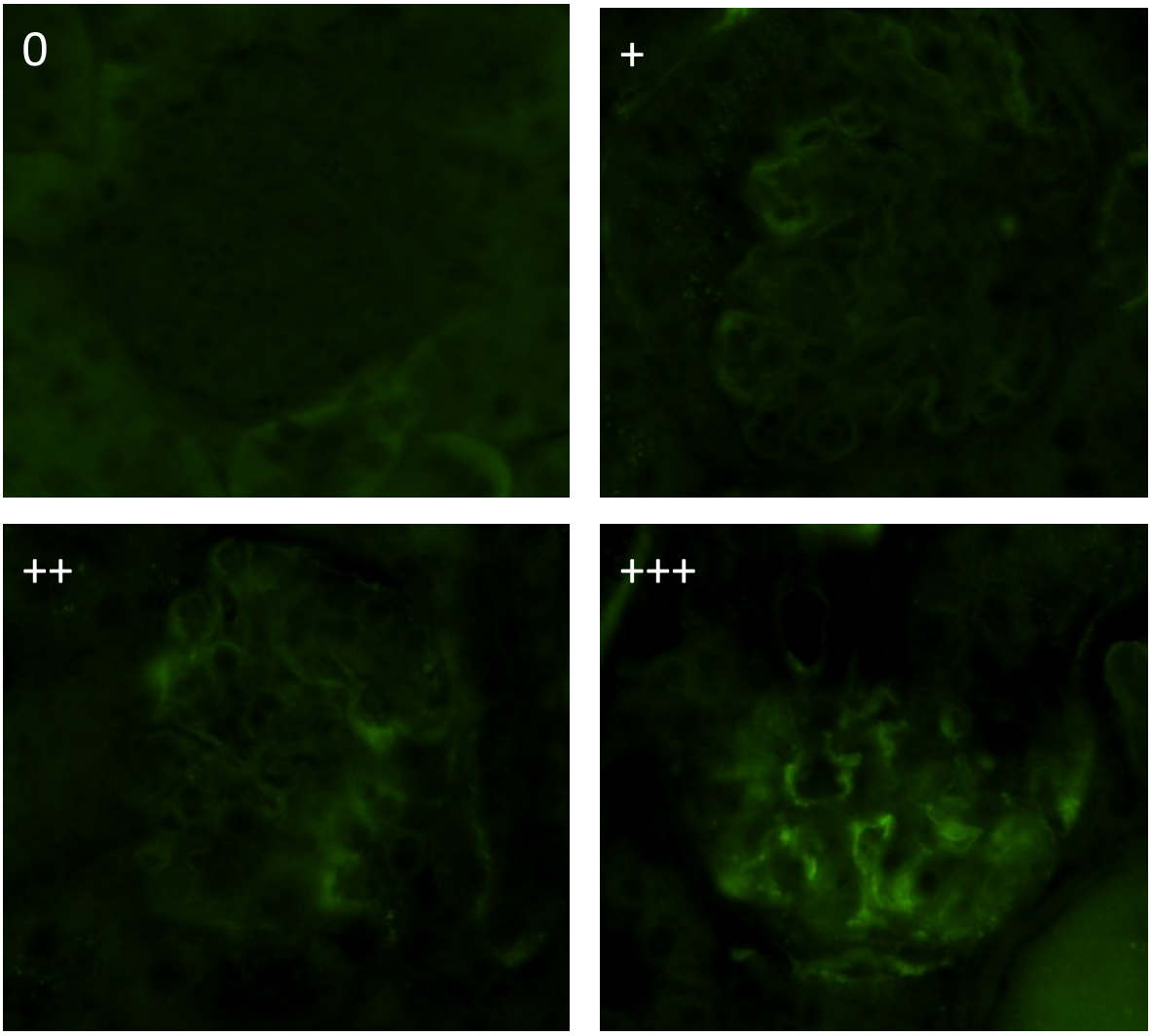

Figure S1: Kidney sections from mice with glomerulonephritis stained for complement C3 were graded after the intensity of fluorescence

Renal tissue from mice either treated with the kinin B1-receptor antagonist (n=5), or left untreated (n=3) were analyzed by immunofluorescence for the presence of C3 deposition. Sections were visualized using a super resolution microscope system (Nikon Ti-E microscope with N-SIM E), each glomerulus was counted and the level of intensity was graded using a scoring system of no staining (0), low (+), medium (++), or high (+++) intensity. This figure was used as a template.

