The 11th Annual Meeting
The Korean Society for Psoriasis

PROGRAM

May 19, 2007
COEX InterContinental Hotel Seoul,
Seoul, Korea

Organized by
The Korean Society for Psoriasis
Co-sponsored by
The Korean Dermatological Association
The Korean Society for Investigative Dermatology

인사말씀

대한건선학회 제11차 학술대회를 맞이하여 그 동안 성원하고 지원해 주신 회원 여러분들께 심심한 감사를 드립니다. 대한건선학회는 비록 회원 수는 그리 많지 않으나 매년 기초연구와 임상연구 분야에서 건선 및 유사한 질환들에 대한 발표를 통하여 최신지견 및 경험에 대한 폭넓은 토론의 장이 될 수 있도록 준비하여 왔으나 항시 미진한 부분이 있었습니다. 학회의 발전을 위해 모두가 합심하여야 하며 또한 이들 질환의 치료와 환자들의 삶의 질 향상을 위해 보다 많은 임상가와 연구자가 모여 토의를 함으로써 질환의 병태생리학적·분자생물학적 기전을 이해하고 새로운 치료법을 개발하는 것이 학회의 궁극적인 목표라고 생각합니다. 이를 위해 향후 보다 많은 임상가와 기초 연구자의 참여가 꼭 필요하다고 생각하고 있습니다. 피부과 고유의 영역인 건선과 유사 질환들에 대한 뜻을 가진 많은 분의 참여와 심도 있는 연구를 통해 피부과 의사들의 자긍심이 증진될 수 있다고 생각합니다.

금년도 건선학회는 보다 이러한 목적에 충실하기 위하여 세분의 특강연자를 모셨습니다. Johann Wolfgang Goethe 대학의 Wolf-Henning Boehncke 교수는 젊은 나이에 건선의 면연학적 병태생리에 깊은 연구를 하신 분이며, Kochi 대학의 Shigetoshi Sano 교수는 건선 발병에서 있어서 STAT3의 중요성을 연구하셨으며, Iwate 의과대학의 Toshihide Akasaka 교수 역시 건선의 병태생리학적 기전에 대한 많은 연구를 하신 분으로 세 분 모두 임상적인 면보다 기초학적인 측면에서 건선에 대한 유익한 정보를 제시하실 것으로 생각합니다.

이번 제11차 학술대회를 준비하는 힘써 주신 건선학회 임원 여러분 그리고 동참해 주신 해외 및 국내 초빙 연자, 좌장 및 발표자 여러분께 깊은 감사를 드립니다. 또한 앞으로 여러 회원들의 지속적인 후원과 지도 편달을 바라며, 회원 여러분 가정에 만복이 함께 하시고 건강하시기를 기원합니다.

2006. 5.

대한건선학회 회장 **김 태 윤**

INVITATION

Upon holding the 11th Annual meeting of the Korean Society for Psoriasis, I wish to express my gratitude to all of the members for their continued encouragement and support. Although our society members are still few in number, we continue to hold yearly conventions dealing with basic scientific and clinical studies on psoriasis and other similar diseases. These conventions provide an ideal media for debates on current technologies and updates. However, I have always felt that there was something missing. First of all, I believe that everyone must work together in one accord for the future development of our society. Clinicians and researchers alike should come together in discussion for a better understanding of the pathophysiology and molecular biology of psoriasis, with the ultimate goal of finding a cure and improving the quality of life of patients around the world. Only the participation of those with a purpose and the continuation of in-depth studies can help spread the fact that psoriasis is a dermatological disease, as well as endow dermatologists with additional pride.

This year, in order to follow through with these goals, we have invited three special speakers. From an early age, professor Wolf-Henning Boehncke from Johann Wolfgang Goethe University has performed much research in the field of immunopathophysiology of psoriasis. Professor Shigetoshi Sano of the Kochi University studied the importance of STAT3 in the pathogenesis of psoriasis, and professor Toshihide Akasaka of Iwate medical school also performed extensive research on the pathophysiology of psoriasis. I am sure all three professors will provide us with comprehensive information on the basic science of psoriasis, which is critical in understanding clinical aspect of psoriasis.

Finally, I would like to thank all board members who have lent a hand in preparing this year's convention, as well as all invited speakers, both domestic and from abroad, all chairmen, and all of those present today. I would like to encourage the participation and support of all members in the future, and also wish all members and their families health and happiness.

Thank you very much.

Tae-Yoon Kim, M.D.
President of the KSP

INFORMATION

◆ **Advance Registration**

Not available

◆ **On-site Registration**

Physicians: 20,000 W (including annual membership)
Residents: free

◆ **Official Language**

Oral presentations will be made in Korean language. However, all the presentation material should be prepared in English. Non-Korean participants are allowed to use English language in oral presentations.

◆ **Venue: COEX InterContinental Seoul**

159 Samsung-Dong, Gangnam-Gu
Seoul 135-975, South Korea
Tel: +82 2 3452 2500, Fax: +82 2 3430 8000
E-mail: coexseoul@interconti.com

◆ **Presentation**

Please be advised that slide projection has been completely replaced by beam projection and will be no longer available. Those who would like to use beam projection are advised to use Microsoft PowerPoint (version 2000 or compatible). Double slide projection or overhead projection is not available for the presentation.

- Suggested duration of presentation:

Free communications	7 minutes+3 minute discussion
Case reports	5 minute presentation+2 minute discussion
Educational lectures	13 minute presentation+2 minute discussion
Special lectures	50 minute presentation+5 minute discussion

All the presenters are required to submit their presentation material at least 1 hour prior to the scheduled presentation time. Recommended media for digital files are CD-ROM or USB type memory. Digital files in presenter's notebook computers will not be accepted.

◆ **Social Program**

Standing cocktail party (free admission 6:00-7:00) is planned for all the participants. Please enjoy tasty cuisine and beverage with your colleagues and friends.

PROGRAMS

MORNING SESSION

09:30~10:00 Registration

09:55~10:00 Opening Remark **Tae-Yoon Kim, MD** *(President of the KSP)*

10:00~11:00 Free Communications[FC] / Invited Topic[IT] **Chair: Jee-Ho Choi, MD**

[IT1] ANIMAL MODELS IN PSORIASIS

LEE Jeung-Hoon, MD
Choongnam University College of Medicine

[FC1] EXPRESSION OF INTERLEUKIN-18 BINDING PROTEIN (IL-18 BP) AND INTERLEUKIN-18 RECEPTOR α CHAIN(IL-18Rα) IN PSORIATIC SKIN LESIONS

LIM Sang-Hee, OH Byung-Ho, SONG Young-Chan, KIM Sang-Min, YIM Seon-Mi, LEE Yang-Won, CHOE Yong-Beom, AHN Kyu-Joong
Department of Dermatology, College of Medicine, Konkuk University

[FC2] THE EFFECT OF TNF-α AND INF-γ ON THE TELOMERASE ACTIVITY OF CULTURED HUMAN KERATINOCYTE

HONG Kyung-Kook, M.D.[1], KIM Young-Il,[2] LEE Jin-Woo,[2] PARK Jae-Kyung,[2] KIM Nack-In, M.D.[1]
Department of [1]Dermatology, [2]Immunology Research Laboratory, College of Medicine, Kyunghee University, Seoul, Korea

[FC3] COMPARISON OF EXPRESSION OF HEAT-SHOCK PROTEIN 60, TOLL-LIKE RECEPTORS 2 AND 4, AND T-CELL RECEPTOR γδ IN PLAQUE AND GUTTATE PSORIASIS

KANG Min-Hee,[1] SEUNG Na-Reu,[1] PARK Eun-Joo,[1] KIM Chul-Woo,[1] KIM Kwang-Ho,[1] KIM Kwang-Joong,[1] JO Hee-Jin,[2] PARK Hye-Rim[3]
[1]Department of Dermatology, Hallym University Sacred Heart Hospital, Anyang, Korea
[2]Department of Dermatology, Chun Chon Sacred Heart Hospital, Chun Chon, Korea
[3]Department of Pathology, Hallym University Sacred Heart Hospital, Anyang, Korea

[FC4] EPIDEMIOLOGY OF GENERALIZED PUSTULAR PSORIASIS IN JAPAN

UMEZAWA Yoshinori,[1] AKASAKA Emiko,[1] MABUCHI Tomotaka,[1] MATSUYAMA Takashi,[1] OZAWA Akira,[1] MATSUURA Hironori,[2] IWATSUKI Keiji,[3] and KITAJIMA Yasuo[4]
[1]Tokai University School of Medicine, Japan; [2]Kawasaki Medical School, Japan;
[3]Okayama University, Graduate School of Medicine and Dentistry, Medical School, Japan;
[4]Gifu University Graduate School of Medicine, School of Medicine, Japan

[FC5] FACIAL PSORIASIS; COMPARISON STUDY ACCORDING TO DISTRIBUTION OF FACIAL LESIONS

WOO Seung-Man, CHOI Jung-Won, YOON Hyun-Sun, YOUN Jai-Il

Department of Dermatology, Seoul National University College of Medicine, Seoul, Korea

[FC6] CLINICAL OBSERVATION OF GENERALIZED EXFOLIATIVE DERMATITIS INDUCED BY PSORIASIS

BAE You-In, YUN Sook-Jung, LEE Jee-Bum, KIM Seong-Jin, LEE Seung-Chul, WON Young-Ho

Department of Dermatology, Chonnam National University Medical School, Gwang Ju, Korea

11:00~12:00 Special Lecture[SL] **Chair: Nack-In Kim, MD**

[SL1] **STAT3 AS A TARGET OF TREATMENT FOR PSORIASIS**

Prof. **Shigetoshi Sano**

Department of Dermatology, Kochi Medical School, Kochi, Japan

12:00~13:30 *Lunch*

Council Meeting

AFTERNOON SESSION

13:30~14:30 Special Lecture[SL] **Chair: Jai-Il Youn, MD**

[SL2] **BIOLOGICS IN THE TREATMENT OF PSORIASIS: PATHOGENESIS-ORIENTED THERAPIES FOR UNMET MEDICAL NEEDS**

Prof Dr. Wolf-Henning Boehncke, M.A.

