manifestations when sustaining infections (eg, higher fever and more pronounced leukocytosis). We speculate that our age-matched patients with OAI caused by the classic pathogens presented with more acute clinical and biologic signs than older children with *S. aureus* arthritis. Thus, we suggest that it would be interesting for Bonacorsi et al to apply the predictive score only to those children aged less than 4 years to determine whether the discrimination is more accurate.

Regarding children with *K. kingae* OAI, we disagree with the assumption of Bonacorsi et al that our patients were less severely affected in terms of median temperature, WBC, and CRP as the median values were roughly the same in both studies. We can hypothesize that the difference in data dispersion observed for *K. kingae* OAI between the 2 groups is because of the fact that larger joints were affected in the Bonacorsi series (75% of hip and knee arthritis), whereas only 25% to 30% of our children had larger joints involved. We also speculate that the less pronounced inflammatory response observed in our patients might be attributed to the fact that *K. kingae* infections detected by molecular assays could encompass OAI with lower bacterial loads.

As suggested by Bonacorsi et al, the better discrimination of the score in our patients may be due to the "underrepresentation" of patients with *S. aureus* (8 cases), compared with those with *S. pyogenes* (12 cases) or *S. pneumoniae* (5 cases). A few reports have suggested that OAI due to these latter 2 pathogens were characterized by a marked inflammatory syndrome and high fever.[5] However, young children with acute OAI due to *S. aureus* present mostly as ill-appearing children with high fever, increased CRP, and elevated WBC. A few studies demonstrated that more than 90% of children with OAI caused by *S. aureus* are febrile at the time of consultation, and that CRP ranges between 70 and 118 mg/L for infection due to MSSA.[6,7] For our 8 cases of OAI caused by *S. aureus*, we observed that 5 children had 4 positive predictive features (fever $\geq$38°C, CRP $\geq$55 mg/L, WBC $\geq$14,000 leukocytes/ mm^3, and band shift $\geq$150 forms/mm^3), 2 had 3, and only 1 showed 2 of these features.

In our opinion, the clinical presentation of OAI is not only dependent on the responsible microorganism but also on the age of the child. The results of our study emphasize the need for prospective studies to better define the clinical presentation according to age and the causative organism. Finally, we are in complete agreement with Bonacorsi et al regarding the need for a large multicenter study to improve the predictive model and to better differentiate OAI caused by *K. kingae* from those due to pyogenic germs.

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Raoultella Infection Causing Fever of Unknown Origin

To the Editors:

Raoultella spp. are Gram-negative aerobic bacilli belonging to the Enterobacteriaceae family[1–3] and are closely related to *Klebsiella* spp.[1–4] These organisms infrequently cause human infections, but appear to have a pathogenicity similar to that of *Klebsiella pneumoniae*.[3] Fish poisoning is the usual clinical presentation, especially for the fishes that are members of the Scomberesocidae or Scombridae (the so-called scombroid fish) families and that contain large amounts of histamine.[5,6] A hazardous level of histamine is produced by the microbial decarboxylation of the free histidine in the muscular tissue of fish.[7,8] *Raoultella* species cannot be distinguished from *Klebsiella* species using commercialized systems; thus, additional tests are necessary.[3,9,10] It has been demonstrated that *Raoultella planticola* and *Raoultella ornithinolytica* can infect human beings, and they are resistant to amino- and carboxypenicillins unless combined with clavulanate.[10,11] In 2009, Morais et al[4] reported an enteric, fever-like syndrome caused by *R. ornithinolytica*; Vos and Laureys[12] reported on a giant renal cyst caused by *R. ornithinolytica* in a patient with a colonic obstruction. In 2010, Mau et al[13] reported bacteremia caused by *R. ornithinolytica* in an infant with visceral heterotaxy.

A 16-month-old female child was brought to our hospital because of a persistent cough for 2 months and fever for 4 months. Body temperature purportedly increased 4 to 5 times per day, reaching a maximum of 38°C. When her body temperature increased, urticaria-like plaques occurred all over her body and were particularly prominent on the trunk and face. Recently, she had not been to an aquatic environment and had not consumed any fish. The hemogram, electrolyte values, liver and renal function, urinalysis, and an otoscopic examination were normal. Test result of C-reactive protein level was negative. No abnormalities were found in echocardiographic or ultrasonographic examinations. Virus, *Brucella*, and *Salmonella* serologies and investigations for tuberculosis were negative. Blood and urine cultures were sterile. *R. ornithinolytica* was isolated in bronchoalveolar lavage fluid, obtained by bronchoscopy at another hospital. Pathologic examination of biopsy material taken from cutaneous lesions showed urticaria. Aminopenicillin and clavulanate medication was administered, resulting in a decrease in body temperature to normal and resolution of the rash. The bronchoalveolar lavage culture was sterile when it was repeated. Stool cultures were obtained from all family members to identify the source of the microorganism, but no pathogenic organisms were isolated.