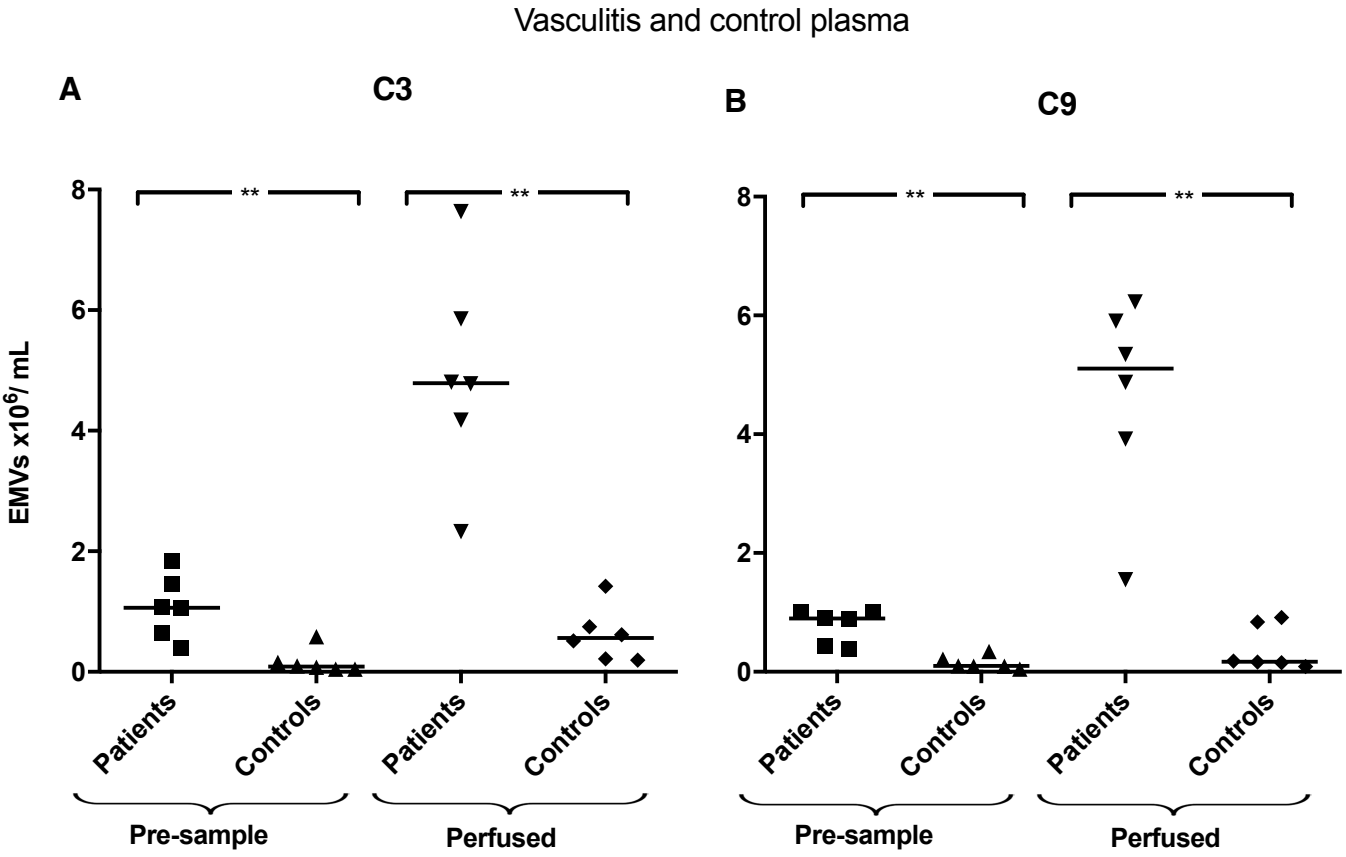

Figure S2: Release of C3- and C9-positive endothelial microvesicles from primary glomerular endothelial cells after perfusion of plasma from vasculitis patients and controls

Patient samples (Patients 7-9, 12, 19, and 22 in Table 1) were perfused over primary glomerular endothelial cells (PGECs) and shed endothelial microvesicles (EMVs) positive for C3 and C9 were detected by flow cytometry. The figures show the amount of EMVs in the sample before perfusion (pre-sample) as well as after perfusion (perfused sample). The values correspond to Figure 2A-B in the paper. A) Vasculitis patient samples induced the release of significantly higher levels of C3-positive EMVs compared to controls both before and after perfusion. Median EMVs released in controls, pre-sample 0·09 $x10^6$ and perfused sample 0·56 $x10^6$. B) Patient plasma induced the release of significantly more C9-positive EMVs than control samples before and after perfusion. Median EMVs released in controls, pre-sample 0·10 $x10^6$ and perfused sample 0·17 $x10^6$, **: P <0·01. Pre-sample: Pre-perfusion sample, EMVs: endothelial microvesicles. The median is represented by the bar. Samples were analyzed using a FACSCanto Cytometer.