Department of Dermatology, Johann Wolfgang Goethe-University, Frankfurt am Main, Germany

14:30~15:30 Free Communications[FC] / Invited Topic[IT] / Case Presentations[CP]

Chairs: Young-Ho Won, MD / Sang-Tae Kim, MD

[IT2] A RANDOMIZED INVESTIGATOR-BLINDED COMPARATIVE STUDY OF CALCITRIOL TWICE A DAY VS. DIFLUCORTOLONE VALERATE MORNING PLUS CALCITRIOL EVENING APPLICATION IN THE TREATMENT OF MILD TO MODERATE PSORIASIS

LEE Joo-Heung,[1] YOUN Jai-Il,[2] KIM Nack-In,[3] KIM Kwang-Joong,[4] KIM Tae-Yoon,[5] CHOI Ji-Ho,[6] LEW Wook,[7] CHOI Yong-Bum[8]

[1]Sungkyunkwan University School of Medicine, [2]Seoul National University College of Medicine, [3]Kyunghee University, [4]Hallym University, [5]Catholic University, [6]Ulsan University, [7]Yeonsei University, [8]Konkuk University, Seoul, Korea

[FC7] XPRESSION OF ANGIOTENSIN CONVERTING ENZYME IN PSORIASIS

BAK Hana, RHEE Do-Young, JUNG Hae-Jin, CHANG Sung-Eun, CHOI Jee-Ho

Department of Dermatology, Dong-A University College of Medicine, Busan, Korea

[FC8] COMPARISION OF CYCLOSPORINE A AND METHOTREXATE IN THE TREATMENT OF REFRACTORY PSORIASIS

CHOI Myoung-Soon, YUN Sook-Jung, LEE Jee-Bum, WON Young-Ho, LEE Seung-Chul

Department of Dermatology, Chonnam National University Medical School, Gwangju, Korea

[FC9] THE STUDY ON DOSE INCREMENTAL REGIMEN FOR NARROW-BAND UVB PHOTOTHERAPY IN PSORIASIS PATIENTS

WOO Seung-Man, CHOI Jung-Won, YOON Hyun-Sun, YOUN Jai-Il

Department of Dermatology, Seoul National University College of Medicine, Seoul, Korea

[FC10] COMPARATIVE STUDY OF GROSS INTERPRETATION OF PHOTOTEST AND OBJECTIVE MEASUREMENT USING SPECTROPHOTOMETER IN PATIENTS WITH PSORIASIS AND OTHER DERMATOSES TREATED WITH NBUVB

LEE Yeong-Kyu, CHOI Kyu-Won, KU Bon-Seok, KIM Young-Hun, LEE Chae-Wook, KIM Ki-Ho

Department of Dermatology, Dong-A University College of Medicine, Busan, Korea

[CP1] A CASE OF INVERSE PSORIASIS MIMICKING TINEA CRURIS

SONG Young-Chan, OH Byung-Ho, KIM Sang-Min, LIM Sang-Hee, YIM Seon-Mi, LEE Yang-Won, CHOE Yong-Beom, AHN Kyu-Joong

Department of Dermatology, Konkuk University School of Medicine, Seoul, Korea

[CP2] PSORIASIS ASSOCIATED WITH ULCERATIVE COLITIS

WOO Seung-Man, CHOI Jung-Won, YOUN Jai-Il

Department of Dermatology, Seoul National University College of Medicine, Seoul, Korea

[CP3] TACROLIMUS OINTMENT FOR THE TREATMENT OF FACIAL PSORIASIS

CHOI Jung-Won, WOO Seung-Man, YOON Hyun-Sun, YOUN Jai-Il

Department of Dermatology, Seoul National University College of Medicine, Seoul, Korea

15:30~16:00 *Coffee Break*

16:00~17:00 Special Lecture[SL] **Chair: Tae-Yoon Kim, MD**

[SL3] **CYTOKINES AROUND TNF-α FROM ENDOTHELIAL CELLS, LYMPHOCYTES AND KERATINOCYTES IN PSORIASIS VULGARIS**

Prof. Toshihide Akasaka

Department of Dermatology, Iwate Medical University, School of Medicine, Japan

17:00~18:00 Educational Lectures[EL] **Chair: Young-Chul Kye, MD**

PSORIATIC ARTHRITIS (PSA)

[EL1] PSORIASIS AND PSORIATIC ARTHRITIS IN KOREA

Jai-Il Youn *(Department of Dermatology, Seoul National University Hospital)*

[EL2] CLINICAL FEATURES AND DIAGNOSIS OF PSA

Sang-Heon Lee *(Division of Rheumatology, Konkuk University Hospital)*

[EL3] MANAGEMENT OF PSA

Hoon-Suk Cha *(Department of Medicine, Sungkyunkwan University School of Medicine)*

[EL4] SAPHO SYNDROME

Duk-Hyun Sung *(Department of Physical Medicine and Rehabilitation, Samsung Medical Center)*

18:00 Concluding remark

SPECIAL LECTURES

CURRICULUM VITAE

Shigetoshi Sano, M.D., Ph.D.

Chief and Professor
Department of Dermatology
Kochi Medical School
Okocho, Nankoku, Kochi 783-8505, Japan

Date of Birth: February 10, 1957

Adress: Okohcho, Nankoku, Kochi 783-8505, Japan
Office Tel +81-88880-2363, Fax +81-88880-2364
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Education and Training:

1983	M.D., Ehime University Medical School
1983-1984	Resident in Osaka University Hospital (Dermatology)
1984-1988	Ph.D., with Toshiyuki Hamaoka (Immunology), Osaka University Medical School
1988-1992	Postdoctoral fellow with Barry R. Bloom, Howard Hughes Institute, Microbiology and Immunology, Albert Einstein Medical School. NY, U.S.A.
1992-1994	Clinical Director, Department of Dermatology, Sakai Municipal Hospital, Osaka, Japan
1994-2003	Assistant Professor, Department of Dermatology, Osaka University Medical School
2003(June)	Visiting Assistant Professor, Department of Carcinogenesis, The University of Texas M.D. Anderson Cancer Center
2004	Director of Department of Dermatology, Sumitomo Hospital, Osaka, Japan
2005	Assistant Professor, Department of Dermatology, Osaka University Medical School
2006	Associate Professor, Department of Dermatology, Osaka University Medical School
2007-present	Chief and Professor, Department of Dermatology, Kochi Medical School

Societies:

The Japanese Dermatological Association
The Japanese Society for Investigative Dermatology
The Japanese Society for Immunology
The Japanese Society of Allergology
The Japanese Society of Molecular Biology
The Japanese Society of Skin Allergy
American Association for Cancer Research

Reseach Focus:

1. Signal transduction of keratinocytes
2. Mechanism of keratinocyte apoptosis
3. Hair biology
4. Cutaneous gene therapy
5. Ceramides in epidermis and barrier function
6. Biology of thymic epithelial cells and thymus development
7. Pathogenesis of psoriasis
8. Cutaneous Immunology
9. Cutaneous carcinogenesis
10. Metabolic syndrome-associated dermatosis

Invited Addresses and Chairmanship (recent):

1998, "Generation and analysis of keratinocyte-specific Stat3 knockout mice using Cre-loxP system", Plenary speaker of Annual Meeting of at The Japanese Dermatological Association at Osaka.

1999, "Stat3 regulates phosphorylation of focal adhesions required for keratinocyte migration", Speaker in workshop entitled Cell Adhesion and Motility at Annual meeting of Japanese Society for Investigative Dermatology at Kobe.

1999, "Stat3 in wound healing", Invited Speaker in Annual Meeting of Cutaneous Wound Healing Forum at Tokyo.

1999, "Stat3 is required for skin remodeling." Plenary speaker of Annual Meeting of Society for Investigative Dermatology at Chicago.

1999, "Immunology in epidermis" Chair in Annual Meeting of Keratinocyte Study Group at Osaka.

1999, "Stat3 in keratinocyte biology" Invited Speaker in Annual Meeting of Kyushu Investigative Dermatology at Beppu, Oita.

2000, "Stat3 plays an anti-apoptosis role in preventing UVB-induced damage of keratinocytes." Plenary speaker of Annula Meeting of Japanese Society for Investigative Dermatology at Gifu.

2001, "Intracellular signaling of keratinocytes and skin disease". Invited Speaker in Yamanashi Dermatology Association at Kofu.

2001, "Signaling pathways for the hair cycle". Invited Speaker in Annulal Meeting of The Japanese Society for Dermatoallergology at Hamamatsu.

2001, "Intracellular signaling of keratinocytes". Invited Speaker in The Japanese Society for Psoriasis Reseach at Chiba.

2001, "Establishment of keratinocyte-specific ceramide knockout mice". Plenary speaker of Annual Meeting of Japanese Society for Investigative Dermatology at Matsuyama.

2002, "Concurrent Oral Session (Immunology)" Chair in 27th annual meeting of The Japanese Society for Investigative Dermatology at Kyoto.

2004, "Constitutive activation of Stat3 is essential for development of psoriasis" Plenary Speaker of Society of Investigative Dermatology at Province, LI.

2004, Invited Speaker of Annual Meeting of The Japanese Society for Psoriasis at Yamagata

2005, "Forced expression of Activated Stat3 in epidermal keratinocytes reveals a novel role

in malignant conversion of skin tumors" Plenary speaker of Annual Meeting of Japanese Society for Investigative Dermatology at Yokohama.

2005, "Stat3 activation required for development of psoriasis" Invited Lecture at Annual Meeting of Japanese Society for Psoriasis Research.

