Abstract Submissions

2014 JID-Huashan Workshop

May 27-29, 2014

E-Poster Awards

First Prize

Abstract #86: Generation of recombinant anti-collagen VII Fab fragments which compete against autoantibodies in patients with epidermolysis bullosa acquisita Author: Hongjiang Qiao et al Institution: Xijing Hospital, Fourth Military Medical University

Second Prize

Abstract #61: Characterizing the protective roles of mast cells against sporothrix schenckii Infection Author: Zuotao Zhao et al Institution: First Hospital, Peking University

Second Prize

Abstract #53: HLA-B*5901: A marker for Stevens-Johnson syndrome / toxic epidermal necrolysis caused by methazolamide in Han Chinese Author: Fanping Yang et al Institution: Huashan Hospital, Fudan University

Third Prize

Abstract #65: The protective effect of baicalin against UVB irradiation induced photoaging: an *in vitro* and *in vivo* study Author: Jia'an Zhang et al Institution: The First Affiliated Hospital, Nanjing Medical University

Third Prize

Abstract #58: Influence of ALA-PDT on the expression of TLRs in acne lesions and keratinocytes treated with *P. acnes* Abstract #93: Prospective study of topical 5-aminolevulinic acid photodynamic therapy for the treatment of severe adolescent acne in Chinese patients Author: Ying Ma et al

Institution: Huashan Hospital, Fudan University

001

DERMOSCOPY OF PIGMENTED LESIONS OF THE FACE IN CHINA: RESEARCH ON DERMOSCOP

DERMOSCOFT Yan You, Yulian Li, Qingwei Meng, Shuhuai Wang Department of Dermatology, Third Affiliated Hospital, Harbin Medical University, 150 Haping Road, Harbin 150081, P.R.China. E-mail: yyxy1999@strac.om

Abstract: Dermoscopy is a simple-to-use, in vivo method for the diagnosis of cutaneous melanomas and the differential diagnosis of pigmented lesions. Over the past decades, the number of studies on the other sites of the skin has grown exponentially. The dermoscopic patterns for benign and malignant lesions on the face are not well established. The aim of the present study was to analyze the dermoscopic patterns observed in pigmented lesions of the face. We analyzed retrospectively 592 histopathologically proven dermoscopic images of facial lesions. Sensitivity, specificity, diagnostic accuracy values were calculated for defined dermoscopic criteria in relation to the diagnosis of facial pigmented lesions. In current study, 81.65% of benign melanocytic nevi (sensitivity [85.6%], specificity [77.9%], and diagnostic accuracy [72.9%]; 94.65% of seborrhoeic keratoses (sensitivity [96.3%],specificity [90.6%] and diagnostic accuracy [83.5%]), 92.42% of basal cell carcinomas (sensitivity [94.8%], specificity [91.6%], and diagnostic accuracy [82.6%]), 82.14% cutaneous melanomas (sensitivity [85.9], specificity [82.5%], and diagnostic accuracy [73.8%]) were correctly diagnosed by using pattern analysis. Dermoscopy can play a role in the noninvasive classification of facial pigmented lesions. It may be a useful diagnostic technique for evaluating early cutaneous melanoma before biopsy and it is well accepted by patients because of a better cosmetic outcome than surgical excision on the face.

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PROTEIN TYROSINE PHOSPHATASE RECEPTOR TYPE C (PTPRC) RS4915154 POLYMORPHISM INCREASES THE SUSCEPTIBILITY TO PSORIASIS