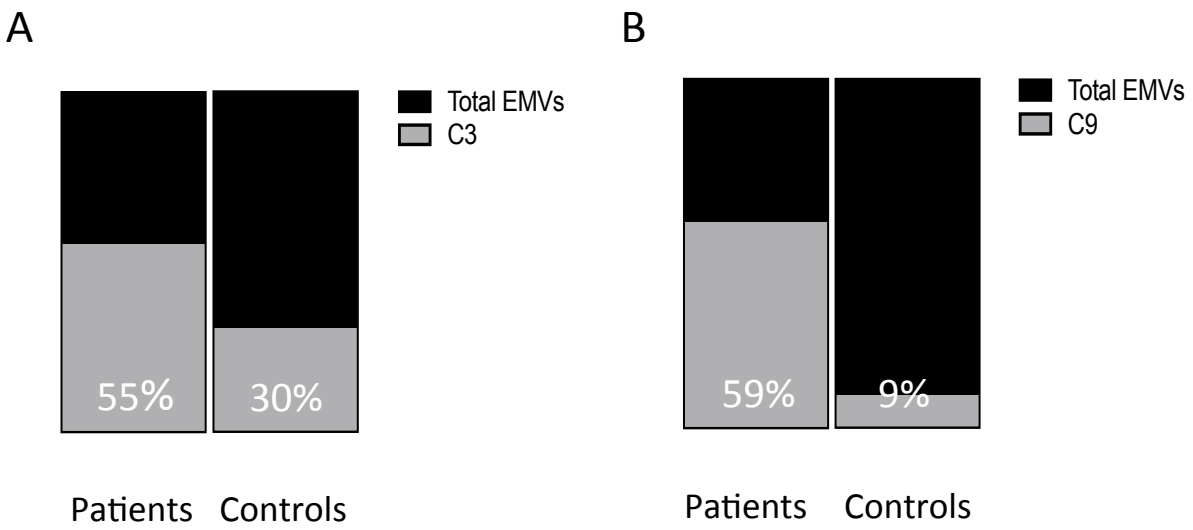

Figure S3: Percentage of C3- and C9-positive endothelial microvesicles in patient plasma and control plasma

Plasma from patients (n=6 Patients 7-9, 12, 19, and 22 in Table 1) and controls (n=6) perfused over primary glomerular endothelial cells (PGECs) induced the release of complement-positive endothelial microvesicles (EMVs). A) The percentage of C3-positive EMVs was higher in patient samples, compared to controls. B) A greater proportion of C9-positive EMVs were detected in patient samples than controls. EMVs: endothelial microvesicles.

