INTRODUCTION & Aim of work

The morbidity and mortality associated with bacterial meningitis have remaind distressingly high decades after the introduction of antibiotics (Schlech *etal.*, 1985), and the advent of newer more potent antibiotics has not substanially improved the outcome of this disease (Yogev R., 1985).

One explanation is that the pathologic consequences of the disease progress despite bacteriologic cure (Swartz M.N., 1984). During the course of bacterial meningitis bacterial cell wall products including peptidoglycan, techoic acid or endotoxin(Lipopolysaccharide, LPS) which are generated in vivo or released during antibiotic therapy (Tauber M.G. and Sande,1984) induce an intense host inflammatory response in the subarachnoid space (Tuomanen E, *etal.*, 1985) .The induction and amplification of these host inflammatory responses, to control locally the infectious process, may actually exert a destructive effect on the CNS and thus may contribute to the morbidity and mortality of meningitis (Tuomanen E; 1988).

The pathophysiologic effects of LPS are mediated by endogenous factors such as tumor necrosis factor α, ineterleukin -1, and platelet activating factor. Although LPS - host interactions and and the systemic effects of locally released cytokines and other mediators has remianed unclear. Recent studies have shown that local overproduction of

cytokines, especially if prolonged , sustain the inflammatory response and induce consequences potentially deterimental to the host (Dinarello CA *etal.*, 1989) .

Because of the benifcial effects of early therapy in bacterial meningitis as well as the potenial medical risk of antimicrobial drugs it would be ideal to differentiate viral from bacterial disease at the time of initial assessment of the patient (Ohga etal ., 1994) .

Our present work aimed at :

1)Studying the TNF-α level in both CSF and plasma in cases of bacterial and aseptic meningitis .

2)Differentiation between the two types of meningitis through measuring initial TNF -α level of both plasma and CSF .

3)Determination whether the initial CSF or plasma concentration of TNF - α correlate with the clinical presentation or with outcome .