

*INTRODUCTION
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Epidermodysplasia verruciformis (EV) is a rare familial skin disease first described by lewandowsky and lutz in 1922. It is characterized by long standing, widespread flat wart-like lesions and macular pityriasis versicolor-like lesions. The disease usually begins in childhood (Lutzner, 1978).

EV is caused by certain types of human papilloma viruses (HPV) and to the present, more than 15 types of HPV have been isolated from the lesions of EV (Orth et al., 1978b). the causative HPVs include types 3,5,8,9,10,12,14,15,17, and 19-24. (Coggin and Zur Hausen, 1979). The most common virus found in EV lesions is HPV-5 (Lutzner et al., 1983).

The role of viruses was demonstrated by auto- and heteroinoculation experiments (Jablonska et al., 1968) and by the detection of HPV particles in benign lesions (Ruiter and Van Mullem, 1966). Viral particles are even more abundant than in warts and more easily detectable by electron microscopy and/or immunofluorescence techniques (Jablonska et al., 1970).

The aim of the work is to study the ultrastructure of the lesions of EV.