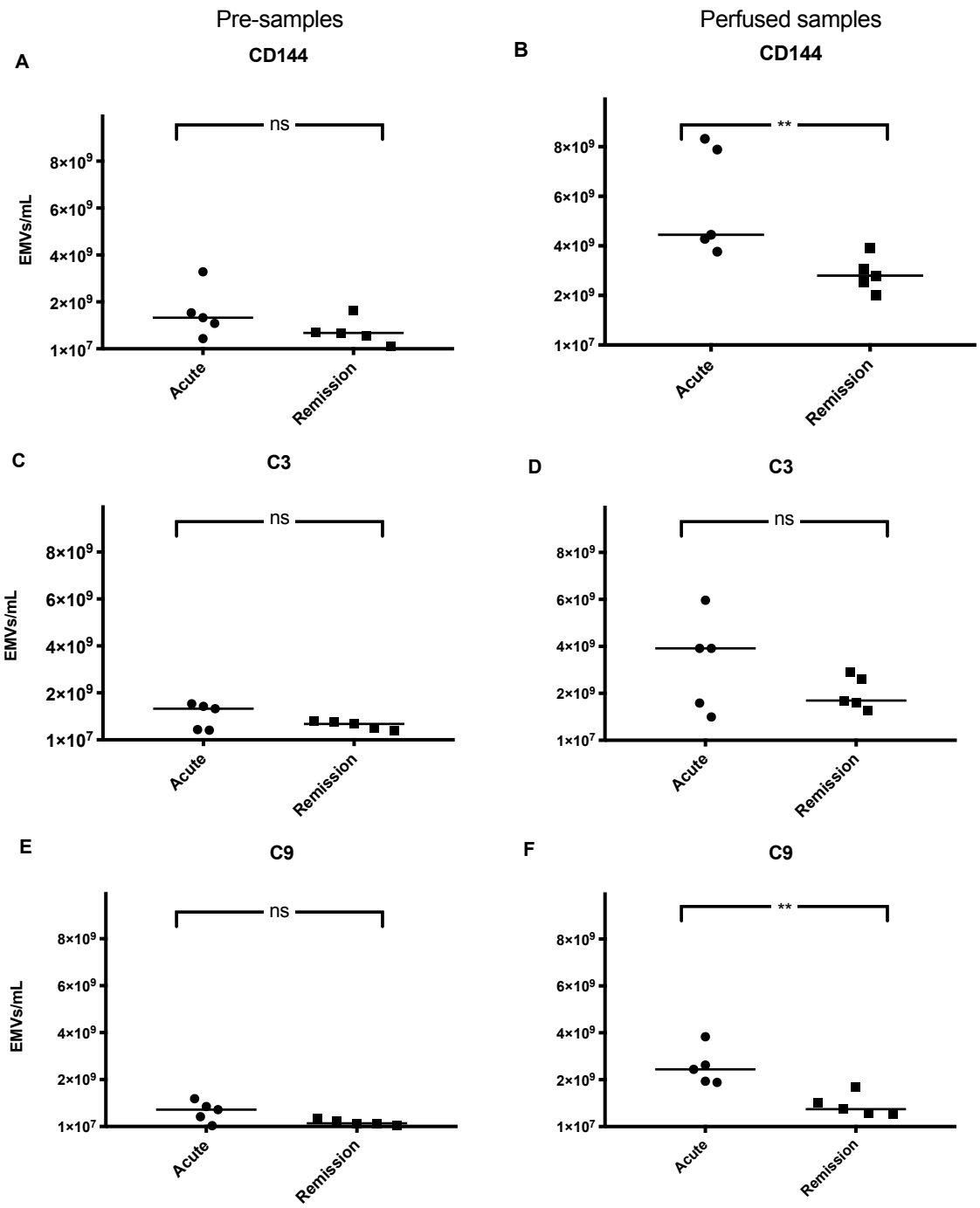

Figure S4: Release of C3- and C9-positive endothelial microvesicles from primary glomerular endothelial cells after perfusion of plasma from vasculitis patients during the acute phase of disease and in remission

Vasculitis patient plasma samples (Patients 1-5 in Table 1) were perfused over primary glomerular endothelial cells (PGECs) using a flow system. Endothelial microvesicles (EMVs) positive for C3 and C9 were measured by flow cytometry in both the pre-perfusion sample as well as in the perfused sample. The median is represented by the bar. Values correspond to Figure 2C-E in the paper. A) The pre-perfusion sample taken during the acute phase of disease did not release more EMVs compared to the samples during remission. B) The perfused samples from the same vasculitis patients during the acute phase of disease released significantly more EMVs compared to the same patients in remission. C-D) The acute samples had C3 deposits on the EMVs in both the pre-perfused sample as well as in the perfused sample, but not significantly more than at remission. E) The acute samples did not exhibit more C9-positive EMVs compared to samples from remission in the pre-perfused samples. F) The acute samples exhibited significantly more C9-positive EMVs compared to samples from remission in the perfused samples. **: $P < 0{\cdot}01$. ns: not significant. EMVs: endothelial microvesicles. The bar depicts the median. Samples were analyzed using a CyFlow Cube 8 flow cytometer.

