

Supplementary Figure S1

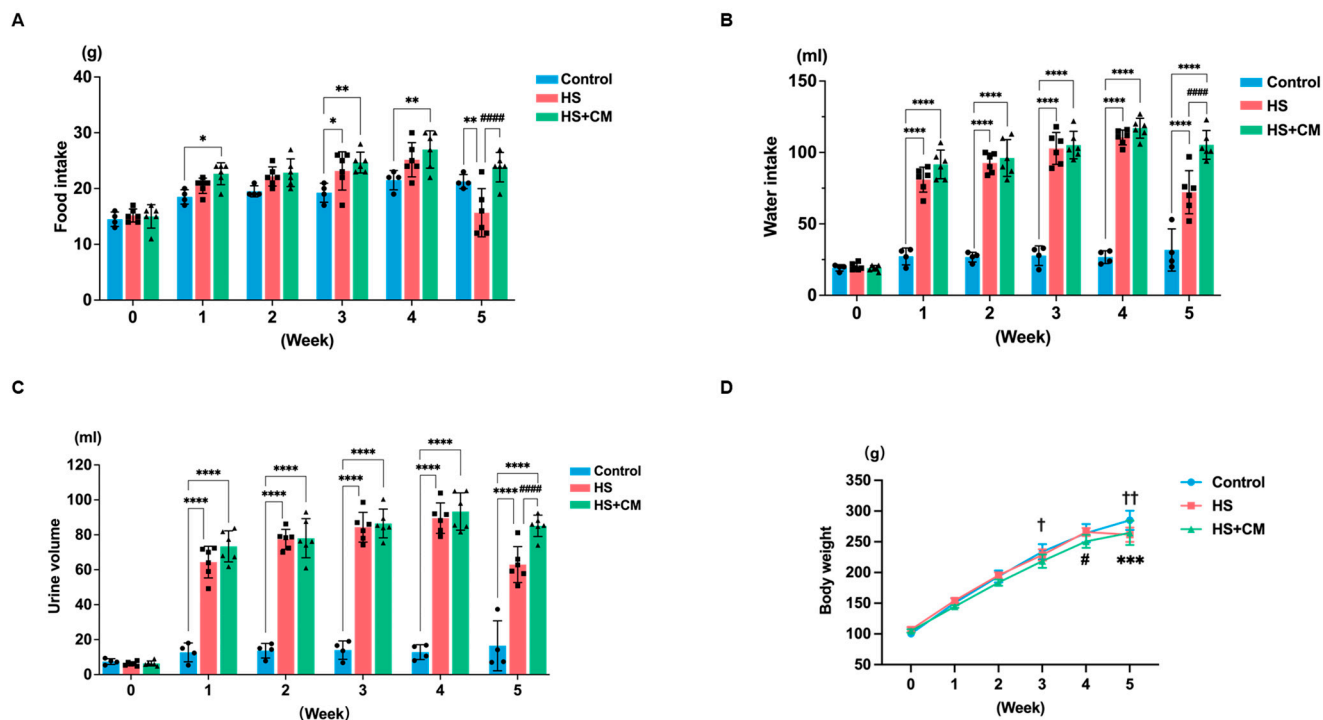


Figure S1. General parameters in the Control, high-salt (HS), and HS+camostat mesilate (CM) groups.

(A) Food intake, (B) water intake, (C) urine volume, and (D) body weight (BW) measured weekly; $n = 4, 6$, and 6 for the Control, HS, and HS+CM groups, respectively. The results were reported as the average and the standard deviation. We analyzed results using two-way ANOVA, followed by the Tukey test. *: $P < 0.05$, **: $P < 0.01$, ***: $P < 0.001$, **** $P < 0.0001$ vs Control group. #: $P < 0.05$, ##: $P < 0.01$ vs HS group. †: $P < 0.05$, ††: $P < 0.01$, Control group vs HS+CM group.

