Research Article

Clinicopathological study of viral skin lesions

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Abstract

Introduction: Many viral infections have prominent skin manifestations. Most commonly encountered are herpes simplex, molluscum contagiosum and human papilloma virus which causes verruca vulgaris, condyloma acuminatum, deep palmoplantar wart and verucca plana. They are of increased significance in immunocompromised patients. Most of the viral lesions are diagnosed clinically and serologically, some require biopsy confirmation. **Aims and objectives:** To identify the specific histological changes in individual viral lesions. To differentiate the lesions that can clinically mimic as bullous lesion or soft tissue mass. **Materials and Methods:** The skin biopsies received in the department of pathology, SMVMCH for a period of 3 years from 2011 to 2013 were taken for this study. **Results:** Out of 2160 skin biopsies studied, 31 patients were diagnosed to have skin manifestations of various viral infections. The most commonly encountered entity was Verruca vulgaris (14), followed by condyloma accuminatum (6), deep palmoplantar wart (4), molluscum contagiosum (4), herpes (2) and verucca plana (1). The most characteristic histological findings that helped in the diagnosis were, intranuclear inclusions in herpes; cytoplasmic viral inclusion bodies in MC, deep palmoplantar wart; koilocytes in Verruca and condyloma .These were also associated with other epidermal changes. **Conclusion:** Virus can produce warty, bullous and mass like lesions which can mimic non infectious conditions. Histopathological evaluation serves as a valuable tool for identification of virus induced skin changes and aids in appropriate management of these lesions. In our study most common infection was HPV.

Keywords: anal condyloma, hpv, intranuclear inclusions, verruca vulgaris.

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INTRODUCTION

Many viral infections have prominent skin manifestations. The two most common manifestations are viral warts and vesiculo bullous lesions. Viral warts include common wart (verruca vulgaris), genital wart (condyloma acuminatum), deep palmoplantar wart, verruca plana and epidermodysplasia verruciformis. Vesiculobullous lesions are commonly due to herpes simplex, varicella and herpes zoster. Viruses induce alterations in cell function, antigenicity, cell death and host responses. These are of increased significance in immune compromised patients, who have defective cell mediated immunity. Also disseminated viral infection is more likely to occur in such people. Most of the viral lesions are diagnosed clinically and serologically, but few may mimic other lesions clinically which necessitates biopsy.[1,2,3,4]

AIMS AND OBJECTIVES

To identify the specific histological changes in individual viral lesions and to differentiate the lesions that can clinically mimic as bullous lesion or soft tissue mass.

MATERIALS AND METHODS

A retrospective study was conducted on cases of viral skin lesions diagnosed in the department of Pathology, SMVMCH between January 2011 and December 2013. The specimens received were fixed in 10% formal saline, routinely processed and stained with Haematoxylin and Eosin (HandE) stains. Relevant clinical and histopathological data were extracted and analyzed.

OBSERVATIONS AND RESULTS

During the period of 3 years, 2160 skin biopsies were studied. Of which 31 patients were diagnosed to have

skin manifestations of various viral infections. Of which verruca vulgaris (VV) was the most common (14 cases), followed by condyloma acuminatum (CA) 6 cases, deep palmoplantar wart (DPW) 4cases, molluscum contagiosum (MC) 4cases, Herpes (2 cases) and verruca plana (1 case).

Table 1: Types of viral skin lesions, site of involvement and their clinical presentations

Histopathological diagnosis	Sites of involvement	Clinical presentation	Clinical diagnosis
Verruca vulgaris[14]	Forearm, Hands, feet[11] Gluteal region[3] Ear lobule[1]	Multiple to solitary , painless, hyperkeratotic papules, plaques (few hyperpigmented) with verrucous surface m/s 7x4 to 1x1.	Hypertrophic lichen planus, cutaneous horn, tuberculous verrucosa cutis, seborrheic keratosis
Condyloma acuminatum[6]	Anus [5] Penis[1]	Soft sessile, smooth to digitated m/s5x4 to 1x0.5	Papilloma, anal stricture, squamous cell carcinoma
Palmoplantar wart[4]	Sole[3] Palm[1]	Solitary firm nodule, smooth surface, painful.	Corn foot
Molluscum contagiosum[4]	Face[3] Forearm[1]	Multiple dome shaped papules, skin coloured to whitish with a central pore m/s less than 0.5cm.	Molluscum contagiosum
Herpes[2]	face[1] Gluteal region[1]	Clusters of vesicles, erythematous with burning sensation.	Varicella Zoster infection
Verruca plana[1]	Lower limb[1]	Solitary sessile lesion	Molluscum contagiosum

