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ADVANCES IN THE CO-HOST IMMUNE RESPONSE TO MULTISYSTEM INFLAMMATORY SYNDROME AND KAWASAKI DISEASE IN CHILDREN WITH AI-GUIDED FEATURES

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Objective. To explore the role of artificial intelligence in the immune response mechanism of children with multi-system inflammatory syndrome and Kawasaki disease. **Method.** To search the domestic and foreign literatures about the immune response mechanism of these two diseases, and analyze the literatures according to the characteristics of artificial intelligence. **Result.** AI analysis showed that the two kinds of children's syndrome were concentrated in the cytokine storm centered on il-15/IL15RA, which confirmed that the two diseases had the same initial immune pathway, but the differences in immune phenotype, cytokine, cell count and other aspects suggested that KD and MIS-C were two different diseases. Conclusion.It shows the applicability of AI in this research direction, and points out the limitations of the current research scope and samples. The difference between the results of KD and MIS-C research guides the direction of future research. Accurate and comprehensive laboratory indicators and parameters can be applied to artificial intelligence and provide basis for diagnosis and treatment of diseases. As the number of infected people increases, the problem of sample limitation in the current work can also be improved.

Background. In April 2020, children with symptoms similar to incomplete Kawasaki disease (KD) or toxic shock syndrome were reported in the UK. The disease was later defined as coronavirus-associated multisystem inflammatory syn-drome in children (MIS-C), which can be fatal in severe cases. Children's multisystem inflammatory syndrome (MIS-C) and Kawasaki disease are highly inflammatory diseases related to infectious diseases, but are they different syndromes or continuous?

Content. The SARS - CoV - 2 pandemic has inspired many research groups to analyse more than 45000 pandemic transcriptome datasets ,166 gene signatures were extracted with ACE2 as the "seed" gene, and ViP and severe ViP features were named. AI analysis showed that the two kinds of children's syndrome were concentrated in the cytokine storm centered on il-15/IL15RA, which confirmed that the two diseases had the same initial immune pathway. Prolonged and excessive IL-15 stimulation can lead to significant depletion and reduction of NK cells in severe COVID-19 infection cases, and this reduction occurs as early as 6 days after symptoms appear. We conclude that fatal COVID-19 is characterized by a contradictory immune response, that is, inhibiting the function of epithelial cells and NK cells in the context of cytokine storm (excessive immune response) (immunosuppression). The results showed that: (1) compared with KD, the level of cytokine in patients with misc was higher, the decrease of whole blood cells was more serious, and the host immune response of MISC was significantly higher than that of KD; (2) Misc has the key distinguishing characteristics of thrombocytopenia and low eosinophil count, and both of these characteristics are negatively correlated with serum

il-15 and VIP levels. Eosinophilia seems to be a significant common feature between misc and COVID-19, but not KD. These findings are consistent with the fact that KD is known to exhibit higher (rather than lower) eosinophil counts. Thrombocytopenia has been shown to be significantly associated with mortality. Like thrombocytopenia, persistent eosinophilia after admission is associated with the severity and low recovery rate of COVID-19. (3) Misc had impaired cardiac contractility, but KD did not. These two kinds of pediatric syndromes focus on the cytokine storm centered on il-15/il15ra, suggesting that there is a common proximal pathway for immune pathogenesis. However, they differ in other laboratory parameters (platelets, eosinophils) and cardiac phenotype (cardiac function decline, coronary artery dilation). These relevant clinical/laboratory parameters (low PLT and AEC) may be useful indicators of disease severity and prognosis, and can be used to guide hospital treatment and nursing decisions.The above studies confirmed that MIS-C and KD have a common initial immune pathway, but in cytokines (IL-17A increased in Kawasaki disease and significantly decreased in misc patients), T cell subsets (CD4 and CD8 counts in Kawasaki disease were higher than misc,), immunophenotypes (CD57 markers were higher in misc), antibodies (the overexpression of EDIL3 autoantibodies in Kawasaki disease was the most obvious, and CSNK and MAP2K2 family proteins were highly expressed in MIS-C) There are differences in blood coagulation status (fibrinogen increase, d - dimer increase and platelet decrease in acute phase of Misc, not in Kawasaki disease), cell count (neutrophil and platelet increase in KD, and eosinophil count decrease in misc), etc. which suggested that KD and MIS-C were two different diseases.

Conclusion. In the future, we hope to find more extensive and accurate gene features in existing studies (related to cytokines, cellular immunophenotypes, antibodies, gene susceptibility, etc.), use artificial intelligence algorithms to define and layer diseases from clinical or laboratory parameters, and identify the phenotypes and complications of disease spectrum, including cardiogenic shock (such as MIS-C shock and Kawasaki disease shock syndrome), MAS (cytopenia and coagulation dysfunction related to cytokine storm caused by infection), Kawasaki disease (typical and complete Kawasaki disease phenotype, caused by SARS - CoV-2 or other infectious factors), and provide evidence for the formulation of treatment strategies. The amount of research data should be further expanded in clinical practice, and the special method of artificial intelligence should be used to diagnose, differential diagnosis and treatment of the above diseases to serve the clinical.