



Figure 1. The amplification of *C. trachomatis* by PCR. L: size marker of 100 bp, lanes 1 to 6: Positive samples for *C. trachomatis*; lane 7: positive control, lane 8: negative control.

Discussion

Due to the silent nature of *C. trachomatis* infections, most infected women are asymptomatic and therefore remain unrecognized and untreated. There are some evidences that show screening and treating women infected with *C. trachomatis* can reduce PID and its complication (16).

In our study, the prevalence of *C. trachomatis* among infertile women by PCR and IgM was 5% (5 case) and 6% (6 cases), respectively. Similar to our study, the prevalence of *C. trachomatis* among infertile women in some regions has been reported such as 52.8% in Brazil by PCR (17), 3.3% in Rwanda by serology (18), and 3.9% in Jordan by PCR (19). This heterogeneity could be due to the diversity in epidemiological condition, the study population, technique used (molecular or serology) or specificity of target primers in molecular methods.

In our study, the incidence rate of *C. trachomatis* among fertile women using PCR and IgM was 1.6% (2 cases) and 1.6% (2 cases), respectively. Also, we did not find any seropositive IgG in both groups. The low titers or lack of IgG may be due to the absence of previous exposure with *C. trachomatis*. According to results of Malik and colleagues, it seems that IgG detection and past chlamydial infections have a strong role in women with secondary infertility rather than primary infertility (20).

According to laparoscopy results, the past infections with *C. trachomatis* are associated with a significantly increased risk of tubal

infertility in women and these results were confirmed by serology. Furthermore, the severity of tubal damage found in infertile women is directly related to serum antibody titer levels (17). In research of Malik and colleagues IgG antibodies were present in 55% of women with secondary infertility compare to 5.5% in health women (20). In our study since almost infertility cases (82%) was primary, the absence of IgG antibodies can be explained. Also, we showed that the most prevalent etiology of infertility was ovarian defect.

However, *C. trachomatis* infection was recognized with more frequency in women with tubal defect using PCR and serology. Similar to our study, in study of Rashidi and colleagues, the ovarian defect was reported as the main cause of infertility (15). However, *C. trachomatis* infection was seen significantly in women with ovarian defect ($p>0.05$). Since in our study, the prevalence rate of *C. trachomatis* infection was lower than study of Rashidi, we can't find an association between *C. trachomatis* infection and infertility causes. So, the selection of a large statistical community for determining this association is essential.

There are some challenges on the relation of *C. trachomatis* with infertility. Moreover, Malik, Sattari, Badami and Marashi indicated that *C. trachomatis* infection can be as an infertility risk factor. While, Al-Ramahi, Rashidi and Muvunyi didn't find any significant difference between fertile and infertile women for *C. trachomatis* infection (13-15, 18-21). In our study, using different diagnostic methods, no significant difference was found between these fertile and infertile groups for *C. trachomatis* infection ($p>0.05$). This difference in the results can be explained due to technique used, the number of study population and type of infertility (primary or secondary). Moreover, similar to our study, in the research of Rashidi the dominant type of infertility was primary and no meaningful relation was found between fertile and infertile groups (15).

As mentioned above, the past infections with *C. trachomatis* can be a potential factor for infertility especially secondary type that in our work, numbers of the women with this type of infertility was low (18 cases). In the other hand, the techniques used by Malik and colleagues, Sattari *et al* and Badami and