

**MISSING DATA IN THE DIAGNOSIS OF SUDDEN SENSORINEURAL HEARING LOSS**

**To the Editor:**

I would like to comment on a recent paper published in IMAJ [1]. It stated that the patient (identified as having H1N1-positive infection) complained of right-sided sudden hearing loss. There was no physical examination or audiometric testing, imperative for this important diagnosis.

First, examination with tuning forks revealed: "... bilateral normal eardrum with normal pure-tone air and bone conduction (Weber and Rinne tests)." Assuming the patient had symmetric normal hearing before admission – in the case of acute sudden unilateral sensorineural hearing loss I would expect lateralization of the Weber to the contralateral ear, hence to the left side – "normal Weber test" does not comply with the mentioned diagnosis. Second, a full audiometric testing is essential to confirm the diagnosis of sudden sensorineural hearing loss. The accepted (not absolute) criteria are sensorineural hearing loss of more than 30 dB in three consecutive frequencies occurring within less than 3 days. Moreover the PT (pure tone) curve and SRT (speech reception threshold) and word discrimination are important for determining the severity of the loss and other prognostic factors as well as a baseline for future audiometric tests [2]. Despite all this, there was no mention of audiometric test results.

**M. Warman MD**

Dept. of Otolaryngology Head and Neck Surgery, Kaplan Medical Center, Rehovot, Israel [meirwa@clalit.org.il]

**References**

1. Blum A, Simsolo C. Acute unilateral sensorineural hearing loss due to H1N1 infection. *IMAJ* 2010; 12: 450.
2. Arts HA. Sensorineural hearing loss: evaluation and management in adults. In: Cummings Otolaryngology Head and Neck Surgery, 4th edn. 2005; Chapter 155: 3550-5.

**PERIODONTAL DISEASE AND OBESITY IN CHILDREN**

**To the Editor:**

Childhood and adolescent obesity are related to health risks, medical conditions, and increased risk of adult obesity. The prevalence of childhood overweight has more than doubled in the past 25 years. The National Health and Nutrition Examination Survey (NHNES) data from a survey of children in 1999–2000 revealed that 30.3% of children 6 to 11 years of age had a > 85th percentile body mass index (BMI) [1].

Although periodontal disease affects only 2%–5% of the pediatric population compared to 30–40% of the adult population, the early-onset form of periodontitis and puberty gingivitis comprises a distinctive and significant morbidity in this age group [2].

In a case-control study of a nationally representative sample using data from the Third National Health and Nutrition Examination Survey, Reeves et al. [3] studied 2452 non-smokers, aged 13 to 21 years, who underwent a periodontal examination and had complete information for age, gender, and smoking habits. This study suggested that body weight and waist circumference were associated with periodontitis, but the association varied by age. Adolescents aged 13 to 16 years were not at increased risk of chronic periodontitis, while adolescents aged 17 to 21 years had an increased risk per 1 kg increase in body weight. Similarly, adolescents aged 13 to 16 years were not at increased risk for periodontal disease, while adolescents aged 17 to 21 years were at an increased risk of periodontal disease per 1 cm increase in waist circumference. The authors concluded that periodontitis may follow patterns similar to other chronic conditions that originate early in life and are related to central adiposity [3].

Al-Zahrani and colleagues [4] found a significant association between the measures of body fat and periodontal disease among younger adults, but not

middle or older adults; the authors conclude that in a younger population, overall and abdominal obesity are associated with increased prevalence of periodontal disease, while underweight (BMI < 18.5) was associated with decreased prevalence. "Obesity could be therefore a potential risk factor for periodontal disease especially among younger individuals" (18–34 years old). Promotion of healthy nutrition and adequate physical activity may be additional factors to prevent or halt the rate of progression of periodontal disease [4].

The cytokine tumor necrosis factor-alpha (TNFα) plays a significant role in the pathogenesis and development of periodontitis, and an increased level of TNFα has been found in gingival crevicular fluid (GCF) in patients with periodontitis. Additionally, the cytokine interleukin-8 (IL) is also enhanced in GCF in subjects with periodontal disease. IL-8 is a powerful neutrophil chemoattractant that is produced in response to various inflammatory stimuli, including TNFα. Obesity contributes to a pro-inflammatory environment by producing pro-inflammatory cytokines, and the levels of TNFα and IL-8 in GCF are directly linked to the extent of obesity in relation to BMI. Other mechanisms associated with the chronic inflammatory response from the oral cavity may be linked with obesity.

We suggest that the medical community be aware of chronic oral inflammation as a potential hazard associated with obesity in children. Periodontal disease in children may predispose overweight and should therefore be monitored carefully.

**J. Katz and E. Bimstein**

University of Florida College of Dentistry

**References**

1. Boney CM, Verma A, Tucker R, Vohr BR. Metabolic syndrome in childhood: association with birth weight, maternal obesity, and gestational diabetes mellitus. *Pediatrics* 2005; 115: e290-6.
2. Grossi SG, Collier DN, Perkin RM. Integrating oral health to the care of overweight children: a model of care whose time has come. *J. Pediatr* 2008; 152: 451-2.
3. Reeves AF, Rees JM, Schiff M, Hujuel P. Total body weight and waist circumference associated

with chronic periodontitis among adolescents in the United States. *Arch Pediatr Adolesc Med* 2006; 160: 894-9.

- Al-Zahrani MS, Bissada NF, Borawskit EA. Obesity and periodontal disease in young, middle-aged, and older adults. *J Periodontol* 2003; 74: 610-15.

