Discussion

In current study the age of most cases was between 10-39 years and more than half cases were below 27 years old. This was concordant with the results of Iraqi Cancer Registry in 2006 which showed that most of the Hodgkin lymphoma (HL) cases were between 15-35 years [6] and it was similar to other Iraqi studies done in 2007, 2005,2004 and 2001 [17-20] and to studies done in other Arab countries like Kuwait, Jordan and Egypt [21-23]. Thus we may conclude that in Iraq the age distribution of HL followed the pattern in developing countries in which the disease occurred earlier than in developed countries.

The slight male predominance observed in this study was in agreement with Iraqi Cancer Registry (ICR) results in 2006 [6] and in other Iraqi study in 2005 [20], but it differed from Al-Safi results in 2007 which stated that incidence of Hodgkin lymphoma was equal in both male and female [17]. This could explain by the small number of the samples.

Similar to this study, other studies done in other Arab countries like Kuwait in 2003 [21] and Egypt in 2010 [23] and worldwide [24-26] showed that there was male predominance with ratio of 1.2-2.4:1.

Similar to the results of ICR in 2006 [6] and other local studies in 2005 [18], 2003 [27], 200120 and a Kuwaiti study in 2003 [21], the mixed cellularity was the commonest histological subtype.

Cervical lymph nodes were the commonest site involved by tumour which was in agreement with the results of Iraqi study done in 2007 [19], Turkish study in 2005 [29] and a Kuwaiti study done in 2003 [21].

In the current study, LMP-1 expression was positive in 90% of HD versus 60% of control group. This high expression was comparable to an Iraqi study that was done by Al-Safi in 2007 [17] in which LMP -1 was found in 75% of Hodgkin lymphoma cases and also in line with other developing countries reaching 63% in Egypt [23], 60% in Nigeria [30], 82% in India [31], and 93% in Iran [32], whereas it was less common in developed countries, with percentages of 20-50% for North American [33] and European cases [34], and 39% in China [26].

Although LMP-1 EBV antigen was detected in HL and control subjects ,the expression was significantly higher in HL and since LMP-1 is the major EBV oncogene and is essential for B-cell immortalization; thus we may conclude that the presence of the virus have played an important role in the pathogenesis of the disease.

In the present study all patients below 16 years, were infected with EBV and showed high expression of LMP-1. These results were in agreement with Al-Safi study which revealed that EBV expression was highest in childhood [17]. This followed the pattern of EBV expression in developing countries such as Kuwait [21], Jordan [22] and Iran [35]. And this in contrast to the results found in USA [33], United Arab Emirates [36], and Netherlands [34], in which high frequency of LMP-1 expression was seen in young adult. In developing countries, infection usually occurs in early childhood and usually passed unnoticed and the vast majority will be persistently infected with a reservoir of infection in memory B-cells this may lead to Hodgkin's Lymphoma to develop in childhood group [21]. Whereas in industrialized countries primary infection is often delayed until adolescence and frequently results in infectious mononucleosis (IM) [7].

The high expression of LMP-1 in mixed cellularity HL that was seen in the present study was in concordance with several studies done in Jordan [22], China [26] and Rio de Janeiro [37].

Bcl-2 is an antiapoptotic protein, it was detected in 66% of HD cases, and this was in line with results of Rassidakis *et al* [38], Wang and Taylor [39], Kim *et al* [40] and Adelusola studies [30], which found that Bcl-2 expression was detected in HRS cells in 61%, 56.45%, 43.7%, 56% and 40% of the cases respectively. Moreover the highest expression was detected in the aggressive Lymphocyte depleted subtype followed by mixed cellularity. This result was concordant with results of Flangea *et al* [41] and it explain the role in the pathogenesis of the disease, since overexpression of Bcl-2 may result in accumulation of cells in the G0 phase of cell cycle [14]; causing resistance to chemotherapeutic drugs and radiation therapy thus Bcl-2 was considered as a