

at time of diagnosis, they show 100% response and their viral load lowered below cutoff value, these results are in agreement with Josting et al, 2002 who observed continuously low or undetectable level of EBV in serum samples of NPC patients who reported that EBV DNA load is a valuable tool for monitoring of NPC patients against tumor recurrence.

Our results obtained confirm reports that patients with HL have an excellent prognosis with modern chemotherapy even if the disease is far advanced at diagnosis<sup>(18,19)</sup>.

In studies assaying QC-PCR in lymphoma AIDS patients, our results come in agreement with others who reported that the viral load in 17 EBERS-positive lymphoma patients ranging from 34-1,500,000 copies per ml, this viral load fall rapidly upon initiation of lymphoma therapy and remaining undetectable except in two patients with persistent tumor<sup>(20)</sup>.

In group II ( Table 3) QC-PCR shows that Patients viral load decrease after chemotherapy but still above cutoff value, similar results obtained by Fan et al. 2004 who work with EBV viral load of lymphoma in AIDS patients , they found that the viral load usually falls upon initiation of chemotherapy except in two patients with persistent tumor. It is reported that when analyzing the EBV status in the peripheral blood of pediatric patients with HD where no EBV DNA was detected in plasma of HD with complete remission while 2 of 5 HD patients will relapse and are positive for EBV DNA<sup>(15)</sup>.

In group III (Table 3) who have high viral load above the cutoff value and increase in the EBV load after chemotherapy. The presence of high viral load at diagnosis time is in agreement with Gallagher et al. 1999 who reported that EBV DNA was detected in 91% of serum samples from patients with EBV positive HL. The increase in the EBV load after completion of treatment are in

consistent with Drouet et al. 1999, that this viral DNA was probably a consequence of viremia which is related to increased viral replication in a non neoplastic compartment such as oropharynx which explain the increase in the viral load during the period of treatment.

In the field of EBV associated NPC, the same finding of persistence in the viral load after chemotherapy was observed, workers in this field explained that this persistence of viral load following primary therapy was predictive of relapse<sup>(23,24,25,16)</sup>.

Other worker with EBV viral load of lymphoma in AIDS patients, found that the viral load usually falls upon initiation of chemotherapy except in two patients with persistent tumor<sup>(26)</sup>, others analyze the EBV status in the peripheral blood of pediatric patients with HD where no EBV DNA was detected in plasma of HD with complete remission while 2 of 5 HD patients relapse and were positive for EBV DNA<sup>(15)</sup>.

For group IV of patients with initial viral load at the time of diagnosis below cutoff value, some of them are EBV negative, after the completion of the chemotherapy they showed slightly increase in the viral load but still below the cutoff value. The absence of elevated EBV DNA load in those patients who constitute 33.3% in HL and 55.5% in NHL as shown in Table 2 and falls in group IV which constitute 44.45% in table 4, This absence of elevation of EBV load in these groups of patients might be explained by the fact that those patients developed an EBV-negative lymphoma and these results could be debated by that some patients have lymphoma not due to oncogenic potential of EBV this is consistent with<sup>(10)</sup>. These results are also consistent with studies done<sup>(10,21)</sup>, they reported that EBV DNA was detected in 23% of EBV negative HL patients. Others