

rate in mice ⁽²⁰⁾. But the inefficiency of NK cell, macrophage, and Th1-type cytokines in killing trophoblasts led to question the mechanism whereby the cytokines produced their effects. A target other than trophoblasts for cytokines was sought; a maternal vascular target was suggested by pathologic specimens of aborted material that showed hemorrhagic necrosis at the trophoblast-decidual interface ⁽²¹⁾.

Pro-inflammatory cytokines such as IL-1, TNF-α and INF-γ collaborate to activate procoagulant expression in endothelial cells that are in direct contact with maternal blood. Prothrombin is converted to thrombin; thrombin then catalyzes generation of fibrin and activates IL-8 secretion by endothelial cells. IL-8 recruits polymorphonuclear leukocytes (PMNs) which kill endothelium that has been activated by IL-1, TNF-α and INF-γ ⁽²²⁾. The end result of unchecked thrombin production is clot formation occluding blood supply to the embryo leading to its death ⁽²³⁾. The procoagulant stimulated by these cytokines, which is responsible for prothrombinase activity in abortions, has been identified as the prothrombinase called fibroleukin gene (fgl2) ⁽²¹⁻²⁴⁾. The fgl2 is present in both decidua and trophoblasts of aborted but not control tissue ⁽²³⁾. Clotting initiated by fgl2 is known to lead to ischemic damage in a variety of inflammatory disease models such as hepatitis and endotoxic shock ⁽²⁵⁾.

Patients and Methods

Patients were collected from Al-Kadhimya and Al-Ulwiya teaching hospitals in Baghdad in the year 2004, and were divided into three groups; **Group A:** 24 pregnant ladies presented with incomplete first trimester abortion, all of whom gave a history of previous 3-6 consecutive first trimester abortions, with no medical diseases,

family history of genetic diseases or uterine anatomical anomaly, also all of them were negative for acute infection with rubella, cytomegalovirus and toxoplasmosis. **Group B:** 10 pregnant ladies presented with incomplete first trimester abortion and had at least three previous normal pregnancies with no previous abortion, and no history of any medical illness. And **Group C:** 6 pregnant ladies with elective termination of pregnancy in the first trimester for a maternal indication under approved consent of two senior gynecologists and a physician. Curate samples of the materno-fetal interface were taken from all these women at the end of evacuation curate operation, samples were embedded in paraffin and subjected for *in situ* hybridization technique.

***In situ* Hybridization:** For *in situ* hybridization technique (ISH), DNA Probe Hybridization/Detection System In situ kit (Maxim Biotech, Inc., USA) was used. Kit contents included: biotinylated housekeeping gene probe, hybridization solution (ready to use), protein block, detergent wash buffer, RNase A (15 µg/ ml), streptavidin-AP conjugate, substrate (BCIP/NBT), and lyophilized proteinase K (4 mg); which is dissolved in a 2 ml DNase and RNase free dilution buffer to form 10X proteinase K, then diluted by deionized water to 1X proteinase K. The probes were biotin-labeled DNA probes for human IFN-γ (249 bp), and human IL-10 (223bp), (Maxim Biotech, Inc., USA).

Tissue sections were deparaffinized in xylene for 5 minutes and rehydrated through a series of ethanol dilutions. After digestion with 1X proteinase K at 37°C for 15 minutes, the sections were quickly dehydrated in ethanol. Hybridization was carried out by applying 10 µl hybridization mixture (0.8 µl of heat