Rheumatic Heart Disease (RHD) is associated with a proteomic signature that indicates persistent inflammation.

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Abstract

In poor countries, Rheumatic Heart Disease (RHD) is still a major cause of illness and mortality. A better understanding of the pathogenetic mechanisms behind RHD could lead to drug repurposing, secondary penicillin prophylaxis recommendations, and/or the development of near-patient diagnostics. Logistic Regression was used to calculate the case–control differences and contribution to Area under the Receiver Operating Curve (AUC) for each of the 56 proteins discovered by the Boruta method, after adjusting for age, sex, and BMI. ClueGo pathway analysis was used to find biological pathways and activities that were enriched for proteins. These findings confirm the existence of a persistent inflammatory response in RHD, which occurs at a time when serious valve disease has established and is distinct from previous episodes of acute rheumatic fever.

Keywords: Rheumatic fever, Mitral stenosis, Plasma proteomics, Inflammation, Immunotechniques.

Introduction

This biomarker profile should be useful in identifying distinct levels of continuing inflammation in RHD patients, which could be linked to prognostic severity. Damage to the cardiac valves caused by an immunological reaction to Group A Streptococcal infection is the leading cause of morbidity and mortality in rheumatic heart disease (RHD) (typically, childhood sore throat). RHD is the only global cardiovascular disease that has been proven to be entirely avoidable. RHD, which continues to be a major source of illness and mortality in low and middle-income nations, is aided by poor socioeconomic conditions, overcrowding, and restricted access to medical resources (LMICs) [1]. RHD affects around 40 million people globally, the majority of whom live in countries with modern medical technology as percutaneous or surgical intervention are not accessible [2].

According to the Global Burden of Disease survey, RHD affects about five million more individuals than HIV and results in the loss of about ten million disability adjusted life years worldwide. RHD manifests a wide range of symptoms and indications, and there is no single conclusive laboratory test available; this complicates the identification and treatment of early RHD cases. Given the human and financial costs of failing to diagnose the disease until late in its advancement, a better knowledge of the basic basis of ARF and its subsequent progression could lead to new preventative and treatment approaches [3].

This study aimed to complement our recent GWAS study that confirmed an association between RHD and genetic susceptibility loci in African individuals by identifying a plasma protein signature of RHD that could aid biological understanding of the processes involved and potentially point to economically feasible interventions to prevent severe RHD in poorer countries based on repurposing of readily available and inexpensive medicines [4].

While pure mitral regurgitation predominates in the young, mixed valvular disease is the most common finding in chronic RHD, indicating progression, according to clinical observation. Our findings imply that these clinical alterations are due to continuous inflammation-driven valve scarring and remodelling in RHD, even in the absence of recurrent ARF episodes. Furthermore, while such valve tissue studies provide immediately pathologically relevant information, they may not always provide the foundation for a future field diagnostic. The proteins that revealed the most significant alterations between cases and controls are discussed next, as well as their possible relation to RHD [5].

Conclusion

In conclusion, we discovered a rheumatic heart disease plasma protein signature that indicates a continuous inflammatory activity in the chronic phase of the disease. A modest number of proteins combined correctly distinguished chronic, severe RHD sufferers from healthy controls. This research could lead to drug repurposing options, prophylactic suggestions, and/or the development of near-patient diagnostics.

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