2006, "Stat3 as a master player for the development of psoriasis and skin cancer" Japanese Society of Investigative Dermatology Award Lecture.

Awards:

2000. Minami Award (Best research award for dermatology in Japan).

2001. Galderma Award

2005. Eugene Farber Award (Best research award for Psoriasis Research in the world)

2006. Japanese Society of Investigative Dermatology Award

Publication List:

1. Nakajima H, Fujiwara H, Takai Y, Izumi Y, Sano S, Tsuchida T, Hamaoka T. Studies on macrophage-activating factor (MAF) in antitumor immune responses. I. Tumor-specific Lyt-1+2- T cells are required for producing MAF able to generate cytolytic as well as cytostatic macrophages. J Immunol 1985;135:2199-205.
2. Tomita S, Fujiwara H, Yamane Y, Sano S, Nakajima H, Izumi Y, Arai H, Kawanishi Y, Tsuchida T, Hamaoka T. Demonstration of intratumoral infiltration of tumor-specific Lyt-1+2- T cells mediating delayed-type hypersensitivity response and in vivo protective immunity. Jpn J Cancer Res 1986;77:182-9.
3. Sano H, Kosugi A, Sano S, Fujiwara H, Hamaoka T. The augmentation of tumor-specific immunity using haptenic muramyl dipeptide (MDP) derivatives. II. Establishment of tumor-specific immunotherapy models utilizing MDP hapten-reactive helper T cell activity. Cancer Immunol Immunother 1987;25:180-4.
4. Sano S, Izumi Y, Sugihara S, Nakajima H, Fujiwara H, Hamaoka T. The generation of tumor-specific *in vivo* protective immunity in the tumor mass from mice rendered tolerant to tumor antigens. Cancer Immunol Immunother 1987;25:105-10.
5. Sano S, Suda T, Qian JH, Sato S, Ikegami R, Hamaoka T, Fujiwara H. Abrogation of the capacity of delayed-type hypersensitivity responses to alloantigens by intravenous injection of neuraminidase-treated allogeneic cells. J Immunol 1987;139:3652-9.
6. Fujiwara H, Yoshioka T, Nakajima H, Fukuzawa M, Sakamoto K, Ogata M, Sano S, Shimizu J, Kiyotaki C, Hamaoka T Cellular and molecular mechanisms involved in tumor eradication *in vivo*. Development and Recognition of the transformed cell, Plenum Press, 331, 1987.
7. Suda T, Sano S, Hori S, Azuma T, Tateishi N, Hamaoka T, Fujiwara H. Prevention of suppression of alloreactive capacity following intravenous injection of neurominidase-treated allogeneic cells by co-injection of agents competing for asialoglycoprotein receptor. Reg Immunol 1988;1:24-31.
8. Sano S, Kiyotaki C, Tatsumi Y, Fujiwara H, Hamaoka T. Cytotoxic T lymphocyte unresponsiveness induced by prolonged treatment with immobilized anti-CD3 antibody. Association of impairment of cytolytic activity with temporary depletion of intracellular protein kinase C. J Immunol 1989;143(9):2797-805. (Corresponding author)
9. Sullivan L, Sano S, Pirmez C, Salgame P, Mueller C, Hofman F, Uyemura K, Rea TH, Bloom BR, Modlin RL. Expression of adhesion molecules in leprosy lesions. Infect Immun. 1991;59:4154-60.
10. Tanaka Y, Sano S, Nieves E, De Libero G, Rosa D, Modlin RL, Brenner MB, Bloom BR, Morita CT. Nonpeptide ligands for human gamma delta T cells. Proc Natl Acad Sci U S A 1994;91:8175-9. (Co-first author)
11. Sano S, Kume S, Nishitani N,Higashi N. Occupational pigmented dermatitis from raw materials of azo dyes, 2,4,5-trichloroaniline and 4-benzamide-2,5-diethoxyaniline. Envi Dermatol 1994;1:195-198. (Corresponding author)
12. Komamura H, Maeda T, Higashiyama M, Sano S, Yoshikawa K. Three cases of contact dermatitis due to ketotifen fumarate. Envi Dermatol 1996;3:91-96.

13. Sano S, Matsui Y, Itami S, Yoshikawa K. Immunological study on CD3 defective cutaneous T cell lymphoma cells from a patient with Sezary syndrome. Clin Exp Immunol 1998;113:190-7. (Corresponding author)
14. Miura H, Sano S, Higashiyama M, Itami S, Yoshikawa K. Candida is not involved in the development of of periungual psoriatic lesion. J Dermatol Sci 1998;18:64-5.
15. Sano S, Itami S, Azukizawa H, Araki Y, Higashiyama M, Yoshikawa K. Interleukin 5-inducing activity in the blister fluid of eosinophilic pustular dermatosis. Br J Dermatol. 1999;141:154-5. (Corresponding author)
16. Sano S, Itami S, Takeda K, Tarutani M, Yamaguchi Y, Miura H, Yoshikawa K, Akira S, Takeda J. Keratinocyte-specific ablation of Stat3 exhibits impaired skin remodeling, but does not affect skin morphogenesis. EMBO J 1999;18:4657-68.
17. Yoshino T, Asada H, Sano S, Nakamura T, Itami S, Tamura M, Yoshikawa K. Impaired responses of peripheral blood mononuclear cells to staphylococcal superantigen in patients with severe atopic dermatitis: a role of T cell apoptosis. J Invest Dermatol 2000;114:281-8.
18. Takeda J, Sano S, Tarutani M, Umeda J, Kondoh G. Conditional gene targeting and its application in the skin. J Dermatol Sci 2000;23:147-54.
19. Sano S, Kira M, Takagi S, Yoshikawa K, Takeda J, Itami S. Two distinct signaling pathways in hair cycle induction: Stat3-dependent and -independent pathways. Proc Natl Acad Sci U S A 2000;97:13824-9.
20. Kawamoto S, Niwa H, Tashiro F, Sano S, Kondoh G, Takeda J, Tabayashi K, Miyazaki J. A novel reporter mouse strain that expresses enhanced green fluorescent protein upon Cre-mediated recombination. FEBS Lett 2000;470:263-8.
21. Miura H, Sano S, Higashiyama M, Yoshikawa K, Itami S. Involvement of insulin-like growth factor-I in psoriasis as a paracrine growth factor: dermal fibroblasts play a regulatory role in developing psoriatic lesions. Arch Dermatol Res 2000;292:590-7.
22. Yamaguchi Y, Kubo T, Tarutani M, Sano S, Asada H, Kakibuchi M, Hosokawa K, Itami S, Yoshikawa K. Epithelial-mesenchymal interactions in wounds: treatment of palmoplantar wounds by nonpalmoplantar pure epidermal sheet grafts. Arch Dermatol 2001;137:621-8.
23. Nakajima T, Sano S, Itami S, Yoshikawa K. Cutaneous inflammatory pseudotumour (plasma cell granuloma). Br J Dermatol. 2001; 144: 1271-3.
24. Sano S, Takahama Y, Sugawara T, Kosaka H, Itami S, Yoshikawa K, Miyazaki J-I, van Ewijk W, Takeda J. Stat3 in thymic epithelial cells is essential for postnatal maintenance of thymic architecture and thymocyte survival. Immunity 2001;15:261-273. (Corresponding author)
25. Sano S, Takeda J, Yoshikawa K, Itami S. Tissue regeneration: hair follicle as a model. J Invest Dermatol Symposium Proceedings 2001;6:43-48. (Corresponding author)
26. Yamaguchi Y, Hosokawa K, Nakatani Y, Sano S, Yoshikawa K, Itami S. Gastrin-releasing peptide, a bombesin-like neuropeptide, promotes cutaneous wound healing. Dermatol Surgery 2002;28:314-319.
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STAT3 AS A TARGET OF TREATMENT FOR PSORIASIS

Shigetoshi Sano

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Psoriasis is one of the most common diseases affecting approximately 2% of the population in Western countries. It has been considered that psoriasis results from complex, aberrant relationships between the skin and immune system, as well as genetic predisposition and environmental factors, although its pathoetiology still remains elusive. Over the last decade, researchers and clinicians have regarded T cells as the predominant contributors to this disease, with the development of models in immunodeficient mice in which psoriatic skin is grafted with and without T cells, and with the introduction of immune-directed therapies, including T cell-targeted immunosuppressive agents and new biologics. In contrast, an aberrancy of epidermis has been also proposed for the pathogenesis of psoriasis, with a hypothesis that psoriatic keratinocytes show an abnormal, exaggerated wound healing response. This hypothesis is clinically highlighted by the Koebner phenomenon, which was originally described as the localization of psoriasis to skin injured by a wide range of stimuli, such as wounding. Since skin wound healing requires activation of keratinocyte Stat3 as we have previously described, we examined the status of Stat3 in the lesional epidermis of psoriatic patients. Strikingly, we found that Stat3 was activated in the lesional keratinocytes from virtually all the psoriatic patients. This up-regulation of Stat3 activation in psoriatic lesions did not appear to be a secondary outcome of epidermal hyperplasia, because lesions from nonpsoriatic inflammatory skin diseases with characteristic acanthosis showed a Stat3 staining pattern similar to normal epidermis. This finding suggested that Stat3 activation in keratinocytes might be necessary for development of psoriasis. To this end, we took advantage in K5.Stat3C transgenic mice, whose epidermal keratinocytes harbored constitutively activated Stat3. The skin of K5.Stat3C mice appeared normal at birth without histological alterations, however, by 2 weeks of age, their skin was reddened and scaly, hyperkeratotic lesions developed in the tails, in which histological alterations were similar to human psoriasis. Furthermore, a distinct psoriasiform lesion was induced in K5.Stat3C mice following full-thickness wounding, topical treatment with TPA, or stimulus by tape stripping, with a marked similarity in histological alterations to human psoriasis. Thus, these data indicate that constitutive activation of Stat3 in these mice increased the sensitivity of the epidermis to external stimuli, leading to a psoriatic phenotype, similar to

