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Perspective

The SFTS Thrombocytopenia Mechanism

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DESCRIPTION

A new viral hemorrhagic fever condition called Severe Fever with Thrombocytopenia Syndrome (SFTS) is transmitted by ticks and is brought on by infection with the Dabie bandavirus (SFTS virus, SFTSV). The main clinical manifestation of SFTS is thrombocytopenia, which has a strong correlation with illness severity. Uncertainty persists regarding the pathogenic mechanism of thrombocytopenia in SFTS.

RNA transcriptome analysis were performed on platelets that had been isolated from SFTS patients. The functions and transcriptional levels of Differentially Expressed Genes (DEGs) in the platelets of deceased and living individuals were described. The platelets of COVID-19 and dengue fever patients were compared with DEGs associated with cell death. Using flow cytometry, it was possible to calculate the proportion of platelets that were positive for the biomarkers of pyroptosis, apoptosis, necroptosis, autophagy, and ferroptosis. Transcriptome of RNA Additionally, investigations using platelets extracted from non-lethal SFTSV infection model mice were carried out. Platelet death was also looked at, and DEGs reflecting the functional alterations in mouse platelets were described. To identify the various processes producing thrombocytopenia in people and animals, functional platelet alterations in SFTS patients and SFTSV-infected mice were studied.

The platelet response to SFTSV infection in mice and people was described in this work. The molecular processes underpinning platelet functioning and death are still a mystery, and further research is needed to fill in the information gaps about the disparities in reactions between people and mice. This study has drawbacks as well. Platelets isolated from four patients who died and four patients who survived were randomly chosen for RNA transcriptome analysis. SFTS sufferers who have been identified. As a result, their individual variances may also have an impact on the platelet response, as indicated by the data collected from their platelets. It was difficult to purify enough platelets from the blood samples of the fatal patients to match those purified from the blood samples of the surviving patients due to the substantial loss of platelets in fatal patients. By adding all fatal cases, we were unable to investigate the relationship between viremia and necroptosis, autophagy, and ferroptosis in the platelets. Our findings still provide important information for future studies on platelet activity and the creation of illness treatment methods that control platelet activity.

For surviving of SFTS patients' platelets with pure RNA transcriptome analysis, for the randomly chosen groups of healthy donors, fatal patients, and healthy donors all had distinct gene expression patterns. In comparison to the healthy donors, we discovered 146 upregulated genes and 59 downregulated genes in the fatal patients, and 467 upregulated genes and 2 downregulated genes in the surviving patients. The (Diethylene glycol) DEGs in the platelets of patients who died appeared to be involved in type I interferon signaling, control of the viral cycle, neutrophil activation and neutrophil-mediated immune response, according to Gene Ontology (GO) functional analyses. While the elevated DEGs revealed heightened interferon-mediated signaling pathways and viral infection, the downregulated DEGs were connected to the transport protein particle complex and indicated decreased protein transport in platelets. Replication of the genome.

Genes related to immunological response, viral transcription, neutrophil activity, and survival were elevated in the survivors. Using platelet transcriptome analysis, this study examined the altered platelet response to SFTSV infection in people and a nonlethal mouse model. It also identified potential pathways that can encourage platelet loss, which would then result in the usual thrombocytopenia symptoms of SFTS. The findings contribute to a better understanding of the etiology of SFTS and offer important data for future research into the molecular causes of thrombocytopenia as well as the creation of targeted treatment plans. Furthermore, while assessing the impact of antiviral medications that modulate platelet activities using animal models, it is important to take into account the difference from human platelet response in animals. Our results understanding of the active adaptive response in nonlethal mouse platelets may offer clues for the creation of novel SFTS therapeutic approaches.

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