neutrophil count (ANC) of 500/mm$^3$. Thin smear revealed 2.5% parasitemia. Mother was diagnosed with acute Lyme disease in the seventh month of pregnancy. Maternal serologies were positive for $B. microti$ (IgM 1:100 and IgG 1:320). The infant received 1 PRBC transfusion and was treated with 10 days of atovaquone and azithromycin. Case 2 is a 5 week old female twin A admitted with 2 days of pallor, fatigue and poor feeding. She was treated with atovaquone and atovaquone for 10 days. The mother had an acute, self-limited febrile illness at 23 weeks gestation. At infant’s presentation, maternal serologies revealed negative $B. microti$ IgM and positive IgG (1:160). Placental tissue from both twins was positive for $B. microti$ DNA by PCR. Twin B was asymptomatic, had negative $B. microti$ blood PCR, a negative $B. microti$ IgM, positive IgG at 1:30 felt to represent transplacental maternal antibody, and did not require treatment.

Results: Both infants were successfully treated without relapse.

Conclusions: Congenital babesiosis is rare and may cause profound hematologic disturbances. We report 2 cases exhibiting neonatilia in addition to anemia and thrombocytopenia, supporting recent assertions by Wormser et al. that this is a common finding. In addition, Case 2 presented with a severe hemolytic anemia significantly worse than previously reported. Finally, we demonstrated successful treatment in infants without exchange transfusion, even with severe anemia.

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2590. Streptolysin O Enhances Binding of the Group A Streptococcus NAD+- Glycohydrolase Toxin to Oropharyngeal Keratinocytes

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Background: Streptolysin O (SLO) and the NAD$^+$-glycohydrolase (NADase) are co-toxins secreted by group A Streptococcus (GAS) that play a significant role in virulence. NADase requires SLO for translocation into the host cell cytoplasm, a process termed cytolsin-mediated translocation (CMT). Recently, we noted that interaction of the two toxins mutually increased their stability. Although NADase is predicted to bind to the host cell surface, this interaction is incompletely understood. Here, we investigate potential mechanisms by which NADase binds to oropharyngeal keratinocytes.

Methods: The amino terminal region of NADase has been implicated in CMT, but the structure of the native translocation domain has not been characterized. We determined the solution structure of this domain by NMR spectroscopy. We used flow-cytometry and confocal microscopy to investigate whether NADase could interact directly with oropharyngeal keratinocytes. Finally, since we expect that NADase and SLO are co-expressed from the same operon, are secreted in a coordinated fashion, and interact in solution, we tested whether SLO affects NADase binding to host cells.

Results: The solution structure of the NADase translocation domain revealed a $\beta$-sandwich fold with an elongated N-terminal intrinsically disordered region. Structural homology searches (DALI) identified a potential carbohydrate binding module, suggesting the translocation domain could play a role in glycan binding. We also demonstrated by flow-cytometry that purified recombinant NADase toxin is able to independently interact with the cell surface of oropharyngeal keratinocytes. Importantly, interaction with SLO significantly enhanced the association of NADase with the cell surface, resulting in a 5-fold increase of the geometric mean fluorescence intensity.

Conclusion: The structure of the NADase translocation domain reveals a potential carbohydrate binding module, which may mediate binding of the toxin to a cell-surface glycan. Binding of NADase to host cells is markedly enhanced by its interaction with SLO. We conclude that interaction of the two toxins contributes to the CMT process by functionally increasing the local concentration of NADase at the cell surface.

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2591. The Role of Neutralizing Antibodies (nAb) Against Cytomegalovirus (CMV) Epithelial Cell-entry in Patients with Self-limited (SL) CMV infection after Hematopoietic Cell Transplantation (HCT)

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Background: CMV transmission after HCT occurs in 20–30% of CMV-seronegative recipients with CMV-seropositive donors (i.e., CMV D+/R$-$) and a distinct subset develop transient CMV DNAemia without progression to culture positivity in the absence of antivirals (BMT 1992; 9:221; J Med Virol 2019; 91:1128). The mechanism of SL CMV infection is unknown but may involve nAb, which have been implicated in primary viral clearance, including nAb that inhibit viral epithelial and endothelial cell-entry via CMV pentameric complex (PC). We aimed to describe viral kinetics and the influence of nAb on CMV infection progression in SL CMV-infected patients and controls within a unique cohort from the pre-antiviral era.

Methods: Weekly serum samples from 456 CMV D+/R$-$ allogetic HCT patients collected between 1978–95 were screened using quantitative CMV DNA PCR. Patients with CMV DNAemia in the first 100 days after HCT following by a sustained return to undetectable levels without positive surveillance CMV cultures were defined as having SL CMV infection. SL CMV-infected patients were matched 1:1:1 to CMV-infected controls (patients with CMV infection by culture in the same period after HCT ±14 days) and non-infected controls (patients without CMV DNA or culture positivity) to compare viral kinetics and nAb.

Results: We identified 9 patients with SL CMV infection and baseline demographics are shown (Table 1). SL CMV-infected patients had a median of 21 days (range 7–54 days) until detectable CMV DNAemia. The mean peak CMV DNAemia was 94.2 IU/mL (range 40.2–225.9 IU/mL) and 46.18 IU/mL (range 1,132–284,362 IU/mL) in SL CMV-infected and CMV-infected controls respectively. There was no difference in nAb titers between groups at infection day 0 or in the preceding 4 weeks (Figure 1, Figure 2).

Conclusion: nAb against CMV PC-mediated cell entry did not appear to play a critical role in viral clearance in SL CMV infection after CMV D+/R$-$ allogetic HCT. Our study illustrates the potential for clearance of relatively low CMV DNAemia after HCT without the addition of antivirals. CMV-specific T-cell immunity or innate immune mechanisms may be more important in early viral control.

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