the clinical Koebner phenomenon. RT-PCR analysis, Western blotting, and FACS analysis revealed an alteration of psoriasis-related genes in K5.Stat3C mice. Stat3 activation directly or indirectly impacted on the expression of these genes, and most of them were reportedly up-regulated in human psoriasis. Since an involvement of immunocytes, in particular T cells, is required for development of psoriasis, we examined whether Stat3 activation in keratinocytes and T cells interacted in development of psoriatic lesions in this mouse model. Like human psoriatic lesions, infiltration of CD4 cells was predominant in K5.Stat3C. The grafted skin from K5.Stat3C mice onto nude mice developed psoriatic lesions following tape stripping when in vitro-activated T cells were topically injected, but did not develop either without injection of T cells, nor in the grafts from normal mice even in the presence of T cells. Furthermore, a severe combined immunodeficiency (SCID)-human skin graft model revealed that Stat3 underwent activation in keratinocytes in the psoriasis-converted lesions following injection of pathogenic immunocytes, i.e., CD4 cells. These data provide compelling evidence that Stat3 activation impacts an important link between keratinocytes and immunocytes, both of which interdependently participate in the pathogenesis of psoriasis. Given that Stat3 activation was required for development of psoriatic lesions, an inhibition of Stat3 signaling might reverse the phenotype. Expectedly, a topical pretreatment of K5.Stat3C mice with Stat3 decoy oligonucleotides abrogated the de novo generation of tape stripping-induced psoriatic lesions with less T cell infiltrates. The Stat3 decoy treatment reversed preexisting psoriatic lesions as well, suggesting that an inhibition of Stat3 activation would be a reliable therapy for psoriasis.

CURRICULUM VITAE

Prof Dr. Wolf-Henning Boehncke, M.A.

Personal Data	
Date of birth	January 7th, 1964
Place of birth	Karlsruhe, Germany
Citizenship	German

Education

1970-1974	Grammar school
1974-1982	High school ("Gymnasium")
1982	Early graduation, scholarship of the German Scholarship Foundation

Medical School

1982-1988	Medical schools, Universities of Kiel (Germany) and Glasgow (UK)
1988	Graduation
1988	M.D. degree ("magna cum laude")

Professional Qualifications

1989/90	Postdoc, Natl. Institutes of Health, Bethesda/USA (R.N. Germain)
1991	Resident, Dept. of Dermatology, University of Kiel (Christophers)
1991-1994	Fellow, Dept. of Dermatology, University of Ulm (Sterry)
1994	Board certification in "dermatology"
1995	"Habilitation" Board certification in "allergy"
1996	Assistant professor at the Dept. of Dermatology, University of Frankfurt (Kaufmann)
1998	Board certification in "environmental medicine" Head of the section for allergy and clinical immunology
2003	Full professorship, head of the section for allergy/immunology at the Dept. of Dermatology, University of FRankfurt
since 1998	Investigator or principal investigator in several prospective multicenter studies including studies on biologicals in psoriasis (current number: 24)

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"Mile Stones"

- characterization of the function of β2 microglobulin in antigen presentation Nature 1991, 349: 74
- triggering of psoriasis by bacterial superantigens in a xenogeneic transplantation model Nature 1996, 379: 777, Nature Med 1997, 3: 702

- Characterization of a novel class of small-molecule selektin inhibitors
  Nature Med 2002, 8: 366-372
- Awarded with multiple national and international prizes including the 3 major German prizes related to clinical research in 2003, namely
- Novartis prize for pharmaceutical research
- GlaxoSmithCline prize for clinical research
- Galenus von Pergamon prize for clinical research

Miscellaneous

- Member of 5 scientific societies
- Speaker of the German Psoriasis Study Group
- Continuous funding by the German Research Foundation (DFG) throughout my career
- Nation-wide recognition beyond the field of dermatology and allergy (invitations as speaker to non-dermatological meetings, e.g. meeting of the German Society for Internal Medicine)
- Visiting consultant in private hospitals in Qatar, Kuwait, and Oman

BIOLOGICS IN THE TREATMENT OF PSORIASIS: PATHOGENESIS-ORIENTED THERAPIES FOR UNMET MEDICAL NEEDS

Prof Dr. Wolf-Henning Boehncke, M.A.

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Psoriasis is among the most common and severe skin diseases. High prevalence, severity, a complex pathogenesis, and novel therapies have fuelled activities in the field that comprise experimental, clinical, and epidemiologic investigations, as well as initiatives in evidence-based medicine such as guidelines or registries, and finally pharmaco-economic studies.

Clinical work is substantially influenced by a trend towards recognising patient-reported aspects of the disease. Health-related quality of life and improvement thereof are now considered standard items in the documentation of clinical trials. Noteworthy, biologics have consistently shown to be highly effective in this regard. This is particularly important since patients have reported their frustrations with conventional therapie sins ebveral large surveys.

A “hotspot” of current research focuses on the interdependence between inflammation, diabetes, and atherosclerosis. In line with this hypothesis, myocardial infarction and atherosclerosis have already been documented to be increased among psoriatic patients. Recent research points towards an endothelial cell dysfunction triggered by inflammatory processes involving endothelial cells, platelets, and leukocyte extravazation as key factors. Interestingly, psoriasis may well be the driving force behind the above-mentioned co-morbidities, which therefore may better be regarded “complications” of this severe systemic inflammatory disease.

Approval of several biologics to treat psoriasis poses the question of how to use them optimally. The first comprehensive treatment guideline for psoriasis, published only recently by the German Dermatological Society, points towards evidence-based answers. In this context, attempts were also initiated to define an economically optimal approach to treat psoriasis. To this end, there is substantial evidence that the use of biologics is cost-effective in patients suffering from severe plaque psoriasis.

Plaque psoriasis is increasingly recognised as a severe inflammatory disease, associated with numerous serious co-morbidities. Patient-reported outcomes are relevant when defining evidence-based management strategies. Biologics may open the door towards a better future for many patients, as they are advantageous in many regards, based on the patients’ judgement, and their modes of action tackle key processes in the pathogenesis of psoriasis more specifically. Besides, they are cost-effective in severely affected patients. Finally, the future research agenda has to address even more challenging questions, for example how to prevent co-morbidities such as psoriatic arthritis or atherosclerosis/myocardial infarction.

CURRICULUM VITAE

Toshihide Akasaka

Present Appointments

Chairman and Professor of Dermatology, Iwate Medical University, School of Medicine, Morioka, Japan

Education

1976 Iwate Medical University, School of Medicine, M.D.
1980 Iwate Medical University, School of Medicine, Ph.D.

Professional Training and Experience:

1976-1983 Resident of Dermatology, Iwate Medical University, School of Medicine
1984-1989 Assistant Professor of Dermatology, Iwate Medical University, School of Medicine
1990-1991 Chief Doctor of Dermatology, Iwate Prefectural Central Hospital
1992-1997 Associate Professor of Dermatology, Iwate Medical University, School of Medicine
1993-1994 Research fellow of Dermatopathology, Massachusetts General Hospital, Harvard Medical School
1997-Present Chairman and Professor of Dermatology, Iwate Medical University

Committees and Responsibilities

1976- Committee member in Japanese Dermatological Association
1976- Committee member in Japanese Society for Skin Cancer
1976- Committee member in Japanese Society for Psoriasis

Selected Publications

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2. Akasaka T, van Leeuwen RL, Yoshinaga IG, Mihm MC Bayers HR. Focal adhesion kinase (p125FAK) expression correlates with motility of human melanoma cell lines. J Invest Dermatol 1995;105:104-109
3. Akasaka T, Kon S, Mihm MC. Multiple basaloid cell hamartoma syndrome with alopecia and autoimmune disease (systemic lupus erythematosus). J Dermatol 1996;23:821-824
4. van Leeuwen RL, Yoshinaga IG, Akasaka T, Dekker SK, Vermeer BJ, Byers HR. Attachment, spreading and migration of melanoma cells on vitronectin: the role of $\alpha 4\beta 1$ and $\alpha 5\beta 1$ integrins. Experimental Dermatol 1995;5:308-315
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6. Akasaka T, Onodera H, Matsuta M, Kon S. Cutaneous mixed tumor containing ossification, hair matrix and sebaceous ductal differentiation. J Dermatol 1997;24:125-131
7. Akasaka T, Mori Y, Iwasaki M, Kon S. Trichoblastoma with rippled-pattern. J Dermatol 1997;24:147-178
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cosmetics on the physiological function of the skin. J Iwate Med Ass 2002;52:151-162
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CYTOKINES AROUND TNF-α FROM ENDOTHELIAL CELLS, LYMPHOCYTES, AND KERATINOCYTES IN PSORIASIS VULGARIS

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Histological features of psoriasis are hyperproliferation of epidermal keratinocytes, hyperkeratosis, and infiltration of lymphocytes along with angiogenesis. In these features TNFα and CD4 T cell play an important role also in the adhesion of T cells from the patients with psoriasis to endothelial cells, and hyperproliferation of epidermal keratinocytes.

