replicating latent form EBV is mainly B-cell tropic but capable of infecting T-cells and epithelial cells. EBV is the first human virus implicated in the pathogenesis of lymphoid and epithelial malignancies which reach 80% in developing countries; these malignancies including Burkitt's lymphoma (BL), undifferentiated nasopharyngeal carcinoma (UNFC), HL, NHL, post-transplant lympho-proliferative disease (PTLD), some T-cell lymphoma and more recently certain cancers of stomach and smooth muscles. Hodgkin's lymphoma is uncommon malignant tumor of the lymphatic system where approximately 40% of HL were shown to contain clonal EBV [(2)]. NHL are associated with EBV, like nasal T / natural killer (NK) cell lymphoma and angioimmunoblastic lymphadenopathy [(3)]. Due to EBV associated malignancies, recent advances in PCR technology called Quantitative Competitive PCR (QC-PCR) permit precise measurement of EBV DNA level in clinical samples called EBV viral load. QC-PCR is used to quantify PCR products; it's a method to quantitatively measure DNA amount, and number of its copies in the sample [(4)]. Epstein -Barr virus (EBV) viral load assays able to distinguish low-level infection in carriers from higher levels associated EBV diseased patients. The patients affected by EBV often have high levels of EBV DNA in their body fluids like blood, plasma or serum and this is used as specific marker for EBV carcinogenesis. The QC-PCR co-amplify EBV DNA and a spiked or endogenous control sequence called Internal standard (IS), the relative amount of EBV and control product was measured in EBV related disease for early diagnosis and for monitoring the efficiency of therapy [(5)]. QC-PCR used to quantitate, EBV-DNA in plasma of all EBERs-positive AIDS lymphoma patients; they concluded that QC-PCR is very promising in diagnosis and management of EBV related lymphoma [(6)].

Aim of the study: Utilizing EBV DNA load as molecular biomarker to predict the prognosis and to check response to chemotherapy in HL and NHL patients.

Methods

Patients and samples preparation: Peripheral blood samples, were taken before and 3-4 months after chemotherapy from Eighteen HL and NHL patients at Baghdad Medical City Teaching Hospital. Sampling extended from Feb 2005 to Nov 2005. Nine apparently healthy individual were enrolled in this study as control group.

Viral DNA extraction: Fresh whole blood from healthy group and patients were diluted 10 times in NASBA lysis buffer contains 5M guanidine thiocyanate, 0.75% Triton X-100, 1 M Tris -HCL, stored at –20 °C until DNA extracted by silica based extraction method as described previously by Boom et al, 1990.

EBV DNA: EBV DNA obtained by Transformation of Escherichia coli MM 294 with Wild type (WT) DNA plasmid *pGEMBamHI-K* and according to those reports by Kushner, 1978.

Plasmid DNA used: WT DNA *pGEMBamHI-K* plasmid which has the prototype EBV B95-8 EBNA1 sequence, used as positive control and *PQPCR8* plasmid DNA as in Gene bank was used as Internal Standard (IS) which compete viral DNA in QC-PCR, both obtained from Dr. Stevens, S.J.C, University Hospital Vrije, the Netherlands.

Plasmid DNA extraction: Plasmid DNA extracted from *E. coli* cells as described by Pospiech and Neuman, 1995, number of WT and IS DNA copies was determined.

Primers and Probes: The nucleotide sequences and localization of primers are listed in Table 1.