

INTRODUCTION

Bronchial asthma, one of the chronic inflammatory disease which affect infant, young children even adult. It is a chronic inflammatory disorder of the airways which cause recurrent episodes of wheezing, chest tightness and cough particularly at night or early in the morning (*Terttu et al., 2006*).

Bronchial asthma has been correlated with a variety of viral pathogens, most commonly respiratory syncytial virus in infancy and rhinovirus in older children. Progress is being made in understanding; how virus can adversely affect lung or immune development. In asthmatic children viral infections initiate bronchospasm and air ways obstruction (*Gern, 2004*).

It was stated that R.S.V bronchiolitis constitute a risk factor for the development of bronchial asthma, the risk increase with familial or personal antecedents of atopy (*Martin, 2001*).

Primary multiplication of R.S.V occurs in epithelial cells of U.R.T producing a mild illness. In 50% children less than 8 months old virus subsequently spreads into the L.R.T causing Bronchiolitis, pneumonia and croup. It is suggested that a possible factor in cot death (*Collins et al., 1995*).

Viral infection are common triggers of asthma exacerbation in children and R.S.V. is associated with the most sever exacerbations in children, prevention and/ or treatment of R.S.V infection should

considered a high priority for the management of asthmatic children. (*Christopher et al., 2001*).

Several genes have been associated with susceptibility to asthma and allergy; complex gene-environmental interactions, however, appear to play a key role in the development of the disease. Early sensitization to aeroallergens, presences of atopic dermatitis, maternal smoking during pregnancy and children's environmental exposure to tobacco smoke, lower respiratory tract infections with respiratory syncytial virus, exposure to air pollutants, all are risk factors associated with an increased risk for development of chronic asthma (*Arruda et al., 2005*).