### POSSIBLE LINK BETWEEN MONTH OF BIRTH AND CHILDHOOD LEUKEMIA SUPPORTS THE HYPOTHESIS OF AN INFECTIOUS ETIOLOGY

#### To the Editor:

The etiology of acute lymphoblastic leukemia (ALL) remains largely unknown. Present evidence points to an interplay between genetic and environmental factors. Based on epidemiological studies including population mixing, several environmental causes have been proposed as contributing to the etiology of ALL. The possible contribution of a common infection has been discussed in some reports.

Higgins et al. and others [1] found no evidence of seasonality in month of birth (MOB) for any subgroup of childhood leukemia, while others reported seasonal variations in the onset of ALL in children [2]. Only a few studies have focused on rhythmicity of MOB and the findings were mixed, possibly due to small cohorts [3,4].

If indeed seasonal epidemics play a role in the initiation of childhood leukemia in the genetically predisposed population, these children can be expected to have a different MOB seasonality from that in the general population. To test this hypothesis we analyzed the seasonality of MOB in 543 children with acute leukemia between the age of 0 and 18 years (301 males, 242 females) diagnosed and treated at the Pediatric Hematology Oncology Division of the Schneider Children's Medical

Center of Israel, a tertiary care center, during the period 1984–2007. The MOB of the patients was compared to the MOB of the general population in Israel, which served as a control group (n=1,040,558; 534,650 males, 505,908 females).

We found different patterns of MOB in various subtypes of leukemia. A rhythmic pattern of 6 months was found in MOB patterns of both, male (n=161,  $P < 0.01$ ,  $R = 0.71$ ) and female (n=151,  $P < 0.01$ ,  $R = 0.88$ ) patients with B cell precursor ALL. Two peaks were observed in summer (June) and winter (December–January). These rhythmic patterns, observed in ALL patients, differed from those observed in the general population (males n=45,710 and females n=43,930) in whom the rhythm is annual (12 months) with a peak in September ( $P < 0.01$ ,  $R = 0.85$  for males,  $P < 0.01$ ,  $R = 0.86$  for females).

A subpopulation of B cell precursor ALL, with Tel/AML translocation or hyperdiploidy, showed a different rhythmic pattern. In males (n=66) the rhythm of MOB is similar to the pattern observed in the general population, with a peak in October ( $P < 0.01$ ,  $R = 0.58$ ). The MOB pattern in the females (n=45) showed 8+4 month rhythm peaks in July and November ( $P < 0.01$ ,  $R = 0.79$ ).

Boys diagnosed between age 2 and 10 years with acute myeloid leukemia showed an 8+6 months rhythm as opposed to those diagnosed at a later age having a statistically significant different pattern of 8+12 months.

The findings in this study that the MOB pattern of children with ALL and AML differs from that in the general population resemble those reported by our group for several autoimmune diseases such as Type 1 diabetes mellitus (T1DM), autoimmune thyroid diseases,

celiac disease and multiple sclerosis. Those findings were interpreted as suggestive for a seasonal environmental factor in the etiology of these diseases. In the case of T1DM, evidence for a role of perinatal enterovirus infections has been reported. Also rubella, mumps and other viruses have been linked to T1DM.

The fact that previously reported studies on the MOB in children with leukemia did not show a distinct pattern is probably due to the fact that mixed types of leukemia were analyzed. Our study analyzing subtypes of childhood leukemia unmasked differences in MOB between the groups and the general population. These findings could relate to differences not only in genetic background but also to differences in environmental etiological factors such as viral infections.

I. Yaniv MD<sup>1</sup>, H. Lewy PhD<sup>2</sup>,  
G. Avrahami MD<sup>1</sup>, M. Jeison PhD<sup>1,3</sup>,  
B. Stark MD<sup>1,3</sup> and Z. Laron MD<sup>2</sup>

<sup>1</sup>Pediatric Hematology Oncology and

<sup>2</sup>Endocrinology and Diabetes Research Unit, WHO Collaborating Center for the Study of Diabetes in Youth, and <sup>3</sup>Cancer Cytogenetic Laboratory, Schneider Children's Medical Center of Israel, Petah Tikva, affiliated with Sackler Faculty of Medicine, Tel Aviv University, Israel [iyaniv@clalit.org.il]

#### References

- Higgins CD, dos-Santos-Silva I, Stiller CA, Swerdlow AJ. Season of birth and diagnosis of children with leukaemia: an analysis of over 15 000 UK cases occurring from 1953-95. *Br J Cancer* 2001; 84: 406-12.
- Badrinath P, Day NE, Stockton D. Seasonality in the diagnosis of acute lymphocytic leukaemia. *Br J Cancer* 1997; 75: 1711-13.
- Feltbower RG, Pearce MS, Dickinson HO, Parker L, McKinney PA. Seasonality of birth for cancer in Northern England, UK. *Paediatr Perinatal Epidemiol* 2001; 15: 338-45.
- Sorensen HT, Pedersen L, Oslen J, Rothman KJ. Seasonal variation in month of birth and diagnosis of early childhood acute lymphoblastic leukemia. *JAMA* 2001; 285: 168-9.

**“You desire to know the art of living, my friend? It is contained in one phrase: make use of suffering”**

Henri Frederic Amiel (1821-1881), Swiss philosopher and writer

**“Although a democracy must often fight with one hand tied behind its back, it nonetheless has the upper hand”**

Aharon Barak (born 1936), Israeli law professor and former President of the Supreme Court of Israel