First stage of CD4 T cell infiltration in psoriasis is adhesion of these cells to endothelial cells, however, few adhesion studies have performed *in vitro* using the lymphocyte fractions of the patients with psoriasis. A significant increase in the adhesion of psoriatic CD4 T cells to both endothelial cultures, human skin microvascular endothelial cells from adult (HMVEC-Ad) and human coronary arterial endothelial cells (HCAEC), compared to healthy CD4 T cells was demonstrated in our *in vitro* cell adhesion assay. Pretreatment of both endothelial cultures with TNFα (1,000 U mL^{-1}) induced the most frequent adhesion of CD4 T cells from the patients with psoriasis among the three inflammatory cytokines examined, such as TNFα, IL-1β, and IFNγ. In both endothelial cultures treated with TNFα, the CD4 T cells from the patients with psoriasis exhibited the significantly frequent adhesion compared to those from healthy individuals. The TNFα-stimulated HMVEC-Ad, which exhibited the most frequent adhesion of CD4 T cells was selected for adhesion-inhibition experiments using monoclonal antibodies (mAbs) against adhesion molecules that are up-regulated at the psoriatic lesions, and the combination of anti-lymphocyte function associated antigen 1 (LFA-1)- and anti-intercellular adhesion molecule 1 (ICAM-1)- mAbs significantly reduced the adhesion of CD4 T cells from the patients with psoriasis, and was most effective among all conditions tested, approximately 69% reduction of adhesion. This combination of mAbs significantly reduced also the adhesion of CD4 T cells from psoriasis patients to TNFα-stimulated HMVEC-Ad, compared to the pretreatment with isotype control mAbs.

These findings indicate that LFA- 1/ICAM-1 interaction plays a major role in the adhesion of CD4 T cells to endothelial cells and TNFα might play an important role for the induction

of adhesion molecules on endothelial cells at the psoriatic skin lesions.

Pathogenesis of epidermal proliferation in psoriasis is still unclear, however, the members of the epldermal growth factor (EGF) family are the major growth factors for the proliferation of epidermis *in vivo*, although a wide range of growth factors, such as fibroblast growth factor, Insulin-like growth factor and nerve growth factor, also stimulate the growth of keratinocytes *in vitro*. Heparin-binding EGF-like growth factor (HB-EGF) and amphiregulin (AR) are the members of EGF family that bind to common EGF receptor (EGFR) in the epidermis. HB-EGF is reported as an autocrine growth factor for human epidermal keratinocyte. Although AR is an autocrine growth factor for cultured human neonatal keratinocytes, the effects of AR on adult-type normal human keratinocyte are unknown. The expression of HB-EGF and AR in epidermis was not specific to psoriatic plaques. On the other hand, in the dermis and the papillary dermis, most of vascular endothelial cells and infiltrating mononuclear cells expressed both HB-EGF and AR in normal skins and psoriatic plaques, and these positive cells were more frequent in psoriasis compared to normal skin. In the *in vitro* growth assay, HB-EGF, not AR, stimulated the proliferation of NHEK-AD at the optimal concentration of 1 ng ml^{-1}. Furthermore, HB-EGF compensated the growth-suppressing effects of TNFα, IL-1β and IFNɣ on NHEK-AD, and TNFα promoted the growth of NHEK-AD at the concentration of 2 and 20 U ml^{-1} in combination with HB-EGF and, in lesser extent, with AR. However, TNFα did not affect the expression of EGFR mRNA in NHEK-AD. The epidermal growth factors and inflammatory cytokines produced in the dermis would be important for the epidermal proliferation in psoriatic plaques and TNFα may play a key role in cooperation with HB-EGF and AR in the proliferation of epidermal keratinocytes at the psoriatic skin lesions.

EDUCATIONAL LECTURES

PSORIASIS AND PSORIATIC ARTHRITIS IN KOREA

Jai-Il Youn, MD

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Psoriatic arthritis, presenting as diverse types of arthritis, is an inflammatory arthropathy occurring in patients with psoriasis. The incidence of psoriatic arthritis, previously known to be about 10% of psoriasis patients, seems to increase with the popular use of early detection methods such as radionucleotide scanning, MRI and ultrasound.

The incidence of psoriasis has a peak in the 3rd decade, followed by the 2nd and the 4th decade whereas that of psoriatic arthritis peaks in the 4th or 5th decades. Thus in general psoriatic arthritis is preceded by the occurrence of psoriasis but the reverse situation or concurrence is not a rarity.

Based on the history taken from 1,351 psoriasis patients that visited psoriasis clinic of Seoul National University Hospital (SNUH), 4.8% complained of arthralgia. However, this is not the true percentage of arthritis because not every patient went through further examinations including imaging studies. Spondylitis was the most common presentation followed by oligoarthritis. DIP joint involvement and polyarthritis was relatively uncommon (6.3%) and monoarthritis was not observed.

Another collaborative study with a rheumatologist that investigated 365 psoriasis patients in SNUH recognized 60 patients with joint symptoms, among which 32 (9%) was finally diagnosed as having arthritis. Peak age was in the twenties followed by thirties, forties, and fifties. In the majority (68.8%) of patients psoriatic arthritis was preceded by the occurrence of psoriasis (mean duration: 12.5 years) but some (18.8%) showed concurrent development of arthritis and psoriasis. In some cases (12.5%) development of psoriatic arthritis was followed by that of psoriasis (mean duration: 5.3years). Asymmetric oligoarthritis was the most common type of involvement (75%) and symmetric polyarthritis and DIP joint involvement were common presentation as well. Monoarthritis and spondylitis was also noted.

The prognosis of psoriatic arthritis is known to be milder than that of rheumatoid arthritis. However, when the onset of psoriatic arthritis is early or if the arthritis is associated with severe skin symptoms, the prognosis could be worse. Recent studies indicating that joint involvement can occur even before the development of clinical symptoms underscore the importance of early detection and proper treatment before joint involvement can proceed to structural changes.

CLINICAL FEATURES AND DIAGNOSIS OF PSORIATIC ARTHRITIS

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Psoriatic arthritis is a disabling condition affecting a sizeable proportion of psoriasis patients. While estimates of polyarthritis accompanying psoriasis range from 5-50%, major textbook estimate the prevalence to be 5-7%. Unlike the classical connective tissue diseases such as systemic lupus erythematosus (SLE) or rheumatoid arthritis (RA), the sexual distribution is equal in peripheral arthritis. However, in spinal involvement, the male to female ratio is almost 3:1. Some 84% of psoriasis patients who develop psoriatic arthritis do so about 12 years after the onset of psoriasis, so it is possible that dermatologists and/or rheumatologists may be in a position to first diagnose psoriatic arthritis.

Approximately 95% of patients with psoriatic arthritis have peripheral joint disease. Another 5% have axial spine involvement exclusively. In 1973, Moll and Wright divided psoriatic arthritis into fiver broad categories: 1. asymmetrical oligoarticular disease 2. predominant DIP joint involvement, 3. Arthritis mutilans, 4. polyarthritis (rheumatoid like), 5. axial involvement. They often overlap, creating a heterogenous combination of joint disease.

The course of psoriatic arthritis can be variable and unpredictable, and an understanding of the clinical characteristics will be helpful for diagnosis. As skin symptoms usually appear before joint symptoms, dermatologists should routinely ask psoriasis patients about morning stiffness and swollen or tender joints and family history. Clinical signs that can be indicative of psoriatic arthritis are the involvement of DIP joints (mostly distinguished characteristics in contrast with RA), enthesitis, and dactylitis. The absence of nail anomalies in someone with DIP joint arthritis argues against a diagnosis of psoriatic arthritis. Axial joint disease may present with spondylitis, which can occur in up to 40% of patients. It can occur without sacroilitis and may affect any level of the spine. In case of sacroilitis, it usually manifests as asymmetrical (unilateral) sacroilitis, unlike the typical bilateral sacroilitis in ankylosing spondylitis. In addition to joint involvement, psoriatic arthritis manifest as extra-articular features, but it has only two major extra-articular features; Nail changes and eye diseases. Nail changes are seen in 80% of patients with arthritis, as opposed to only 30% with psoriasis only. These changes include pitting, transverse ridging, onycholysis, hyperkeratosis, and yellowing. Eye disease includes

conjunctivitis in 20% and iritis in 7%. Iritis is more commonly associated with axial involvement.

Over time, joint space narrowing and erosions lead to joint destruction and deformities in most patients, which contributes to reduction in functional capability and quality of life. Important clinical elements in psoriatic arthritis are enthesitis and dactylitis, which also help to distinguish the condition from RA and osteoarthritis, but can be seen in other spondyloarthropathies. The enthesitis is the anatomic location where tendon, ligament, or joint capsule fiber insert into the bone. Enthesitis may occur anywhere in the body, although common locations include the insertion sites of the plantar fascia, Achilles tendon, and ligamentous attachments to the ribs, spine, and pelvis. Dactylitis, or "sausage digit" is a combination of enthesitis of the tendons and ligaments as well as synovitis involving a whole digital ray.

No specific laboratory diagnostic test has not been identified yet. Rheumatoid factor is usually negative, and antinuclear antibody are no more prevalent in psoriatic arthritis patients than in the general population. As in other inflammatory arthritis, ESR , C-reactive protein, and anemia may vary with disease activity. Analysis of synovial fluid reveals inflammatory nature with predominant neutrophils. Radiological findings somewhat helpful in differentiating other inflammatory arthritis such as RA; these include asymmetric involvement, relative absence of juxta-articular osteopenia, involvement of DIPs, erosion of the terminal tufts (acro-osteolysis), whittling of phalanges, cupping of the proximal portion of the phalanges (pencil-in-cup deformity), bony ankylosis, osteolysis of bone (arthritis mutilans) and sacroilitis with spondylitis changes (usually asymmetric).

The past decade has seen significant strides in our understanding of the pathophysiology of psoriasis and psoriatic arthritis, as well as increased awareness on the part of physicians, patients, and the general public about these diseases and their impact. There are similar processes accounting for the pathophysiology of the skin lesions, arthritis, and enthesitis. Both the skin disease and arthritis can affect the function and quality of life for the patient and their family, and so early recognition and management is essential to prevent further deterioration of the disease.

MANAGEMENT OF PSORIATIC ARTHRITIS

Hoon-Suk Cha, M.D.

