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ISSN: 2329-6607
Cardiovascular Pharmacology: Open Access

Perspective

Role of Autoantibodies in Patients with Acute Rheumatic Fever

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DESCRIPTION

Acute Rheumatic Fever (ARF) is a serious multi-focal autoimmune sequela of Group A Streptococcal (GAS) infection that presents with a slew of signs and symptoms, including one or more of the major manifestations used in the Jones criteria for diagnosis: arthritis, carditis, Sydenham's chorea, erythema marginatum, and subcutaneous nodules. With an estimated 33 million people living with RHD worldwide, around 60% of ARF instances proceed to chronic Rheumatic Heart Disease (RHD), which can cause irreversible heart valve impairment. Despite the fact that ARF rates have decreased over the twentieth century, the disease still exists in low-income countries and among disadvantaged communities in some high-income countries, with Indigenous Maori and Pacific children in New Zealand and Aboriginal children in Australia having some of the highest rates in the world.

Multiple streptococcal infections cause the formation of IgG autoantibodies with a high avidity, resulting in ARF and RHD. This pathology does not arise as a result of the immune system's general immaturity, as Tandon believe, but rather as a result of the constant streptococcal infections that sustain the germinal center response and antibody affinity maturation. In ARF and RHD, however, large quantities of immune complexes would trap higher-affinity antibodies, allowing the germinal centers to continue feeding high-affinity autoantibody specificities into the blood, eventually causing valve damage. Prognosis is bad when antibodies against streptococcal group A carbohydrate continue to rise during ARF and RHD. The levels of autoantibody against the valve and group A carbohydrate decline only after valve replacement, along with healing and restoration of heart function. Human heart valves have long been known to bind to anti-group A carbohydrate antibodies.

To identify serum autoantibodies, human Protorrays (Protein microarray platform v5.0) were done according to the manufacturer's instructions. Antibody binding was detected with an Alexa Fluor 647 labelled goat anti-human IgG antibody after samples were diluted 1:500. The GenePix4000B microarray scanner was used to scan the arrays, and the GenePix Pro 5.0 software was used to align the array grids (Molecular Devices).

Raw data were background corrected using the Bioconductor limma package's "saddle" adjustment, and data were quantile standardized before differential expression statistical analysis using linear models and empirical Bayes statistics. Protein antigens with a fold-change of >2.0 and a P=0.05 were considered significant. The variants with the highest absolute fold-change were preserved for further analysis for proteins with duplicated IDs (proteins having more than one variation on the arrays).

The tidyverse suite of packages was used to do the analysis and visualizations in R studio19. The ComplexHeatmap software was used to create upset graphs. jvenn was used to create Venn diagrams. Morpheus was used to create heatmaps and hierarchical clustering (using the average Euclidean distance approach). Metascape was used to conduct a disease pathway study of differentially bound proteins utilizing custom analysis for enrichment in DisGeNet illness pathways. Using "Normal tissue data" retrieved from the human tissue atlas, the tissue specificity of proteins was determined HPA. The "reliability score" and "level" were used to filter data from the HPA.

Over 9000 human proteins produced in insect cells are included in the ProtoArrays used to characterize the autoantibody response in ARF. Because all people have autoantibodies, serum binding from ARF patients was compared to that of healthy children and children with GAS positive pharyngitis. The total antibody reactivity, or fluorescence intensity, was calculated for each array after array QC and normalization. In comparison to controls, ARF arrays revealed a higher number of total reactivates (P=0.0001), indicating an overall increase in autoantibodies in ARF patient serum. The total reactivity observed on the ARF arrays was significantly higher than in both the GAS positive pharyngitis and healthy controls, and the control groups were combined for the subsequent data analysis because the overarching goal was to identify ARF specific autoantibodies rather than those associated with GAS pharyngitis. Only proteins with a >2.0 fold increase in fluorescence intensity relative to the mean of the pooled controls were included in the antibody reactivity signals for ARF patients (healthy and GAS pharyngitis). This allowed autoantibodies with higher reactivity in ARF to be identified and the autoantibody profiles of individual ARF patients to be compared.

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Received: 03-Jun-2022, Manuscript No. CPO-22-17185; **Editor assigned:** 06-Jun-2022, PreQC No. CPO-22-17185 (PQ); **Reviewed:** 20-Jun-2022, QC No. CPO-22-17185; **Revised:** 27-Jun-2022, Manuscript No. CPO-22-17185 (R); **Published:** 04-Jul-2022, DOI: 10.35248/2329-6607.22.11.290

Citation: Tontsch R (2022) Role of Autoantibodies in Patients with Acute Rheumatic Fever. Cardiovasc Pharm. 11:290