Severe coronavirus disease 2019 (COVID-19) is characterized by hyperinflammation, multiorgan injury, and thrombotic complications. Neutrophils have been implicated in COVID-19 pathogenesis through the release of pro-inflammatory cytokines and neutrophil extracellular traps. Fibrin(ogen) and plasmin(ogen) can bind to neutrophils and regulate their pro- and/or anti-inflammatory activities, which may influence COVID-19 severity. The present study aimed to elucidate the contribution of fibrin(ogen)- and plasmin(ogen)-neutrophil interactions to COVID-19 severity and mortality. Here we analyzed male and female 20-week-old C57BL/6J mice sufficient or deficient in fibrinogen (*Fga*$^{+/-}$, *Fga*$^{-/-}$) or plasminogen (*Plg*$^{+/-}$, *Plg*$^{-/-}$) that were mock-infected or infected with mouse-adapted SARS-CoV-2 (MA10) virus. Using immunohistochemistry, we identified and quantified neutrophils in lung tissue harvested 5 and 30 days post-infection (dpi). Compared to MA10-infected male *Fga*$^{+/-}$ mice, MA10-infected male *Fga*$^{-/-}$ mice had higher survival, but no difference in lung neutrophil counts 5 or 30 dpi. All MA10-infected female *Fga*$^{+/-}$ and *Fga*$^{-/-}$ mice survived, despite MA10-infected *Fga*$^{-/-}$ mice having significantly fewer lung neutrophils 5 dpi (P=0.017). Compared to MA10-infected male *Plg*$^{+/-}$ mice, MA10-infected male *Plg*$^{-/-}$ mice had higher survival, a more gradual death rate, and increased lung neutrophils 5 and 30 dpi. All female *Plg*$^{-/-}$ mice survived and had fewer lung neutrophils than males 5 dpi, despite sharing similar lung neutrophil count trends. Following MA10 infection, deficiency in fibrinogen or plasminogen was associated with increased survival. Neutrophil counts in the lung were not predictive of survival. The potential role of neutrophils in COVID-19-associated sex-dependent differences in mortality remains unclear.