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Psoriatic arthritis (PsA) is a complex, multifaceted disease with prominent involvement of peripheral joints, axial joints, periarticular structures (e.g., entheses and other soft tissues, resulting in dactylitis), and the skin and nails. Although PsA was once considered a mild disease for which physicians were reluctant to use disease modifying antirheumatic drugs (DMARDs), it is clear that in some cases the disease results in severe destruction of the joints with disability. The goals of treatment are: to improve the signs and symptoms of disease, prevent damage and disability, optimize functional status and quality of life, and to avoid toxicity.

The treatment of PsA usually begins with nonsteroidal anti-inflammatory drugs (NSAIDs). In general, the efficacy and safety of diverse NSAIDs are similar, but the choice of an NSAID should be tailored according to the individual patient's comorbid conditions because a slightly different toxicity profile exists between different NSAIDs. For example, selective COX-2 inhibitors are recommended in patients with increased risk of gastrointestinal toxicity, but, on the contrary, selective COX-2 inhibitors should be avoided in patients with coronary artery disease.

Use of systemic glucocorticoids in patients with PsA is avoided, since there is a chance of developing pustular psoriasis.

DMARDs are employed when the arthritis does not respond to NSAIDs. Patients with polyarticular involvement may benefit from early introduction of DMARDs. DMARDs should definitely be employed when there is progression of deformities or radiographic evidence of erosive disease. Despite the lack of clear evidence of efficacy, methotrexate is the most commonly used conventional DMARD in clinical practice. Sulfasalazine has a well-demonstrated published efficacy according to a meta-analysis, especially for extraaxial arthritis. Leflunomide was also proven effective for both arthritis and skin disease in a randomized controlled trial. Open-label studies showed that cyclosporine was effective in the treatment of PsA. Addition of cyclosporine to methotrexate may result in additional improvement. Azathioprine was also shown to be effective in a randomized controlled trial. While these conventional DMARDs have demonstrated the ability to reduce inflammatory joint activity in the short term, it is not known whether they retard radiographic disease progression in the long term. Every DMARD

has its peculiar potential side effects, and regular monitoring for possible toxicity is necessary.

Introduction of tumor necrosis factor (TNF)-α inhibiting agents allowed the physicians to achieve higher goals in the treatment of PsA. Controlled trials with etanercept, adalimumab, and infliximab have demonstrated statistically significant improvement in a number of arthritis response measures including American College of Rheumatology (ACR) response and Psoriatic Arthritis Response Criteria (PsARC). Psoriasis also improved significantly when measured by Psoriasis Area and Severity Index (PASI). There are data supporting the inhibition of radiographic structural progression for etanercept, adalimumab, and infliximab. These agents are of benefit both as monotherapy and as add-on therapy to other DMARDs such as methotrexate. Etanercept and adalimumab have been approved for the treatment of PsA by the Korea FDA. Screening for latent tuberculosis infection is mandatory before initiating anti-TNF-α therapy. In addition to the usual contraindications to the use of anti-TNF therapies, these agents should be used cautiously in patients with prior PUVA therapy (>1000 joules) due to an increased risk of nonmelanoma skin cancer, and in HIV/AIDS patients due to a general lack of data on the safety of anti-TNF therapy in this setting.

T cell targeted agents, alefacept and efalizumab, which have been known to be efficacious for plaque psoriasis, showed conflicting results in the treatment efficacy of PsA. While alefacept, a lymphocyte function-associated antigen 3 (LFA-3)/immunoglobulin G fusion protein, in combination with methotrexate was effective for PsA treatment, efalizumab, an antibody directed against CD11a, was not effective in treating PsA.

In summary, management of PsA is comprised of NSAIDs, conventional DMARDs, and anti-TNF therapy. The decision to choose a particular treatment should be based on a variety of factors including disease activity, prognosis, comorbid conditions, and individual preferences of each patient.

SAPHO SYNDROME

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The association between sterile inflammatory bony lesions and dermatologic eruptions has been observed, and more than fifty descriptive terms, including pustulotic arthro-osteitis, sternoclavicular hyperostosis, and acne-associated spondyloarthropathy, have been used to describe this spectrum of clinical and radiologic presentation. In 1987, Chanot et al. first coined the acronym "SAPHO" syndrome for rare syndrome characterized by synovitis, acne, pustulosis, hyperostosis, and osteitis. Although "pustulotic arthro-osteitis" proposed by Japanese authors has acquired some popularity, the term SAPHO syndrome become the most widely used term to classify the patients with inflammatory bone and joint problems combined with palmoplantar pustulosis and severe acne or aseptic osteitis predominantly affecting the anterior chest wall. The fundamental component of the SAPHO syndrome is inflammatory, pseudoinfectious, usually sterile osteitis which may or may not be associated with dermatologic components usually with negative bacterial cultures. Osteitis refers to inflammation of bone which involves the cortex and/or medullary cavity. Clinically, the patients complained that the involved area is painful and tender. On histologic examination, infiltrates of inflammatory cells are found. Radiologically, osteitis manifests as osteosclerotic lesions in plain radiography and hyperintensity lesions in T2-weighted magnetic resonance image (MRI). In gadolinium enhanced MRI, those lesions show marked enhancement which represents bone marrow edema. It is often difficult to differentiate the radiologic findings in the SAPHO syndrome form those in other skeletal disorders such as osteomyelitis, lymphoma, metastatic bone tumor, and Paget's disease. In later stage of this syndrome, findings of hyperostosis (ossification of the costal cartilage, thickening of the trabeculae and cortices, and narrowing of the medullary canal) appear in plain radiography. Bone scintigraphy is extremely useful in the detection of skeletal lesions. The main target area in the skeletal system is the anterior chest wall (sternoclavicular joint, manubrium, sternal end of clavicle, upper costochondral junction, upper sternocostal junction, and sternomanubrial junction), with lesser involvement of the spine, pelvis, long bone of the extremities, and mandible. The "bull's head" pattern of increased radioisotope uptake in bone scintigraphy due to anterior chest wall lesions is a highly specific sign of this syndrome. The dermatologic conditions associated with this syndrome generally fall into two categories, pustulosis and severe

acne (acne fulminans; acne conglobata; hidradenitis suppurativa). The prototypical skin lesion is pustulosis palmaris et plantaris which is a systemic inflammatory skin disease featuring sterile pustules, erythema, and scaling of the palms and soles. Of the patients with palmoplantar pustulosis (PPP), the prevalence of arthro-osteitis is variable but generally ranges from 10% to 33%. On the other hand the incidence of acne and PPP in patients with the SAPHO syndrome has been reported as 18.3% to 55.7% respectively. Skin lesions may precede, occur simultaneously with, or follow the onset of osteoarticular manifestations. In most cases (70% of the patients) the interval between the onset of skin and osteoarticular lesions is less than two years but intervals as long as 20 and 38 years have been recorded.

The SAPHO syndrome has the wide spectrum of radiologic and dermatologic presentations. Therefore, not all the components of this peculiar syndrome can manifest simultaneously. However, although the presence of characteristic bone lesions-even in the absence of any dermatomal skin lesions-allows the classification of the SAPHO syndrome, many cases with this syndrome have posed difficult diagnostic problems, particularly in patients without typical skin lesions at presentation because there is no widely accepted, standardized diagnostic or classification criteria of the SAPHO syndrome. Even in fairly typical cases, the link between skin lesions and skeletal manifestations is often missed. This can lead to invasive unnecessary diagnostic procedures and ill-indicated therapeutic procedures, such as long-term antibiotics. Thus, there is a need for increasing the awareness of the SAPHO syndrome among physicians. A high index of suspicion of this syndrome is necessary when the patients with PPP or acne complain pain on the spine and the extremities as well as the anterior chest wall.

FREE COMMUNICATION

Invited Topic

ANIMAL MODELS IN PSORIASIS

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A body of evidences from the recent investigations suggests that psoriasis is a chronic cutaneous disorder where autoimmune reaction based on T lymphocytes is accompanied with abnormal proliferation and differentiation of epidermal keratinocytes. Although genetic studies indicated involvement of genetic elements in the pathogenesis of psoriasis, exact identity of causative sequences remains elusive. Various evidences depict psoriasis as a disease resulting from interaction of multiple genes rather than from single specific sequence. In the elucidation of the mechanism of a complex disorder such as psoriasis in which multiple genes and environmental factors are involved, the role of an appropriate animal model is critical. A proper animal model is also important in the research and development of specific medications. However, development of a proper animal model in the field of psoriasis research has never been complete despite various efforts.

Early animal models having mutations in asebia (ab), flaky skin (fsn), and chronic proliferative dermatitis (cdp) showed skin manifestations and pathological features similar to human psoriasis to some extent. However, they turned out to be lacking immunopathogenetic basis, which was eventually suggested to be a central dogma in the pathogenesis of psoriasis. Later, insights into the key roles of cytokines and adhesion molecules in the pathogenesis of psoriasis prompted development of cytokine or adhesion molecule-related gene-manipulated animal models with successful reproduction of psoriatic skin manifestations. However, significant progress was not made until the advent of SCID mice model and xenotransplantation mice model where dysregulated T cells were demonstrated to reproduce psoriatic plaques nearly identical with human psoriasis.

Animal models in psoriasis, although not complete as yet, has contributed to our understanding of psoriasis pathogenesis through elucidation of roles and interactions of various genes involved in this chronic cutaneous disorder.