DISCUSSION

The histological findings are characteristic in a few viral lesions. Verruca vulgaris and condyloma acuminatum show hyperkeratosis, acanthosis, parakeratosis, papillomatosis and koilocytes. Koilocytes have an enlarged, hyperchromatic, irregular, raisinoid nucleus with perinuclear halo. Verruca plana shows hyperkeratosis, slight elongation of rete ridges without papillomatosis, basket-weave appearance in horny layer and vacuolated cells with central basophilic nucleus. The parakeratosis and papillomatosis are more pronounced in verruca vulgaris in comparison to condyloma acuminatum and verruca plana.[1,2,3] Seborrheic keratosis, keratoacanthoma and solar keratosis simulate verruca vulgaris. But these conditions lack the koilocytes. Condyloma acuminatum can be differentiated from bowenoid papulosis, squamous papilloma and verrucous carcinoma by identifying koilocytes and an orderly arrangement of epithelial cells with a sharp border between epithelial proliferations and the dermis.[1,2,4,5] In deep palmoplantar warts the cytoplasm contains numerous eosinophilic keratohyaline granules in the lower epidermis. In the upper stratum malpighii they coalesce to form large, irregularly shaped, homogenous inclusion bodies. Few cells in the upper stratum malpighii with vacuolated nuclei contain a small intranuclear eosinophilic inclusion bodies. These characteristic intracytoplasmic and intranuclear eosinophilic inclusion bodies differentiate this lesion from verrucous carcinoma and punctate keratosis.[1,2,4] The hallmark of molluscum contagiosum is intracytoplasmic viral inclusion bodies known as molluscum bodies. These molluscum bodies form initially in lower layers of stratum malpighii and as they increase in size they move towards the surface. In the upper layers of epidermis molluscum bodies compress and displace the nucleus to the periphery of the cell. At the level of granular layer the colour of molluscum bodies changes from eosinophilic to basophilic.[1,2,3,5] The vesicles of herpes virus are characterized by nuclear swelling, margination of chromatin, acantholysis and intranuclear eosinophilic inclusions. Identifying characteristic intranuclear eosinophilic inclusions helps to differentiate from erythema multiforme, bullous diseases, pityriasis lichenoides, graft versus host disease and connective tissue disorders.[2,3] Varicella and herpes zoster are histologically indistinguishable from herpes simplex. Certain histological changes such as vessel wall damage, microthrombi, haemorrhage and intranuclear eosinophilic inclusions in capillary endothelial cells are more common in varicella and herpes zoster.[1,2] Except one verruca vulgaris which occurred in unusuall site (ear lobule), all the remaining cases in our study are from extremeties. In our study human papilloma virus(HPV) was the most common viral infection causing warts. Viral skin lesions were transmitted by close, repeated contact often sexual in nature and self inoculation in our study. Two cases of molluscum contagiosum in our study group were identified in the mother and her child presenting simultaneously. Several variants of warts occur, depending primarily on the HPV subtype but also on the anatomic features of the region.[1,6]

Table 2: Types of warts and their HPV subtypes

Types of warts	HPV subtypes
Verruca vulgaris	1,2,4,7
Condyloma acuminatum	16, 18, 31, 33
Palmoplantar wart	1,2,3,4, 27,29

Replication of DNA viruses takes place inside the epidermal cells. Through cell mediated immunity and by immune complexes, they lead to focal necrosis and

secondary inflammatory response which further produces histological changes such as spongiosis, balloon degeneration, acantholysis, nuclear inclusions and inflammatory response and finally causes cutaneous eruptions. Most of the RNA viruses do not replicate intracellularly and the eruption resolves as the virus gets cleared from the skin.[1,2,6]

Table 3: Major families of virus and their site of multiplication[1]

Families	Individual virus	Site of multiplication
Herpesviridae	HSV1,2 ; VZV ; CMV ; EBV	DNA virus - nucleus
Poxviridae	Small pox ; Molluscum contagiosum	DNA virus - cytoplasm
Papovaviridae	Verruca vulgaris ; Palmoplantar wart ; Condyloma acuminatum	DNA virus - nucleus

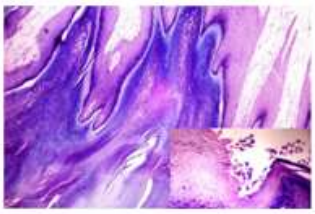

Figure 1: VV with marked hyperkeratosis, hypergranulosis and papillomatosis. HandE 4x Inset- mounds of parakeratosis andE 40x.

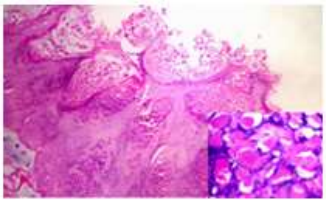

Figure 3: MC with intracytoplasmic molluscum bodies HandE 10x Inset: molluscum bodies HandE 40x

CONCLUSION

Virus can produce warty, bullous and mass like lesions which can mimic non infectious conditions. Histopathological evaluation serves as a valuable tool for identification of virus induced skin changes and aids in appropriate management of these lesions. In our study most common infection was HPV. Knowledge of natural history of viral infection, risk factors, diagnostic tools and therapeutic modalities helps us to prevent, treat and counsel our patients. All these patients must be screened for immunocompromised states.

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HPV viruses have tropism for epithelial cells. The infection is divided into three phases. 1) Latent infection - there is no clinical/ microscopic evidence of disease. 2)Active infection- transcription of HPV takes place in basal cells and viron production in superficial cells(infective viral particles) 3)Progression of infection- the lesions regress spontaneously or persist as benign lesion or progress to precancerous state and eventually to cancer.[1,2,7,8] Immuno compromised hosts are at higher risk because of malignant transformation, especially in anal condyloma which are refractory to standard treatment.[1,6,7] Other laboratory investigations such as isolation of virus in culture, immunoassays, electron microscopy, nucleic acids by PCR and serological rise in antiviral IgG antibody, virus specific IgM antibody help in the identification of subtypes.[2,7,8]

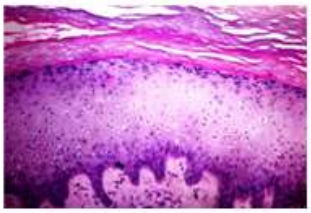

Figure 2: CA with minimal papilomatosis and koilocytic Atypia HandE 10x

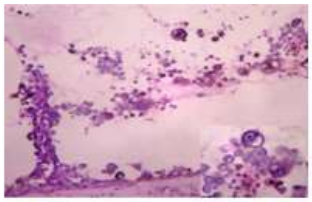

Figure 4: Herpes bullous cavity with acantholytic cells HandE 10x Inset: Intranuclear viral inclusion. HandE 40x

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