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Following MIR, MCMECs release GMCSF to recruit monocyte infiltration, which in turn releases CCL2 to induce CCR2⁺ immune cell infiltration. GMCSF transits CCR2⁺ macrophages to M1 phenotype, mediating and amplifying inflammation that exacerbates MIR injury, while CCL2 transits CCR2⁺ macrophages to M2 phenotype, mediating fibrotic remodeling after MIR. See the article in pages 959–974.

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