A new study looks to define the antibody characteristics, including clonality, of plasmablasts during Kawasaki Disease (KD). Findings from the study will be presented during the Pediatric Academic Societies (PAS) 2019 Meeting, taking place on April 24—May 1 in Baltimore.

"We still don't know the cause of KD, the leading cause of childhood acquired heart disease in developed nations," said Mark Hicar, MD, Ph.D., one of the authors of the study. "During a normal infectious immune response, special B cells called plasmablasts that are specific to the infection are found in the peripheral blood. We are characterizing these responses in a number of children with KD, have created antibodies from these plasmablasts, and are using these to identify the cause of KD."

Researchers used antibody repertoire next-generation sequencing to characterize memory and PB populations. Additionally, pairing of heavy and light chains was performed with Chromium Single Cell Gene Expression (10x Genomics, Pleasanton, CA) using the Human B cell Single Cell V(D)J Enrichment Kit.

From plasmablasts from subject 24, antibody sequences using VH4-34 and a 19 amino acid length complementarity determining region 3 showed a massive expansion between day four and six of fever. Chromium single cell sequencing produced over 946 heavy and light chain paired sequences. Sequence comparison showed 40% of sequences demonstrated markers of clonal expansion, which represented 100 clonal groups. One clonal group (24-01) reflected the massive clonal expansion (VH4-34, CDR3 19) previously shown within the next-generation sequencing data.

This clonal expansion within plasmablast populations supports that KD is caused by an infection. Antigen targeting of monoclonal antibodies from these clones is currently being explored.

More information: Dr. Hicar will present findings from "Clonal expansion within circulating plasmablast populations lends support for an infectious disease etiology of Kawasaki disease" on Monday, April 29 at 10:30 a.m. EDT.