Siglec-F-expressing neutrophils as critical contributors to renal fibrosis

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The roles of neutrophils in renal inflammation are currently unclear. On examining these cells in the unilateral ureteral obstruction murine model of chronic kidney disease, we found that the injured kidney bore a large and rapidly expanding population of neutrophils that expressed the eosinophil marker Siglec-F. We first confirmed that these cells were neutrophils. Siglec-F^{+} neutrophils were recently detected for the first time by several studies on other disease contexts. We then showed that (i) these cells were derived from conventional neutrophils in the renal vasculature by TGF-b and GM-CSF, (ii) they differed from their parent cells by more frequent hypersegmentation, higher expression of pro-fibrotic inflammatory cytokines, and, notably, expression of Collagen 1, and (iii) their depletion reduced collagen deposition and disease progression. These findings have thus unveiled a novel neutrophil subtype that participates in renal fibrosis and may be a new therapeutic target in chronic kidney disease.