Supplementary Figure S1. The scatter plot of HBP for the two groups of patients in Table 1, including the respiratory failure group and the critical COVID-19 group.

Supplementary Figure S2. Correlation analysis between blood gas index and HBP. (PA-aDO2: Arterial alveolar oxygen differential; Qsp: Intrapulmonary shunt volume; ABE: Actual Base Excess; SBE: Standard Base Excess; Spiro-index: Respiratory index; OI: Oxygenation index).

Supplementary Figure S3. Longitudinal trend of changes of myocardial test indexes in COVID-19 patients, and the CCF between myocardial test indexes and HBP. Again, the correlation between myocardial test indexes and HBP is highest with a lag of 5 days (AST; $r=0.499$, $p=0.1179$; CK: $r=0.848$, $p<0.05$; CK-MB: $r=0.438$, $p=0.1983$; cTnI: $r=0.524$, $p=0.0982$; MB: $r=0.789$, $p<0.05$). Abbreviation: AST: Aspartate Aminotransferase; CK: Creatine kinase; CK-MB: Creatine kinase isoenzymes; cTnI: cardiac troponin I; Mb: myoglobin.

Supplementary Figure S4. Trends for liver and kidney function indexes, and CCF analysis. There is a strong positive correlation between BUN, Cr and HBP. The correlation is again highest with a 5 days difference (BUN: $r=0.684$, $p<0.05$; Cr: $r=0.714$, $p<0.05$), but K and HBP do not show any strong positive correlations.

Supplementary Figure S5. The mechanism of increasing capillary leakage induced by HBP. HBP is stored in vesicles in PMN near the cell membrane and is released when they are activated. HBP binds to and internalizes the surface polysaccharide receptor of endothelial cells to activate protein kinase C (PKC) and Pho kinase (PhoK) [53]. PKC in turn activates voltage-gated calcium channels, facilitating $Ca2+$ influx, increasing concentrations of which result in altered actin alignment and PKC also phosphorylates cytoskeletal proteins directly and causes contraction [54].