Supplementary Figure S2

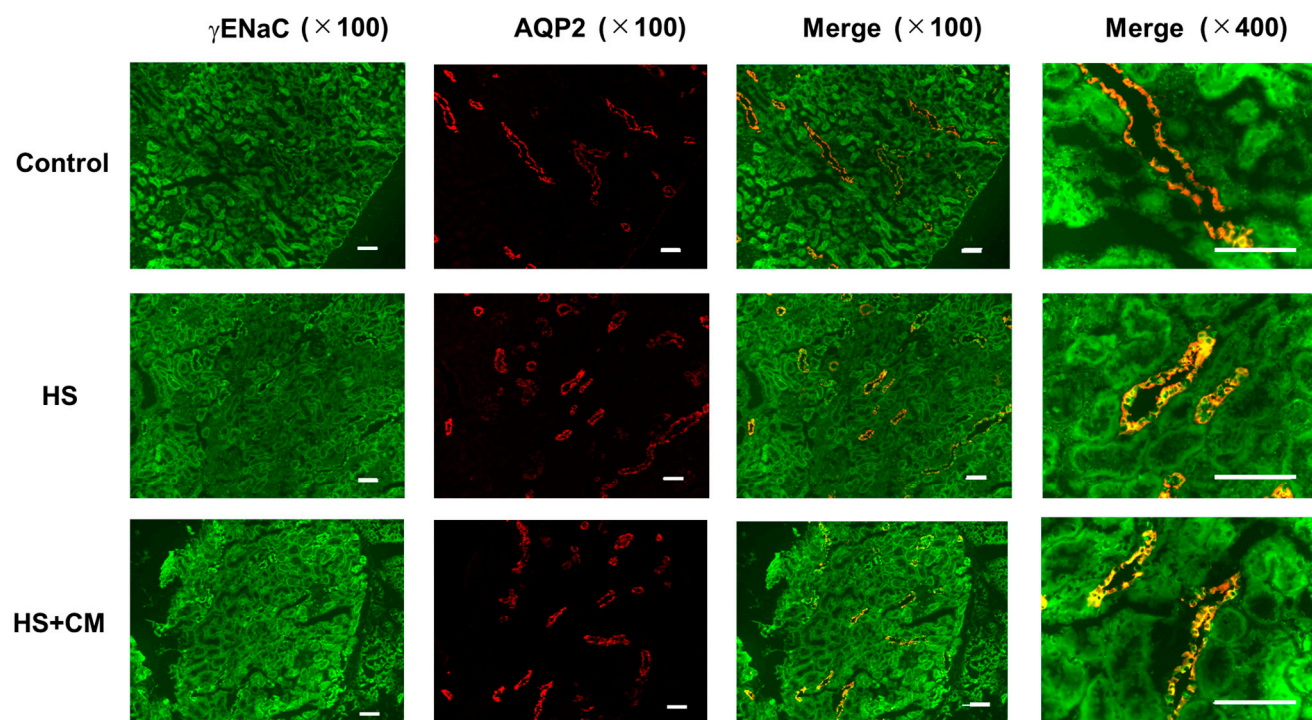


Figure S2. Double immunofluorescence staining of γ ENaC and AQP2.

The immunofluorescent staining confirmed the co-localization of γ ENaC and AQP2 in the collecting duct cells. The HS loading induced the apical trafficking of γ ENaC, which was not apparently affected by CM treatment. White scale bar represents 100 μ m.

Supplementary Figure S3

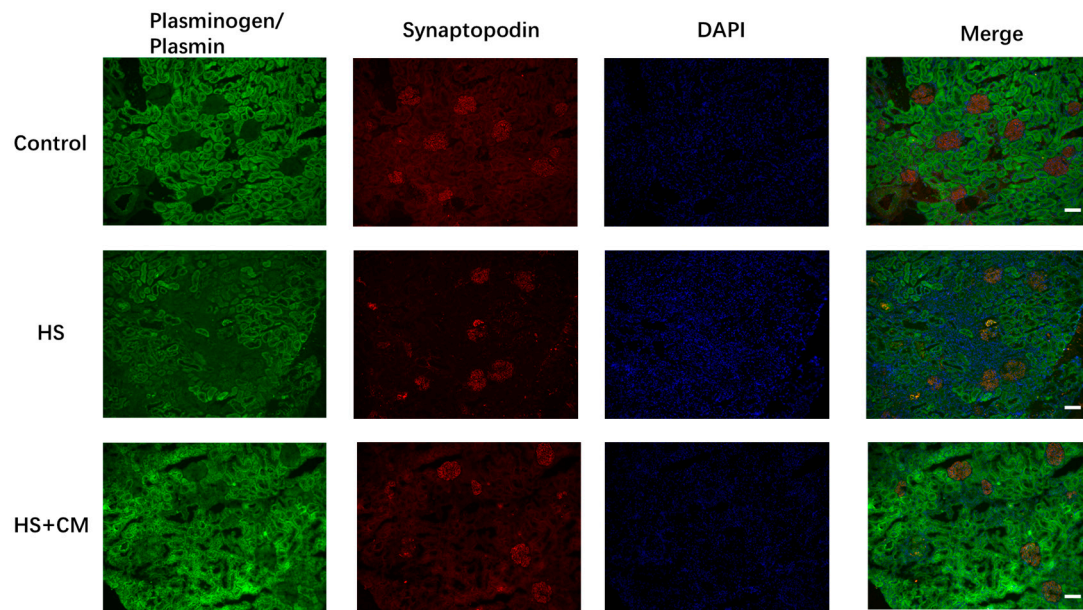


Figure S3. Lower magnification (100× magnification) of immunofluorescence staining of plasminogen/plasmin and synaptopodin.

In the HS group, plasminogen/plasmin adheres to the glomerulus and co-localizes with synaptopodin, which appears reduced due to glomerular injury. These changes are mitigated by CM treatment. White scale bar represents 100 μm .