M Roe, Department of Child Health, Southampton General Hospital, Tremona Road, Southampton SO16 6YD, UK; michael.roe@suht.swest.nhs.uk

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Follow-up analysis of serious bacterial infections in children with fever without localising signs: how do the National Institute for Clinical Excellence guidelines perform with the emergence of non-vaccine pneumococcal serotypes?

After the recommendation in 2006 for PCV7 to be used in the UK, the 2007 National Institute for Health and Clinical Excellence (NICE) guidelines for management of feverish children recommended only evaluation of the urine in children with fever without localising signs (FWLS) that met the “green” criteria.¹ In a previous study published in this journal, we presented data supporting the NICE guidelines.² Reports describing the emergence of non-vaccine serotypes of *Streptococcus pneumoniae*, specifically 19A, have raised concerns about increasing invasive disease.^{3 4} We conducted this follow-up study of children with FWLS to re-evaluate the NICE guidelines in light of a possible increase in non-vaccine serotypes.

All research methods were identical to those discussed in our previous paper.²

To determine the local prevalence of serotype 19A, we examined *S pneumoniae* isolates causing bacteraemia and/or meningitis in paediatric patients since 2004, using reagents from Statens Serum Institut (Copenhagen, Denmark). We performed screening using latex pool B antisera followed by specific antiserum against serotype 19A.

From July 2004 to May 2006, there were 219 children with FWLS. There were eight

positive blood cultures, but only one pathogen (0.46%; *Escherichia coli*) and no cases of *S pneumoniae*. These rates were unchanged compared to years 2001–mid 2004.² Seventeen pneumococcal isolates were identified with 13 available for testing. Using the latex pool, two of 13 isolates showed agglutination and one isolate was confirmed serotype 19A.

The NICE guidelines issued in 2007 anticipated the decrease in occult bacteraemia and recommended eliminating blood cultures in FWLS children. Recent publications have described a multiple antibiotic-resistant serotype, 19A, which is causing an increasing percentage of superficial and invasive disease.^{3 4} While this may cause practitioners concern, it is important to keep in mind that rates of US invasive disease have been stable.⁵ The total number of US cases of invasive pneumococcal disease compared to pre-PCV7 years is still significantly lower by 70%. Our study documents that despite circulating 19A causing invasive disease at our institution at rates comparable to national levels, there has been no pneumococcal occult bacteraemia in over 5 years. We hypothesise that the pathogenesis of non-vaccine serotypes like 19A is different than vaccine serotypes but this remains to be proven. Practitioners that are no longer performing blood cultures on feverish children should be reassured that the NICE guidelines are still valid based on our findings.

E A Waddle,¹ K E Hanson,^{2,3} R Jhaveri¹

¹Department of Pediatrics, Duke University Medical Center, Durham, North Carolina; ²Department of Medicine, Duke University Medical Center, Durham, North Carolina;

³Department of Pathology, Duke University Medical Center, Durham, North Carolina

Correspondence to: Ravi Jhaveri, Division of Pediatric Infectious Diseases, DUMC Box 3499, Duke University Medical Center, Durham, North Carolina 27710, USA; ravi.jhaveri@duke.edu

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Evolution of BCG scar and its relation to atopic diseases/asthma and the nutritional status of the child

Bacille Calmette Guérin (BCG) has been the sole vaccine for tuberculosis since 1921. The BCG scar disappears in a up to 25% of children.¹ The presence or absence of a scar, however, does not necessarily correlate with the degree of immunoprotection. Some studies mention its immunostimulant effect which primes the Th1 lymphocytes to produce cytokines that antagonise atopy in both animal models and man.²

We conducted a cross-sectional study in a rural part of Maharashtra, India. The study population included children from seven randomly selected villages in Kallamb taluka of Osmanabad district in Maharashtra. To determine the evolution of the scar, children from two different age groups were selected: 1–2 years and 4–5 years.

1. Anthropometric assessments were performed on each child, and body mass index (BMI) was calculated as a marker of nutritional status.
2. The BCG scar size (diameter) was measured on those children who had a scar after vaccination.
3. For assessment of children with asthma/wheezing or atopy, the validated questionnaire of the ALSPAC study was used.³ The questions were:
 - a. “Has your child had wheezing with whistling on his/her chest when he/she breathes?”
 - b. “Has your child had an itchy/sore rash in the joints and the creases of his/her body (eg, behind the knees, under the arms)?”
4. To assess immunological status, the following questions were asked:
 - a. “For how many days in total has your child had fever in the last month?”
 - b. “For how many days in total has your child had diarrhoea in the last month?”

All analyses were performed using Stata V6. Continuous variables were analysed by Student t test and/or multiple linear regression. Dichotomous outcomes were analysed by unconditional logistic regression and expressed as odds ratios (ORs). The outcomes were calculated both unadjusted (scar status only) and adjusted for BMI.

The main findings were that the presence of a BCG scar is associated with a consistent non-significant protective effect spanning our age range against (verbally reported) atopic symptoms (eczema and asthma). The protective effect was stronger for eczema than for asthma. Unadjusted ORs (95% CI) for atopy at 1–2 and 4–5 years were, respectively, 0.59 (0.18 to 1.97) and 0.45 (0.13 to 1.6). After adjustment for BMI, they were 0.56 and 0.45, respectively.

For asthma, unadjusted ORs at 1–2 and 4–5 years were, respectively, 0.67 (0.25 to 1.9) and 0.45 (0.12 to 1.7), and 0.46 and 0.47, respectively, after adjustment for BMI. In the older age group, scar presence had a significant additional protective effect against fever: mean days of fever per month was 1.6 in children with a scar and 2.7 in those without ($p = 0.04$).

