



**FIGURE 1. The pathophysiological pathway of NLRP3 inflammasomes activation by periodontitis.** The NLRP3 structure contains three domains: NLRP3, adaptor apoptosis speck (ASC), and pro-caspase-1. Activation of the pathway results in binding of the three domains forming NLRP3 inflammasome complex. This activation is induced mainly by *Porphyromonas gingivalis* pathogens in periodontitis, which is influenced by genetics and environmental factors. The signals from pathogen-associated molecular patterns (PAMPs) or by damage-associated molecular patterns (DAMPs) which activates the NF-κB pathway to release pro IL-1β and IL-18 from nucleus into the cytoplasm. The activation and formation of NLRP3 inflammasome complex has two main effects: (i) activation of Gasdermin D GSDMD and inducing pyroptosis and/or (ii) active caspase-1 which then cleaves pro-IL-1β and pro-IL-18 into their active forms IL-1β and IL-18 as proinflammatory cytokines that released out the cell causing periodontitis.