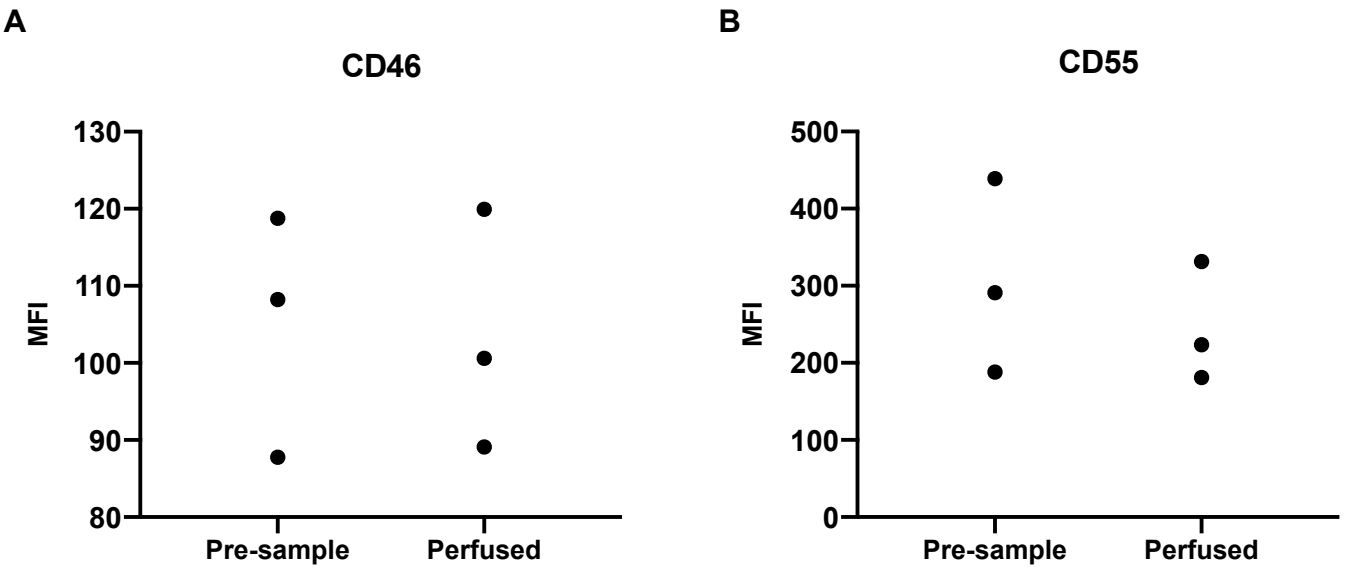

Figure S5: Perfused patient samples did not differ in expression of cell-bound complement regulators
Patient samples (n=3) were analyzed for expression of cell-bound complement regulators CD46 and CD55 on endothelial microvesicles (EMVs) before and after perfusion over primary glomerular endothelial cells (PGECs). No differences were detected. MFI: mean fluorescent intensity.

