SUMMARY AND CONCLUSION

Apoptosis or programmed cell death (PCD) is a physiologic phenomenon that ensures the balance between cell proliferation and cell death. Apoptosis removes the senescent, damaged abnormal cell interfering with the normal function of the organism. The abnormalities of apoptosis play an important role in the proliferative neoplasic disease, degenerative disorders and autoimmune disease ***(Schuler and Szendes, 1997).***

The leukemias are the most common form of cancers. They account for about one third of new cases of cancer diagnosed each year. The acute lymphoblastic leukemia represent the quintessential example of malignancy caused by failed programmed cell death (Apoptosis), as apposed to altered cell cycle regulation. In all self-renewing tissues, new cell production is normally offset by commensurate amount of cell destruction through programmed cell death ***(Tsangris et al., 1996).***

Imbalances in the activities of opposing genes that either promote or block physiological cell death can therefore slow or halt the rate of cell turnover, creating a selective survival advandage for a particular clone that permit expansion, often at the expense of its normal neighbors ***(Thompson, 1995).***

The cell surface Fas receptors (CD95) is a cystein rich transmembrane glycoprotein, and belongs to the nerve growth factor (NGF), tumour necrosis factor (TNF) receptor family ***(Oehm et al., 1992).***

Fas receptor can transduce signals that lead to apoptotic cell death. The Fas receptor based death pathway plays an important regulatory role for the elemination of cell in-vivo ***(Ogasawara et al., 1995).***

In cancer treatment, the induction of apoptosis in malignant cells seems to be one of the most successful mechanism ***(Kerr et al., 1994).***

In order to assess the sensitivity of leukemic cells to chemotherapy we studied:

*Apoptosis of peripheral blood blast cells before initiation of induction chemotherapy and 24 hours after therapy.

*sFas level in serum of patients with acute lymphoblastic leukaemia before therapy, one day and one month after therapy.

We correlated our results with the clinical response of these patients to evaluate apoptosis and sFas level as a prognostic factor.

The chemotherapy response was determined by the number of blast cells in peripheral blood and bone marrow on day 30 of treatment. Good clinical response is defined as a decrease in the number of blast cells in peripheral blood to less than 1% and in bone marrow to less than 5% on day 30 ***(Pui., 1996).***

This study was applied on 30 patients of ALL and 10 healthy controls with matched age and sex. Standard morphological criteria for the diagnosis of ALL were used based upon Leishman stain and cytochemical studies of peripheral blood and bone marrow cells. The cellular phenotype was determined on bone marrow mononuclear cells

cytochemical studies of peripheral blood and bone marrow cells. The cellular phenotype was determined on bone marrow mononuclear cells using standard panel of monoclonal antibodies. Mononuclear cells were harvested from leukemic patients at initial presentation and 24 hours after induction chemotherapy.

- Apoptosis was detected by immunofluorescence technique.
- sFas level was measured by ELISA technique.

In this study, it was found that:

- There was a negligible apoptosis at presentation (ranged from 0-4%) with a mean of 0.6 ± 0.18.
- There was highly significant increase in the percent of apoptotic cell one day after induction therapy with a mean of 23.833 ± 1.55.
- Our study also revealed a high level of sFas in the serum of all patients of ALL at presentation and there was no significant difference in its level 24 hours after therapy, but there was a highly significant decrease between its level one month after therapy than before treatment .
- As regard serum sFas level in high and low risk group, there was significant increase in serum sFas level in high risk group than low risk group regarding to age, haemoglobin, TLC, LDH, IPT, tumour load, and there was no significant difference in its level in high and low risk group regarding to sex, platelets count, and FAB classification.
- There was a negative correlation between the percentage of apoptotic cells and TLC and serum level of sFas, but there was no correlation between the percentage of apoptotic cells and age, TLC, Hb level, or platelets count.

- sFas level correlated with TLC, , and LDH ($P < 0.05$), and no significant correlation was found between sFas level and other prognostic factors including age, Hb level, platelets count, and percentage of blast cells. This implies that sFas is an independent prognostic factor of ALL.

This means that to evaluate the success of induction therapy studying apoptosis from peripheral blood is a good marker.

While to follow-up case with successful induction the use of sFas is a good marker. Also sFas is can act as a putative marker for active resistant leukaemia.