Our findings suggest that the presence of a BCG scar is associated with immunological differences in children, and it is the presence of a scar rather than its absolute size that is of more importance. Two further studies are planned to delineate the natural evolution of the scar and to determine whether revaccination of children without a scar confers additional benefits.

V Mundada,¹ N Brown²

¹ Princess of Wales Hospital, Bridgend, UK; ² Salisbury District Hospital, Salisbury, UK

Correspondence to: Dr V Mundada, Princess of Wales Hospital, Bridgend CF31 1RQ, UK; vbmundada@gmail.com

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Stuttering and bilingualism

Bilingualism is an asset in modern society and any recommendations to parents about language use during the preschool years must be supported by convincing evidence. Hence we write to refute the conclusions of Howell *et al* that speaking two languages during the preschool years increases the chances of a child starting to stutter and decreases the likelihood of recovery from stuttering.¹

The problem, as we see it, is that the authors have drawn conclusions about the general population of bilingual children from a clinical cohort. They found that in a clinical group of 38 stuttering bilingual children aged 8–10 years, those who had spoken two languages during the preschool years outnumbered those who had spoken only one non-English language during the preschool years. Since the proportions were reversed in a cohort of age matched non-stuttering bilingual children, the authors

concluded – erroneously – that speaking two languages increases the risk of stuttering. We suggest that the best way to establish if there is a relationship between stuttering and bilingualism is with an epidemiological study.

In addition, their clinical cohort is extremely biased. The children were first assessed at age 8–10 years and mean age of stuttering onset was determined to be around 4 years 9 months. However, the generally accepted mean age of onset is around 2 years 10 months.² Given that almost all natural recovery occurs in the first 3 years after onset,² Howell *et al*'s cohort would not have contained the many children who started stuttering and either recovered naturally or were successfully treated during the preschool years (for an effective treatment see Jones *et al*³). Also, the ratio of males to females in the cohort was around 4:1. This ratio is known to be close to 1:1 soon after onset of stuttering and only increases to that of the older children in this study because many more girls than boys recover naturally.²

The authors' conclusion that deferring speaking English until going to school increases chances of recovery from stuttering is also flawed. Those children deemed to have recovered from stuttering when seen again at age 12 years may not have recovered at all: "All recovered speakers had less than 4% stuttered syllables, whereas all persistent speakers had more than 4% stuttered syllables". Yairi and Ambrose's metric is "stutter-like dysfluencies",² not "stuttered syllables" as claimed by Howell *et al*, and 4% stuttered syllables approximates one stutter every 12 words.

A Packman,¹ M Onslow,¹ S Reilly,² J Attanasio,³ R Shenker⁴

¹ Australian Stuttering Research Centre, The University of Sydney, Lidcombe, New South Wales, Australia;

² Murdoch Childrens Research Institute, Department of Pediatrics, University of Melbourne, Parkville, Victoria, Australia; ³ Department of Communication Sciences and Disorders, Montclair State University, Montclair, New Jersey, USA; ⁴ Montreal Fluency Centre, Westmount, Quebec, Canada

Correspondence to: Ann Packman, Australian Stuttering Research Centre, The University of Sydney, PO Box 170, Lidcombe, NSW 1825, Australia; a.packman@usyd.edu.au

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Successful dietary treatment of recurrent intussusception

We report the first case of an infant with recurrent intussusceptions secondary to intestinal lymphoid hyperplasia due to food allergy (soya) that was ultimately successfully treated by dietary restriction.

A female infant presented with frequent, worsening clinical episodes of sudden collapse secondary to intussusceptions over a 12-month period confirmed by ultrasound on three occasions. She also had pronounced infantile colic and moderately loose stools. Following her seventh admission, an ileo-caecal resection with limited right hemicolectomy and anastomosis was performed. Perioperative findings included a thickened terminal ileum and caecum with multiple prominent nodes. Histopathology showed pronounced non-specific nodular lymphoid hyperplasia in the small bowel. Despite surgery a further episode of collapse occurred requiring aggressive intravenous resuscitation. Revision of the dietary history showed a strong temporal association between the ingestion of soya products and her intussusceptions. There was a family history of food allergies; her father was allergic to fish and her brother to cow's milk and peanut.

DISCUSSION

Various investigations were performed in an attempt to find the cause of the recurrent intussusceptions (table 1).

Following a positive patch test to soya and a mildly positive patch test to wheat and egg, the patient was started on an amino acid based formula (Neocate; SHS, Liverpool, UK) and a strict soya, egg, nut and cow's milk free diet. A complete resolution of all symptoms was noted until 18 months later when she was inadvertently given soya formula and presented within 2 h with another intussusception.

We conclude that that her recurrent intussusceptions were due to food allergy in view of the chronic intestinal lymphoid hyperplasia, positive skin patch testing, temporal association between soya ingestion and intussusceptions, and complete resolution of both intussusceptions and lymphadenopathy on dietary exclusion. Two previous cases have been reported where recurrent intussusception associated with intestinal lymphoid hyperplasia was treated with oral steroids resulting in resolution of the lymphoid hyperplasia and cessation of the recurrent intussusceptions.¹ However this is the first case to be successfully treated with a hypoallergenic diet.

Intestinal lymphonodular hyperplasia may be the hallmark histological lesion in patients with non-IgE-mediated food allergy.^{2,3} Food allergy may play a part in