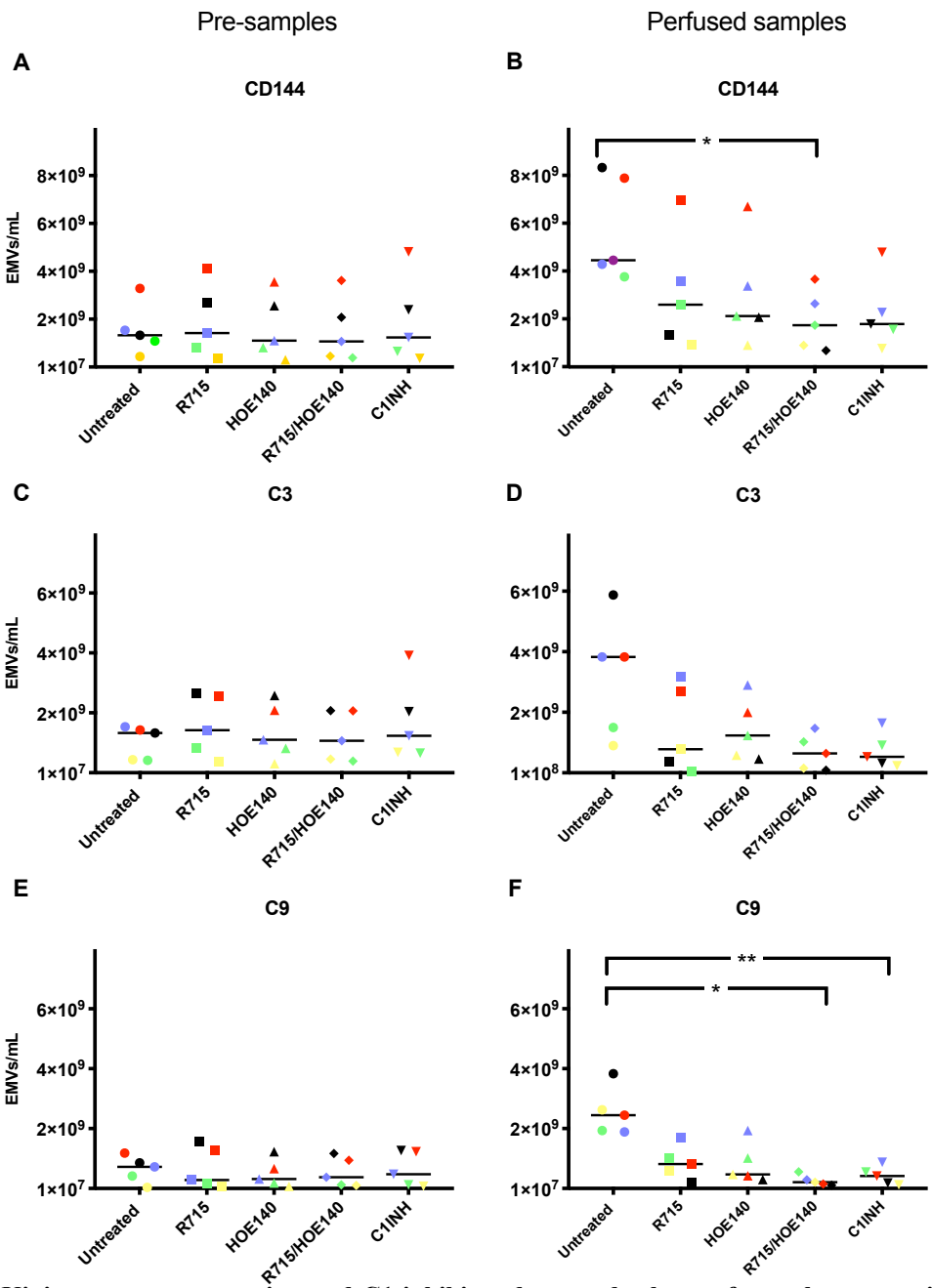

Figure S6: Kinin-receptor antagonists and C1-inhibitor decreased release of complement-positive endothelial microvesicles from primary glomerular endothelial cells

Plasma from vasculitis patients (Patient 1-5 in Table 1) was perfused over primary glomerular endothelial cells (PGECs) in the presence of the kinin B1R antagonist R715 and the B2R antagonist HOE-140 (alone or in combination), or C1-inhibitor. Endothelial microvesicles (EMVs) in the plasma as well as EMVs positive for C3 and C9 in both the pre-perfusion and perfusion samples were measured by flow cytometry, and individual patient samples are marked in color. Values correspond to Figure 3 in the paper. A, C, and E) In the pre-perfusion samples no difference in EMV levels (total as well as C3- and C9-positive) could be measured between the untreated sample and the samples with kinin-receptor antagonists or C1-inhibitor. B) Adding kinin-receptor antagonists in combination significantly reduced the total amount of EMVs released in the perfused sample. D) The release of C3-positive EMVs in the perfused sample was reduced by pre-treating the plasma with kinin-receptor antagonists or C1-inhibitor, but did not achieve statistical significance. F) The release of C9-positive EMVs was significantly reduced by adding kinin-receptors antagonists in combination or C1-inhibitor before perfusion. **: P <0·01. *: P <0·05. ns: not significant. EMVs: endothelial microvesicles, C1INH: C1-inhibitor. The median is represented by the bar. Samples were run using CyFlow Cube 8 flow cytometer.

