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Increased risk of Staphylococcus capitis sepsis in neonates previously treated with vancomycin

Marine Butin*[1], Jean-Philippe Rasigade[2], Fabien Subtil[3], Jean-Charles Picaud[4], Frederic Laurent[5]

[1]*Hospices Civils de Lyon; Nicu*

[2]*National Reference Center for Staphylococci, Hospices Civils de Lyon, Ciri Inserm U1111, University of Lyon*

[3]*Hospices Civils de Lyon; Department of Statistics*

[4]*Hospices Civils de Lyon; Neonatalogy*

[5]*Centre International de Recherche En Infectiologie - Hospices Civils de Lyon; Inserm U1111*

Background: Nosocomial sepsis is frequent in preterm infants hospitalized in neonatal ICUs (NICUs) and coagulase negative staphylococci are the most prevalent pathogens involved. The clone *S. capitis* NRCS-A has been shown to be endemic throughout the world and harbors a multiresistance profile including a decreased susceptibility to vancomycin as well as a rapid adaptation under vancomycin selective pressure *in vitro*. The pathophysiology of *S. capitis* infection has not been evaluated and we hypothesized that gut colonization and translocation could be the mechanisms involved in such infections. Aims: To determine if *S. capitis* gut colonization was a risk factor of *S. capitis* infection and to evaluate the other risk factors of *S. capitis* colonization/infection in NICU patients.

Material/methods: A prospective monocentric cohort study was conducted and included all the patients hospitalized in the NICU of Croix Rousse Hospital (Lyon, France) between June 2011 and January 2012. Weekly stool cultures were collected and incubated on home-made agar plates, selective for multiresistant *S. capitis* NRCS-A. Clinical data were exhaustively and prospectively collected using the ICCA software (Philips). The primary endpoint of the study was the occurrence of a *S. capitis* sepsis, defined by both clinical and bacteriological criteria (as recommended in the literature).

Results: 229 neonates were included. *S. capitis* was detected in 128 stool cultures (83 patients) over the 935 analyzed. A *S. capitis* sepsis was diagnosed in 28 patients (12%). Gut colonization with *S. capitis* was not a risk factor of subsequent *S. capitis* sepsis. The independent risk factors for developing a *S. capitis* sepsis were a prior administration of vancomycin (HR 6,44 [CI 95%: 2.15, 19.3] p=0.001) and the birth weight (HR 0,72 per 100g increase [CI 95%: 0.55, 0.95] p=0.020). A prior administration of vancomycin was also associated with an increased risk of *S. capitis* gut colonization. The risk of *S. capitis* sepsis was higher in patients who received vancomycin before developing a *S. capitis* colonization.

Conclusions: Vancomycin administration in neonates enhances the risk of colonization and infection by the non-vancomycin susceptible *S. capitis* clone. These results highlight the potential adverse effect of vancomycin, a high frequently used antimicrobial agent in neonates. Thus it seems urgent to optimize vancomycin use in NICUs, in order to avoid the selection of such vancomycin resistant strains for which alternative antibiotics lack in neonates. Moreover the higher risk of sepsis in patients receiving vancomycin before *S. capitis* gut colonization suggests a protective immune response in *S. capitis* colonized patients. This last hypothesis needs further explorations.