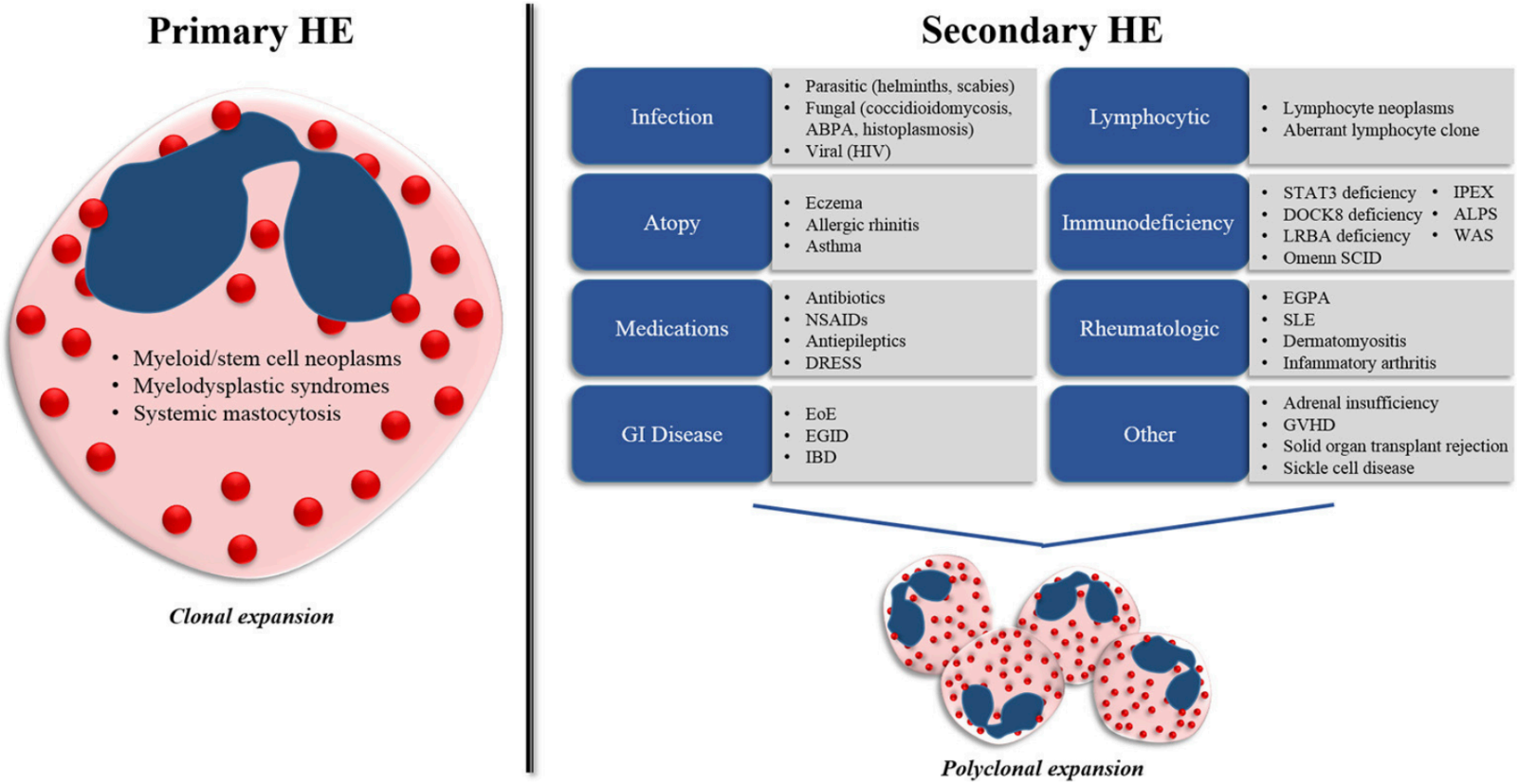

FIGURE 1 | Differential diagnosis for hypereosinophilia (HE) in children. Pediatric HE can be separated into two main categories (primary and secondary HE) on the basis of the underlying mechanisms driving the eosinophil expansion. Primary HE results from myeloid and stem cell abnormalities that propagate the expansion of an eosinophil clone. Secondary HE results from a diverse group of disease states that drive the expansion of the eosinophil population through increased eosinophilopoietic cytokine (IL-3, IL-5, and GM-CSF) production. ABPA, allergic bronchopulmonary aspergillosis; HIV, human immunodeficiency virus; NSAIDs, nonsteroidal anti-inflammatory drugs; DRESS, drug reaction with eosinophilia and systemic symptoms; EoE, eosinophilic esophagitis; EGID, eosinophilic gastrointestinal disease; IBD, inflammatory bowel disease; STAT3**,** signal transducer and activator of transcription 3, DOCK8, dedicator of cytokinesis 8; LRBA, lipopolysaccharide-responsive-beige-like-anchor; WAS, Wiskott-Aldrich syndrome; IPEX, immune dysregulation, polyendocrinopathy, enteropathy, X-linked syndrome; ALPS, autoimmune lymphoproliferative syndrome; EGPA, eosinophilic granulomatosis with polyangiitis; SLE, systemic lupus erythematosus, GVHD, graft-vs.-host disease.