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Abstract: Psoriasis is a common chronic inflammatory skin disorder and characterized by local Abstract: Psoriasis is a common chronic minimizatory skin disorder and characterized by local formation of inflamed, raised plaques. The prevalence of this disease varies from 0.3 to 3% in world populations with different ethnic background. The exact pathogenesis of psoriasis remains unclear. Clinical and experimental evidences indicate that psoriasis is a T-cell-mediated autoimmune disease. The Protein tyrosine phosphatase receptor type C (PTPRC) gene encodes CD45 protein which is lymphocyte receptor-like tyrosine phosphatase. CD45RO is one of CD45 splicing isoforms and a biological mark of activated memory T lymphocytes. In psoriasis lesion there was a substantial increase CD45RO + T lymphocytes in outer margin than in distant uninvolved skin area. PTPRC single-nucleotide polymorphism rs4915154 (A138G variant) results in an amino acid substitution of Thr-47 to Ala and promotes splicing toward the CD45RO isoforms. We examined the single-nucleotide polymorphism in the PTPRC gene (rs4915154 variant A138G) of psoriatic patients and healthy controls. A total of 160 Chinese northeastern Han population patients with chronic plaque psoriasis were recruited from the department of Dermatology. We investigated PTPRC rs4915154 SNP in 160 psoriatic patients and 132 healthy Demiatology, we investigated PTPRC is 4915154 SiVE in too psoriatic patients and 152 healthy controls by using the denaturing high-performance liquid chromatography technique and sequencing. We found that the frequency of G allele significantly increased in psoriasis cases than in healthy controls 35.3 vs 21.2%, P=0.001). After adjusting for psoriasis risk factors including age, gender, smoking, alcohol drinking and family history of psoriasis, the G allele still showed a significant association with increased psoriasis susceptibility (P=0.008, adjust OR = 1.801, 95%Cl: 1.168–2.777). The PTPRC genotype AG + GG was associated in early-corect (adjusted OR = 2.084, 95% (Cl: 1.623, 5457), having proving proving the bittory (dusted or control of the technic proving the techn onset (adjusted OR = 2.984; 95% CI, 1.632–5.457), having psoriasis family history (adjusted OR = 2.984; 95% CI, 1.632–5.457) and moderate-severe psoriasis patients (adjusted OR = 4.103; 95% CI, 2.196–7.666). We found that variant genotype (AG + GG) of PTPRC increased significantly in psoriasis cases compared with healthy control group and associated with an increased risk of psoriasis in Chinese northeastern Han population. The PTPRC combined AG + GG genotype was significantly associated with early-onset, family history psoriasis patients and those psoriasis patients with PASI score >10. PTPRC rs4915154 polymorphism appears to be associated with an increased risk of psoriasis in Chinese northeastern Han population.

PAPULAR ACANTHOLYTIC DERMATOSIS OF THE ANOGENITAL AREAS MIGHT BE A MILD SUBTYPE OF HAILEY-HAILEY DISEASE

MILD SOBTTPE OF HALEY-HALEY DISEASE Xuemin Xiao', Lihong Chen², Guoyi Zhang¹, Baoxi Wang¹, Weixue Jia¹, Qiuxia Mao¹, Rong Zeng¹, Chengrang Li^{1,3} ¹Institute of Dermatology, Chinese Academy of Medical Sciences, Jiangwangmiao Street 12, Nanjing, Jiangsu 210042, China ²Department of Dermatology, the First Affiliated Hospital, Fujian Medical University, Fuzhou Fujian 350005, China ³Jiangsu Key Laboratory of Molecular Biology for Skin Diseases and STIs, Jiangwangmiao Street 12, Nanjing, Jiangsu 210042, China. E-mail: 258260101@qq.com

Abstract: Papular acantholytic dermatosis (PAD) of the anogenital/genitocrural areas is a rare condition regarded as a variant of focal acantholytic dermatosis. PAD is characterised by variably pruritic, multiple, 0.1-0.5 mm, isolated or grouped, smooth papules confined to the anogenital area, almost without family history. The histological morphology includes acantholysis accom-panied by varying degrees of dyskeratosis and almost all results of immunofluorescence studies were negative. Mutations within ATP2C1, being defective in Hailey-Hailey disease (HHD), in 3 cases of PAD have been reported recently. In our test, we detected ATP2C1 gene for sporadic cases of PAD. Two sporadic cases of PAD were recruited into this study with informed consent. Blood samples were taken from patients, their healthy parents and 100 unrelated normal human Blood samples were taken from patients, their healthy parents and 100 unrelated normal human controls, as well as skin biopsy specimen from patient 2 for genetic analysis. Genomic DNA was extracted from the above samples. All the 28 exons of ATP2C1 gene and the flanking splice sites were amplified by PCR followed by direct sequencing. Sequence comparisons and analyses were performed using Phred-Phrap-Consed Version 12.0 program (http://www.phrap.org). Blood specimen from patient 1 illustrated a heterozygous missense c.1748G>A mutation in exon 18 of ATP2C1. Besides, both blood and biopsy specimen from patient 2 manifested a heterozygous missense c.1570T>C mutation in exon 17 of ATP2C1. Both mutations were not seen in the patients' parents or 100 unrelated controls. Furthermore, these mutations Mutation Dathabac, Cirkon UCRU Single Nucleating Polymers Dathabac or the Mutation Putateon. NCBI Single-Nucleotide Polymorphism Database or the Human Gene Mutation Database. Given the above facts, we believe that c.1748G>A and c.1570T>C are mutations causing PAD. This is the first report, to our knowledge, of sporadic cases of PAD with novel mutations in ATP2C1 gene. Taking both clinicopathologic and genetic overlap with HHD into account, we propose that PAD may be considered not a distinct entity, but rather a mild or localised subtype of HHD.