A systemic infection with *Raoultella* spp. should be suspected when making a differential diagnosis for diseases that present with similar signs and symptoms as our case. We emphasize that it is important to

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notify the laboratory workers when *Raoultella* spp. are suspected to be the cause of infection.

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Helicobacter pylori Seropositivity in Institutionalized Children With Down Syndrome

To the Editors:

Helicobacter pylori is usually acquired in early infancy and probably persists throughout the life.[1] Host genetic background, environment, and bacterial determinants contribute to the development of *H. pylori*-associated diseases.[2] Poor living and socioeconomic conditions as well as overcrowding are among the main risk factors for infection during childhood.[3] Children with Down syndrome (DS) have multiple immunologic defects.[4] It remains controversial whether abnormality of immune responses in such children increases the propensity to infections. We undertook a study to find *H. pylori* seropositivity among children with DS compared with age- and gender-matched children having other intellectual disabilities with similar nutritional regimen and living and hygienic environment.

Peripheral blood samples were collected from 24 children with DS and 24 children with other intellectual disabilities who were residents of 3 institutes for intellectually disabled children in Tehran. The paired match of each DS case was selected from other intellectual disabilities cases of the same institute, so the matched cases had similar living conditions. Serum *H. pylori*-specific immunoglobulin G (IgG) antibody was measured using ELISA (enzyme-linked immunosorbent assay) method, according to manufacturer's instructions (IMMUNOLAB GmbH, Germany). The results were considered positive if *H. pylori* IgG titer were >0.1 U/mL. For statistical analysis, χ^2 test and nonparametric tests (Mann-Whitney and Kruskal–Wallis tests) were used. A P value of ≤ 0.05 was considered statistically significant.

The DS and other intellectual disabilities groups each consisted of 13 females and 11 males, ranging in age from 1 to 15 years, and the mean age $\pm$ SD was 5.75 ± 4.35. *H. pylori*-specific IgG was detected in 17 of 24 (70.8%) DS children with a median titer of 30.09 U/mL. Among other intellectual disabilities children, 15 of 24 (62.5%) were seropositive with a median IgG of 34.34 U/mL. The seroprevalence of *H. pylori* infection in children with DS did not significantly differ from that in children suffering from other intellectual disabilities (odds ratio: 1.45; 95% confidence interval: 0.43–4.87). In the current small groups, seropositivity rates were similar in DS and other intellectual disabilities children in relation to age. More than half of seropositive children in DS (52.9%) and other intellectual disabilities (60%) groups were younger than 6 years, and this difference was not statistically significant. The median duration of institutionalization was 30 months (range, 6–96 months) for DS group and 24 months (range, 7–96 months) for other intellectual disabilities group. The antibody response against *H. pylori* was not associated with gender and duration of institutionalization.

The current study demonstrated high seropositivity for *H. pylori* in DS children. The high seropositivity may be attributed to the fact that overcrowding, sanitary conditions, and dietary regimen in childhood affect the route of *H. pylori* transmission and infection.[3] No significant difference was found in the risk of *H. pylori* seropositivity between children with DS and those with other intellectual disabilities. Two studies have reported *H. pylori* seropositivity in children with DS with lower rates. Jaber[5] assessed 24 children with DS in Saudi Arabia and found a 29.2% seropositivity rate. A study of 46 children with DS by Failla et al[6] showed that 19.5% of cases were seropositive. Both studies were of noninstitutionalized children with DS, whereas we examined children reside permanently in the institutes. Hence, the higher seropositivity of *H. pylori* infection might be explained by institutionalization and sanitary conditions as major risk factors. On the other hand, children with DS did not show a significantly higher susceptibility to *H. pylori* infection than children with other intellectual disabilities. It seems that host response against *H. pylori* infection is similarly influenced by environmental conditions in DS versus other intellectually disabled cases.

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