Correspondence and Replies

Genetic stability of *Campylobacter coli* in patients with primary antibody deficiencies

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To the Editor:

In the Clinical Communication, Dion et al[1] reported that in patients with severe primary antibody deficiency (PAD), *Campylobacter* infection is a major cause (6.5%) of chronic or recurrent diarrhea. Moreover, by a molecular study performed in a subset of 18 strains from 5 patients with recurrent infections, they demonstrated that all strains were different, even when the episodes occurred closely over time. Thus, the authors hypothesized that reinfection would be more likely than persistent colonization, although colonization with multiple strains cannot be excluded.

In a previous study,[2] our group showed that *Campylobacter coli* (*C. coli*) (6.7%) was the first cause of diarrhea in patients with symptomatic PAD with a positive stool culture, followed by *Campylobacter jejuni* (*C. jejuni)* (3.9%). Moreover, *C. coli* was also the most frequent isolate (5%) in patients with asymptomatic PAD, followed by *C. jejuni* (1.2%), whereas in immunocompetent individuals, *C. jejuni* is one of the most prevalent etiologic agents of gastroenteritis and *C. coli* has a low prevalence in diarrheal disease.[3,4]

Differently from the French data, we showed that the same *C. coli* strain has been isolated for more than 10 years in an asymptomatic patient with XLA.[2] As previously shown,[5] after this long-lasting silent infection, the same intestinal strain caused recurrent episodes of sepsis and leg cellulitis, without changing its molecular pattern. The same shift from an asymptomatic colonization to a life-threatening disease was observed in a patient with a Good's syndrome. All *C. coli* isolates, including the strains isolated from blood, stool, and skin lesion at the time of the sepsis and the stool strains isolated years before—at the time of the asymptomatic carriage—showed a 100% genetic homology as determined by pulsed field gel electrophoresis, amplified fragment length polymorphism, flaA-restricted fragment length polymorphism, and multilocus sequence typing.

In both patients, all antibiotic treatments were allowed to control the infection at the time of recurrent sepsis and only after a long course of oral treatment with bacitracin/neomycin stool cultures became persistently negative and there was no disease recurrence.

In summary, in our patients, all *C. coli* isolates were genetically identical, suggesting a long-lasting latent infection and not a reinfection nor colonization with different strains.[1] Differently from Dion et al,[1] we hypothesized that inability to mount a specific immune response would account for colonization and infection and for the genetic stability of *C. coli*. As we have previously suggested,[1] periodic stool cultures with highly sensitive techniques should be performed in PAD.

References

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