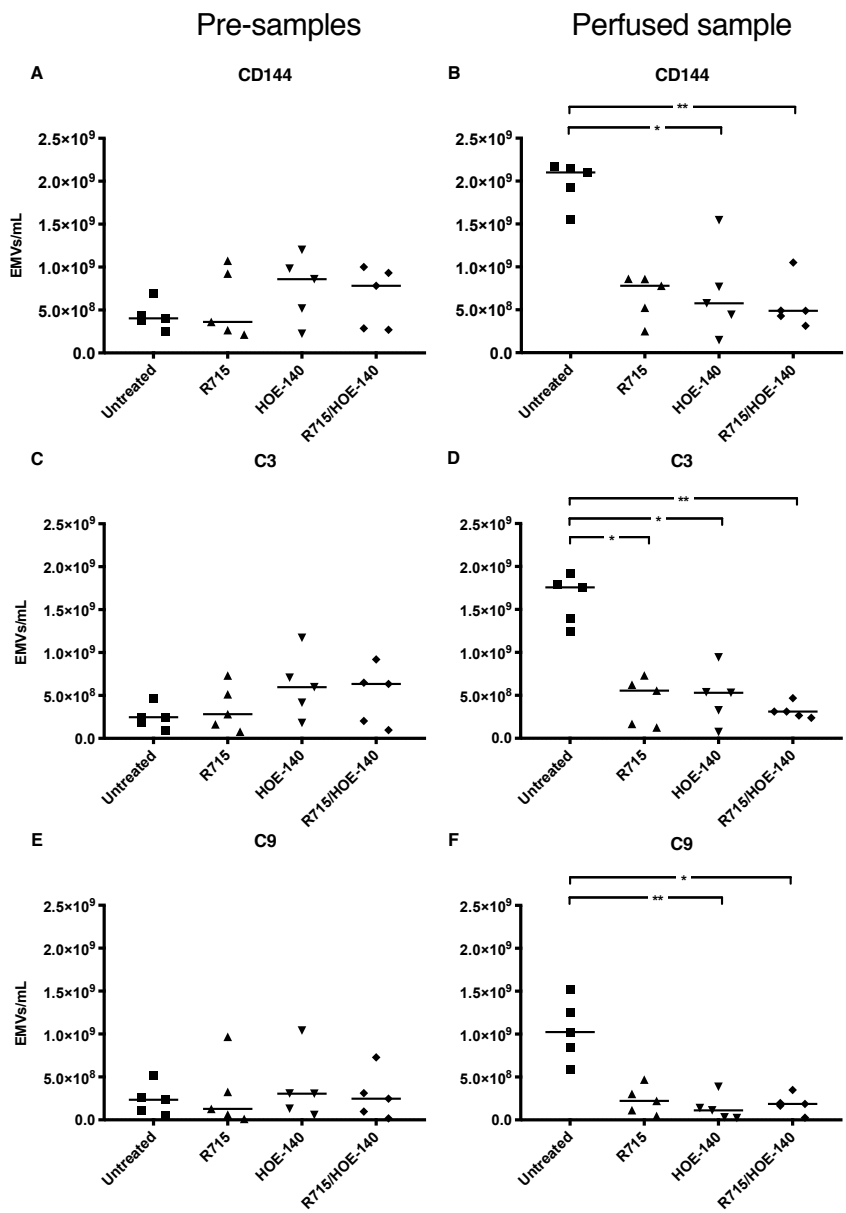

Figure S7: Kinin-receptor antagonists reduced release of C3- and C9-positive endothelial microvesicles after perfusion of C1-inhibitor-depleted plasma over primary glomerular endothelial cells

C1-inhibitor-depleted plasma was perfused over primary glomerular endothelial cells (PGECs) alone or after addition of the kinin B1-receptor(B1R) antagonist R715, the B2-receptor(B2R) antagonist HOE-140, or both antagonists combined and C3- and C9-positive endothelial microvesicles (EMVs) were detected by flow cytometry. The EMVs were measured both before perfusion (pre-perfusion sample) as well as after perfusion (perfused sample). Values correspond to Figure 4 in the paper. A, C, and E) In the pre-perfusion samples no difference in EMV levels (total as well as C3- and C9-positive) could be measured between the untreated sample and the samples treated with kinin-receptor antagonists. The lowest EMV values in untreated samples were $2{\cdot}5 \times 10^8$, $9{\cdot}0 \times 10^7$ and $4{\cdot}8 \times 10^7$ in panels A, C, and E, respectively. B) The total amount of EMVs released from PGECs perfused with C1-inhibitor-depleted plasma after addition of the B2R antagonist HOE-140, alone or in combination with the B1R antagonist R715, was significantly reduced, compared to cells perfused with C1-inhibitor-depleted plasma alone. The B1R antagonist R715 alone did not significantly reduce released EMVs from the cells. D) PGECs exposed to C1-inhibitor-depleted plasma in the presence of either or both the B1R- and B2R antagonists exhibited significantly lower C3-positive EMVs. F) PGECs exposed to C1-inhibitor-depleted plasma exhibited less C9 on the released EMVs when the plasma was combined with the B2R antagonist, alone or in combination with the B1R antagonist, but the reduction was not significant using the B1R antagonist alone. EMVs: endothelial microvesicles. **: $P < 0{\cdot}01$, *: $P < 0{\cdot}05$. The median is represented by the bar. Samples were run using CyFlow Cube 8 flow cytometer.

