To date the coronavirus family is composed of seven different viruses which were commonly known as cold viruses until the appearance of the severe acute respiratory coronavirus (SARS-CoV) in 2002, the middle east respiratory syndrome coronavirus (MERS) in 2012 and the severe acute respiratory coronavirus 2 (SARS-CoV-2) which caused the COVID-19 global pandemic in 2019. Using bioinformatic approaches we tested the potential interactions of human miRNAs, expressed in pulmonary epithelial cells, with the available coronavirus genomes. Putative miRNA binding sites were then compared between pathogenic and non pathogenic virus groups. The pathogenic group shares 6 miRNA binding sites that can be potentially involved in the sequestration of miRNAs already known to be associated with deep vein thrombosis. We then analysed ∼100k SARS-CoV-2 variant genomes for their potential interaction with human miRNAs and this study highlighted a group of 97 miRNA binding sites which is present in all the analysed genomes. Among these, we identified 6 miRNA binding sites specific for SARS-CoV-2 and the other two pathogenic viruses whose down-regulation has been seen associated with deep vein thrombosis and cardiovascular diseases. Interestingly, one of these miRNAs, namely miR-20a-5p, whose expression decreases with advancing age, is involved in cytokine signaling, cell differentiation and/or proliferation. We hypothesize that depletion of poorly expressed miRNA could be related with disease severity.