EXPRESSION OF INTERLEUKIN-18 BINDING PROTEIN (IL-18 BP) AND INTERLEUKIN-18 RECEPTOR α CHAIN(IL-18Rα) IN PSORIATIC SKIN LESIONS

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Interleukin-18 (IL-18) is a proinflammatory cytokine that stimulates natural killer cells (NK) and T cells and enhances innate immunity as well as specific Th1 immune responses in the pathogenesis of psoriasis. IL-18 is involved in immunologically mediated tissue damage, mediates its effects by binding to a heterodimeric receptor made up of a ligand binding α subunit (IL-18Rα) and a signaling β subunit (IL-18Rβ) but its bioactivity is regulated *in vivo* by its soluble decoy receptor, IL-18 binding protein (IL-18BP). IL-18BP is a soluble circulating protein with high affinity for IL-18 which act as inhibitors of IL-18 signaling and can neutralize its bioactivity, in a similar manner to the IL-1 receptor type II for IL-1, known as IL-18 natural antagonist. IL-18BP can effectively diminish IL-18-induced NF-kB activation, with subsequent synthesis of IFN-γ and IL-8 in vitro. It is now appreciated to be an important regulator of innate and adaptive immunity. It has impressive effects on type I immune responses where it induces Th1/Tc1 lineage differentiation and T and NK cell maturation, stimulates IFN-r production, and regulates macrophage and neutrophil accumulation and function and cellular apoptosis. Recent studies demonstrating that IL-18BP derived from human keratinocytes is upregulated by IFN-γ, suggest that the activity of IL-18 is modulated by a negative feedback mechanism mediated by IFN-γ-induced IL-18BP. However, despite its important contributions to immune responses, the role of IL-18BP and IL-18Rα signaling in the pathogenesis of psoriasis has not been addressed.

Therefore, we attempted to clarify the possible role of IL-18BP production and IL-18Rα signaling in the pathogenesis of psoriasis. In order to characterize this, we compared the level of IL-18BP and IL-18Rα expression between psoriatic skin lesion and normal control using immunohistochemistry, ELISA, and Western blot analysis. Here, we report the results of IL-18BP and IL-18Rα expression in psoriatic skin lesions.

THE EFFECT OF TNF-α AND INF-γ ON THE TELOMERASE ACTIVITY OF CULTURED HUMAN KERATINOCYTE

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Psoriasis is a chronic inflammatory skin disease characterized by erythematous scaly patch or plaques accompanied by hyperproliferation and abnormal differentiation of the lesional epidermis. These processes are thought to be mainly driven by various inflammatory cytokines such as TNF-α and IFN-γ from infiltrated T-cells. Although many chronic inflammatory conditions can result to cancer development, there are no evidences of increased incidence of cancer development in psoriatic skin lesion.

Telomerase is an enzyme-reverse transcriptase that protects chromosomes form degradation by stabilizing telomere length. Recent studies suggest that telomerase activity may be responsible in some part of nonmalignant proliferate skin disease. In addition, there was evidence that telomerase activity is relate with proliferation and differentiation of keratinocyte.

In this experiment, we tried to evaluate the effect of key cytokines such as TNF-α and IFN-γ to the telomerase activity and its differential effect thought to be the passage based on the hypothesis that cells of early passage contain more proliferatory compartment such as stem cells or transit amplifying cells. The result showed increased telomerase activity according to stimulation of each cytokine and the increased extent was differed according to the passage of cultured keratinocyte. Regarding these results, we found that the key cytokines of psoriasis such as TNF-α and IFN-γ increased telomerase activity at proliferative cells, which could contribute to hyperproliferation and abnormal or incomplete differentiation of lesional keratinocyte. Moreover, these results could suggest that increased telomerase activity partially contributed to the reason of no increased incidence of skin cancer in the psoriatic skin lesion.

COMPARISON OF EXPRESSION OF HEAT-SHOCK PROTEIN 60, TOLL-LIKE RECEPTORS 2 AND 4, AND T-CELL RECEPTOR γδ IN PLAQUE AND GUTTATE PSORIASIS

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Background: Psoriasis is a chronic skin disease that appears to be autoimmune nature. Recently, it is thought that microbial pathogens of skin can affect the pathogenesis of psoriasis by inducing autoimmunity. HSPs are known to play an important role in immune and inflammatory responses of the skin including psoriasis. Recent studies have suggested that TLR2, 4 and TCRγδ may recognize HSP60 as a ligand and consequently activate the immune system.

Methods: The biopsy specimens of 12 of guttate psoriasis, 12 of plaque psoriasis, and 5 of normal skin were studied using immunohistochemical staining. The expressions of HSP60, TLR2, and TLR4 were evaluated using an immunostaining-intensity-distribution index, and TCRγδ positive cells were counted.

Results: The expression of HSP60 was significantly higher in guttate and plaque psoriasis than in normal skin. The expression of TLR4 was higher in guttate psoriasis than in plaque psoriasis and normal skin. The expression of TCRγδ was higher in guttate and plaque psoriasis than in normal skin, but there was no correlation found between the expression of HSP60 and TLRs 2 and 4, or between that of HSP60 and TCRγδ.

Conclusions: HSP60 may be related to the pathogenesis of both guttate and plaque psoriasis and TLR4 may be related to the pathogenesis of guttate psoriasis.

EPIDEMIOLOGY OF GENERALIZED PUSTULAR PSORIASIS IN JAPAN

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In order to delineate the clinical outline of Generalized Pustular Psoriasis (GPP), the epidemiological aspects of GPP in Japan were examined through registered case-cards of the Ministry of Health, Labor and Welfare from 588 patients in 2004. We have examined the following topics in children and adults. 1) progress over one year, 2) frequency of relapse for that year, 3) clinical symptoms of the patients worst condition in that year, 4) data of blood examination white blood cell (WBC), and erythrocyte sedimentation rate [ESR], and 5) treatments modalities at start and at present. We have compared the frequency of the patients with GPP in children (Ch) and adults (Ad).

The number of children (age?15) with onset of pustulization was 77 (male;33, female;44), and adults (15＜age) number was 511 (male;269, female;262). 1) Regarding progress the results showed that Ch;30.0% and Ad;41.3% patients improved, Ch;46.8% and Ad;47.8% patients were stable, while Ch;15.6% and Ad;6.3% patients' conditions worsened. 2) Regarding relapse into pustulization, Ch;72.7% and Ad;50.3% suffered relapse. Of these patients, Ch;66.1% and Ad;47.8% patients relapsed more than twice in the year. 3) Regarding clinical symptoms of the patients' worst condition, Ch;61.0% and Ad;50.9% patients developed erythema over 50% of their body surface, and Ch;28.6% and Ad;15.1% patients developed pustules over 50% of their body surface. 4) For blood examination, Ch;9.0% and Ad;11.2% patients' WBC was over 10,000/mm^3, Ch;5.2% and Ad;2.9% patients' ESR was over 60 mm. 5) Regarding the starting treatment, etretinate was administered for Ch:41.6% and Ad:56.1%, cyclosporine was administered for Ch;41.6% and Ad;40.7%, topical steroids were administered for Ch;81.8% and Ad;86.5%, topical vitamin D3 was administered for Ch;54.5% and Ad;56.4%, and phototherapy was administered for Ch;29.8% and Ad;23.9%. At present treatment at that time was, etretinate was being administered for Ch;33.8% and Ad;41.9%, cyclosporine was being administered for Ch;54.6% and Ad;34.6%, topical steroids were being administered for Ch;75.3% and Ad;85.5%, topical vitamin D3 was being administered for Ch;68.9% and Ad;69.5%, and phototherapy was being administered for Ch;7.8% and Ad;7.8%. From these results, for children with GPP are suffering from relapse pustulization, the worsening symptoms are often difficult to restrain compared to the adult patients.

FACIAL PSORIASIS; COMPARISON STUDY ACCORDING TO DISTRIBUTION OF FACIAL LESIONS

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Psoriasis on the face gives rise to considerable concern because of its cosmetic problems and psychosocial distress. Several reports have suggested that facial involvement might be a marker of severe psoriasis, and the authors proved that by comparison study in the previous report. However, psoriasis patients with periphery of face involvement show different clinical characteristics from those with central facial lesions. Many of them have history of severe scalp psoriasis instead of severe body psoriasis. The purpose of this study is to compare the severity of body and scalp psoriasis between the patients with central facial lesions and peripheral facial lesions.

Total 110 psoriasis patients with facial involvement seen in psoriasis clinic, Seoul national university hospital were enrolled. The severity of psoriasis on whole body, face and scalp were rated using PASI. Patients were categorized into 3 groups according to distribution of facial lesions; peripherofacial (upper forehead and/or periauricular lesions), centrofacial, and combined. Early onset of disease, nail involvement, and extensive treatments were more frequently found in patients with centrofacial. The correlation of progression between face and body psoriasis were lesser in the group with peripherofacial involvement. Peripherofacial involvement was related to high scalp PASI while centrofacial involvement was associated with high whole body PASI. In conclusion, facial psoriasis can be categorized into two different types. Peripherofacial involvement might be consequences of severe scalp psoriasis, and centrofacial involvement might be a marker of severe body psoriasis.

CLINICAL OBSERVATION OF GENERALIZED EXFOLIATIVE DERMATITIS INDUCED BY PSORIASIS

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Exfoliative dermatitis (erythroderma) is frequently the result of generalization of preexisting chronic dermatoses such as psoriasis, atopic dermatitis. Also, various medications, internal malignancies can play an important role in developing the disease. Moreover, some patients remain without particular cause. So far, in several reported series the largest group of patients had preexisting dermatoses. 75 patients with erythroderma who had visited Chonnam National University Hospital between March 1984 and February 2007 were enrolled the study. A through history taking, clinical examination, laboratory test, and skin biopsies were performed. 26 out of 75 patients (34.7%) had history of preexisting dermatoses. Among them, 10 patients (37%) had clinical history or histopathologic findings of psoriasis as underline dermatological disease. 60% of patients with psoriatic erythroderma developed erythroderma in winter, compared with that of 27% in non-psoriatic erythroderma. In the group of patients with a diagnosis of psoriatic erythroderma, the histopathologic examinations were specific for psoriasis in 8 patients (80%). All 10 patients had treated Psoralen-UVA, Narrow band UVB therapy, and medication such as acitretin for correction of preexisting dermatoses, psoriasis. 7 out of 10 patients (70%) show partial remission after treatment, however, no patient reached complete remission. There is statistically significant difference with proportion of patients with complete remission in both non-psoriatic erythroderma (33.8%) and non-dermatoses induced erythroderma (39%).