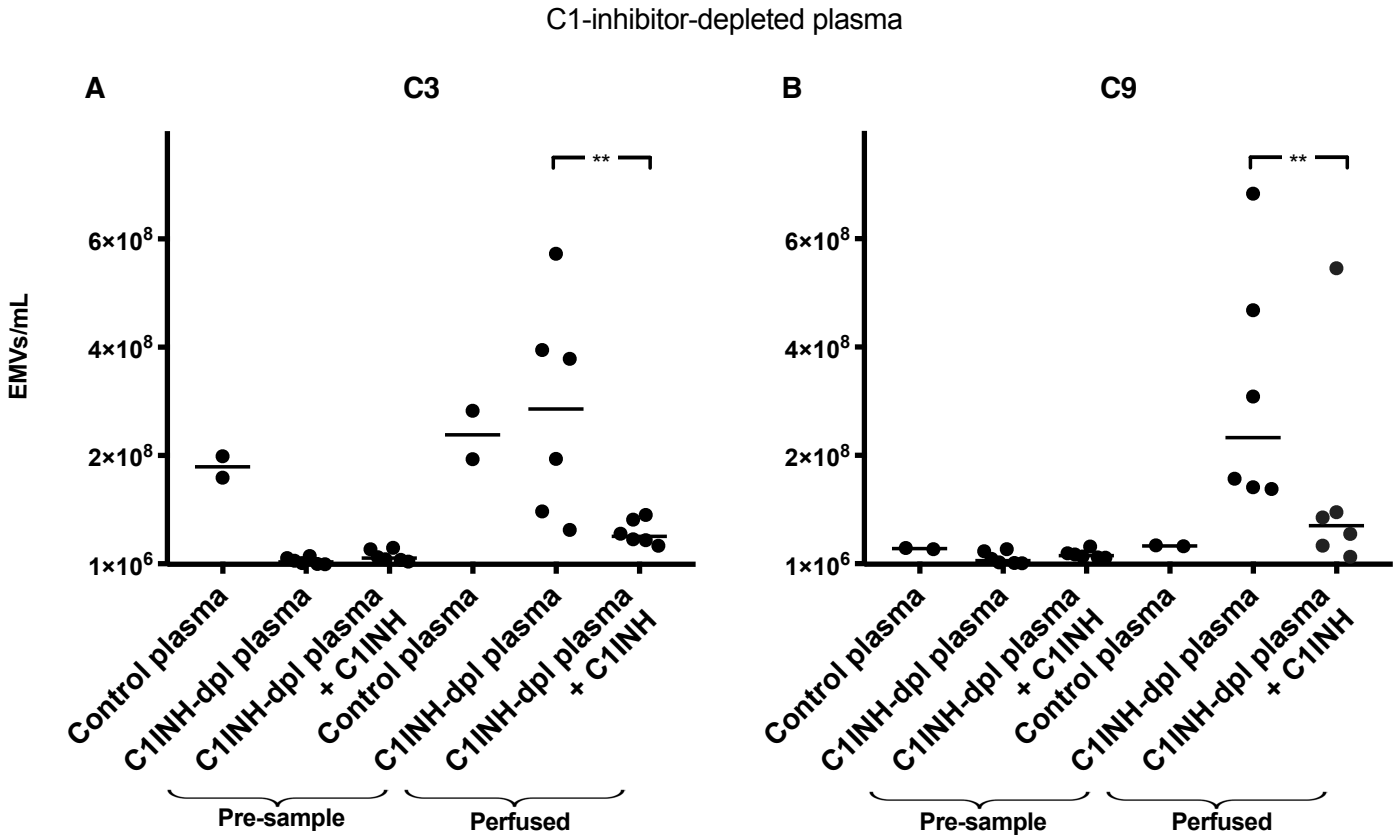

Figure S8: C1-inhibitor reduced release of C3- and C9-positive endothelial microvesicles after perfusion of C1-inhibitor-depleted plasma over primary glomerular endothelial cells

C1-inhibitor-depleted plasma was perfused over primary glomerular endothelial cells (PGECs) and the C3- and C9-positive endothelial cells (EMVs) were detected by flow cytometry in the plasma both before perfusion (pre-sample) as well as after perfusion (perfused sample). Values correspond to Figure 5 in the paper. A) More C3-positive EMVs were released in C1-inhibitor-depleted plasma perfused over PGECs than in control plasma, under the same conditions. Addition of C1-inhibitor to the C1-inhibitor-depleted plasma significantly reduced the C3-positive EMVs. B) More C3-positive EMVs were released in C1-inhibitor-depleted plasma perfused over PGECs than in control plasma, under the same conditions. Addition of C1-inhibitor to the C1-inhibitor-depleted plasma significantly reduced the C9-positive EMVs. **: P <0·01, *: P <0·05. Pre-sample: Pre-perfusion sample, EMVs: endothelial microvesicles, C1INH: C1-inhibitor, Dpl: depleted. The median is represented by the bar. Samples were analyzed using CyFlow Cube 8 flow cytometer.