In the coronavirus family, there are currently seven different viruses, initially known as common cold viruses. This classification changed with the emergence of severe acute respiratory coronavirus (SARS-CoV) in 2002, Middle East respiratory syndrome coronavirus (MERS) in 2012, and severe acute respiratory coronavirus 2 (SARS-CoV-2), which caused the global COVID-19 pandemic in 2019. Utilizing bioinformatic methods, we investigated potential interactions between human miRNAs expressed in pulmonary epithelial cells and available coronavirus genomes. We compared putative miRNA binding sites between pathogenic and non-pathogenic virus groups. The pathogenic group shares six miRNA binding sites that may be involved in sequestering miRNAs associated with deep vein thrombosis. Additionally, we analyzed approximately 100,000 SARS-CoV-2 variant genomes for potential interactions with human miRNAs. This analysis revealed a group of 97 miRNA binding sites present in all analyzed genomes. Among these, six miRNA binding sites were specific to SARS-CoV-2 and other pathogenic viruses, with down-regulation linked to deep vein thrombosis and cardiovascular diseases. Notably, one of these miRNAs, miR-20a-5p, whose expression decreases with age, is involved in cytokine signaling, cell differentiation, and/or proliferation. We hypothesize that the depletion of poorly expressed miRNAs could be related to disease severity."