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CADM1 /TSLC1 INDUCES GROWTH SUPPRESSION AND APOPTOSIS IN MELANOMA CELL LINE A375 Yan You, Jinfeng Zhang, Yulian Li, Yuzhen Li

Department of Dermatology, Third Affiliated Hospital, Harbin Medical University, 150 Haping Road, Harbin 150081, P.R.China. E-mail: yyxy1999@sina.com Abstract: Increasing evidence has demonstrated that CADM1 /TSLC1 (Cell adhesion molecule

1) is crucially implicated in various biological processes including proliferation, apoptosis, and invasion during tumorigenesis. However, its mechanism of suppression of tumor growth and metastasis in melanoma is not known. The purpose of this study was to present if CADM1 / TSLC1 can induce growth suppression and apoptosis in melanoma. The plasmid pcDNA3.1-CADM1 /TSLC1was transfected into A375 cells (a human melanoma cell line). The expression of CADM1 /TSLC1 in the transfected cells was determined by RT-PCR and Western blotting. Cell growth was measured by the MTT method and cell apoptosis by flow cytometry, the ability of invasion was determined by transwell. RT-PCR and Western blotting revealed that pcDNA3.1-CADM1 /TSLC1 expressed higher amounts of TSLC1 protein than pcDNA3.1 and A375 did. The growth of CADM1 /TSLC1-transfected cells was significantly suppressed in vitro, and the ability of invasion was reduced as well, CADM1/TSLC1could induce cell apoptosis. It was concluded that CADM1/TSLC1inhibited cell proliferation, reduced cell invasion in vitro and induced cell apoptosis in A375 cells, suggesting that as a tumor suppressor gene, CADM1 / TSLC1 plays an important role in the progression and metastasis of melanoma

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lesions in an immunocompetent man.

A CASE OF CUTANEOUS PROTOTHECOSIS MIMICS ECZEMA Zhang Qiangqiang, Li li, Kang Yuli, Zhao Ying, Zhu Junhao, Zhu Min Huashan Hospital, Fudan University, Shanghai, China. Abstract: Protothecosis is an opportunistic infection caused by Prototheca, a genus belong to achiorophyllic algae, Prototheca spp, usually as saprophytes, is frequently found in natural and living surroundings, but may cause chronic infection in immunocompromised individuals. In negative the incidence of the disease greated by living and the prototheca which can be apply occur in the skin and subcutaneous tissue, but also involving the internal organs of the human body. We now report a case of Prototheca wickerhamii infection with a specific eczema-like

THE THERAPEUTIC EFFECTS OF DERMAL MESENCHYMAL STEM CELLS CYTOTHERAPY ON VITILIGO MICE

Yiping Zhu, Suiquan Wang, Miaoni Zhou, Qing Li, Aie Xu

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Abstract: The cytotherapeutic potential of mesenchymal stem cells (MSCs) has been evaluated in various disorders including those involving inflammation, autoimmunity, bone regeneration, and cancer. Vitiligo is an autoimmune condition characterized by loss of epidermal melanocytes. However, the effects of dermal mesenchymal stem cells (DMSCs) on vitiligo are unclear. We investigated the effect of DMSCs infusion on the recovery from vitiligo induced by monobenzone in mice. We found that DMSCs delayed the time of appearing depigmentaby infolder2016 in finite. We found that DMSCs and lessened depigmentation area. Moreover, the depigmentation incidence, and lessened depigmentation area. Moreover, the depigmented skin treated with DMSCs indicated gain of epidermal melanocytes by reflectance confocal microscopy (RCM) and the histological examination showed loss perilesional accumulation of CD8+T cells. The levels of inflammatory mediators (IL-6, TNF- α , IFN- γ , and IL-13) were assayed by enzyme-linked immunosorbent assay method (ELISA). Serum cytokine levels were significantly decreased after applicated by DMSCs. Collectively, these data superturbates that processor DMSCs act as wetrander colls to insist the accumulation. data suggest that exogenous DMSCs act as bystander cells to inhibit the occurrence and development of vitiligo.

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