Invited Topic

A RANDOMIZED INVESTIGATOR-BLINDED COMPARATIVE STUDY OF CALCITRIOL TWICE A DAY VS. DIFLUCORTOLONE VALERATE MORNING PLUS CALCITRIOL EVENING APPLICATION IN THE TREATMENT OF MILD TO MODERATE PSORIASIS

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We conducted a randomized investigator-blinded study comparing efficacy, safety and recurrence rate of calcitriol monotherpay vs. calcitriol and diflucortolone valerate for mild to moderate psoriasis patients in Korea.

A total of 142 subjects (73 in monotherapy, 69 in combination arm) with mild to moderate psoriasis (baseline PASI 3.86±2.92 for monotherapy group; 3.64±2.88 for combination group) were enrolled in the study from 6 centers. Topical application was done for 6 weeks and follow-up observation for recurrence was carried out for additional 8 weeks. Monitoring was performed at baseline, 2, 4, and 6 wks during application period and at 4 and 8 wks after discontinuation of therapy. For efficacy analysis both ITT (n=131) and PP (n=71) sets were analyzed and for safety assessment only ITT set was analyzed. For recurrence rate analysis Kaplan-Meier method was applied for 34 subjects who completed both application and follow-up periods.

Percent PASI reduction, the primary end point, which was assessed at the end of application (wk 6) did not show significant difference between the two groups (26.3% for monotherapy vs. 34.3% for combination, p=0.148 for ITT set). Physician's global assessment (PGA), the secondary end point, did not demonstrate significant difference either (Cochran-Mantel-Haenszel test, p=0.111, ITT set). Although adverse symptoms such as erythema, desquamation, and irritation were more frequently observed in calcitriol monotherapy group (8/67) compared with combination group (1/64), most of the symptoms were mild and transient. Discontinuation of application occurred in 2 subjects in monotherapy group due to irritation and itching but

symptoms were mild and transient. Serum calcium concentration was not different between the two groups at the end of the intervention. Recurrence defined by loss of more than 50% of PASI reduction gained at the end of intervention did not show significant difference between the two groups at 4 (p=0.424, ITT) and 8 weeks (p=0.677, ITT) after discontinuation of therapy.

In conclusion, calcitriol monotherapy was demonstrated to be as good as calcitriol plus diflucortolone valerate not only in terms of efficacy and safety but also in the recurrence rate for up to 8 weeks after discontinuation of application.

EXPRESSION OF ANGIOTENSIN CONVERTING ENZYME IN PSORIASIS

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The pathogenesis of psoriasis is incompletely understood but cutaneous inflammation and wound healing process is probably involved. Serum angiotensin converting enzyme (ACE) was reported to be elevated in patients with psoriasis. It was known that angiotensin II, a product of ACE, act as an endogenous pro-inflammatory molecule and involved in wound healing process. In previous reports, ACE can control cutaneous inflammatory responses by degrading neuropeptides. Furthermore, it was found that insertion/deletion polymorphism of ACE is associated with development of psoriasis in indivisuals from psoriatic families. In this study, we have examined tissue ACE and angiotensin II receptors (AT1/AT2) by immunohistochemical stain, laser scanning confocal microscopic images, western blot, reverse transcription polymerase chain reaction (RT-PCR) and high performance liquid chromatography (HPLC).

In results, increased expression of ACE and AT1/AT2 was found in psoriatic skin. We suggest that ACE is probably related to the pathogenesis of psoriasis by alterations of wound healing process and involvement of inflammation.

COMPARISION OF CYCLOSPORINE-A AND METHOTREXATE IN THE TREATMENT OF REFRACTORY PSORIASIS

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Psoriasis is a chronic relapsing disease which requires various combined, sequential and rotational regimens in an attempt to minimize toxicity and enhance therapeutic efficacy. Cyclosporine A (CsA) and methotrexate (MTX) therapies are highly effective one in the treatment of severe psoriasis which is refractory to conventional treatment. We evaluated efficacy and side effects of CsA and MTX in the treatment of moderate to severe psoriasis (more than 7 PASI score), which were refractory to conventional treatment. Total 32 patients (CsA; 20, MTX; 12) were included in this study, and regular follow-up was performed to assess the efficacy and adverse reaction of the agents. CsA was administered with a low-dose regimen (1.5 - 5 mg/kg/day) over average period of 15.86 weeks. Twelve patients (60%) improved to PASI 50, 2 patients (10%) were aggravated, and 6 patients (30%) showed no change. Relapse of the disease occurred in average period of 5.25 weeks after completion of treatment. Side effects, such as gingival hypertrophy (1), gastrointestinal trouble (1), slightly elevated BUN/Cr level (1), were observed. MTX was administered with a regimen in which usual dosage between 2.5 and 5 mg was given every 12 h, 3 times a week. Average cumulative MTX dose was 225.73 mg in average period of 17 weeks (at least more than 6 weeks). Ten patients (83.4%) improved to PASI 50, and 2 patients (16.6%) showed no change. Relapse of the disease occurred in average period of 6.2 weeks after completion of treatment. Side effects of elevated LFT (1) and hair loss (3) were observed. Interestingly, 2 patients, who switched to MTX from CsA due to no response or side effect to CsA, were markedly improved by MTX therapy at an accumulation dose of 55 mg over 4 weeks. From this study, we found that CsA and MTX are effective systemic agents without serious side effects in the treatment of moderate to severe psoriasis, which were refractory to conventional treatment. MTX is regarded as more favorable agent than CsA in terms of therapeutic efficacy.

THE STUDY ON DOSE INCREMENTAL REGIMEN FOR NARROW-BAND UVB PHOTOTHERAPY IN PSORIASIS PATIENTS

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Narrow-band UVB phototherapy using TL-01 lamps is in widespread use due to its greater efficacy and safety compared with broad-band UVB sources. However, the optimum narrow-band UVB dose incremental regimen which will provide maximum efficacy and safety has yet to be defined. Although currently many authors advocate 10% to 20% dosage increments, other incremental regimens such as 5%, 15%, 40% and fixed incremental regimens are also being used according to clinics. The aim of this study was to compare 10% with 20% incremental regimens in narrow-band UVB phototherapy in psoriasis patients.

Total 128 patients were recruited from psoriasis clinic of Seoul national university hospital between March 2003 and December 2006. Ninety-one patients started narrow-band UVB phototherapy with 20% incremental regimen, whereas 37 started with 10% incremental regimen. Twenty-one (23%) patients who started with 20% increment changed into 10% increment during phototherapy because of adverse events such as burning, erythema or pruritus. However, there was no statistically significant difference between both groups in regard to percentage of adverse effects. The mean number of treatment to achieve grade 4 was significantly higher and treatment duration was also longer in 10% increment group than in 20% increment group. On the other hand, final dose and total cumulative dose were higher in 20% increment group comparing with 10% increment group.

Tentative category: phototherapy

COMPARATIVE STUDY OF GROSS INTERPRETATION OF PHOTOTEST AND OBJECTIVE MEASUREMENT USING SPECTROPHOTOMETER IN PATIENTS WITH PSORIASIS AND OTHER DERMATOSES TREATED WITH NBUVB

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In Korea, narrowband-UVB (NBUVB) phototherapy is widely used for treating psoriasis and other dermatoses, for their excellent effect. According to erythema reaction and phototype, phototherapy protocols are being developed actively. Determination of minimal erythema dose (MED) is important for phototherapy protocol development and diagnosis of photosensitivity disorders. But MED is quite diffucult to get precision and reproducibility, because phototest is subjective assessment-based for erythema. Recently, the spectrophotometer has been used for obtaining objective measurements of delicate changes in erythema and pigmentation.

A total of 14 psoriasis and 10 vitiligo patients who receiving NBUVB phototherapy with skin type III, IV were selected. Phototesting with NBUVB was performed in a light source (Waldmann UV 1000K, Waldmann Co., Germany) equipped with emitting a maximum peak at 311nm. To perform phototesting, ten sites on back skin were vertically exposed to NBUVB to a series of 10 doses among 14 doses between 340 and 1400 mJ/cm^2. We interpreted gross findings of erythema and measured L*a*b* values using spectrophotometer CM-2002 (Minolta Co., Osaka, Japan) at each phototest spot and control skin.

In all subjects, MED were measured in the 490-1000 mJ/cm^2 range (average MED: 679.2 mJ/cm^2). The average of colorimetric values in control skin were L*64.8, a*7.9, and b* 19.8. The L* values of conrol skin only decreased significantly according to the increment of MED (L*value=70.0-0.00776MED, $p<0.001$). We measured $\Delta a^*/\Delta$ NBUVB doses and their average among two adjacent phototest spots. The result is the most highest between just below MED and MED and its average is 0.036. The more apart from MED, the average slopes of each interval have fallen to 0.018, 0.0071. This fact means that the dose-response curve of NBUVB erythema show a sigmoid pattern.

In conclusion, spectrophotometer enables UV erythema to assess objectively and quantitatively, it can make up for the disadvantages of subjective gross interpretation in the determination of MED. So, spectrophotometer is a very useful instrument in phototherapy protocol development of psoriasis and other dermatoses and diagnosis of photosensitivity disorders.

A CASE OF INVERSE PSORIASIS MIMICKING TINEA CRURIS

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Inverse psoriasis, rare in clinical practice, refers to psoriasis only or mainly occurring at flexural sites, such as the axilla, antecubital fossae, popliteal fossae, and inguinal creases. Typically, it appears as smooth inflamed lesions with minimal or no scale and is particularly subject to irritation due to rubbing and sweating.

We report a case of inverse psoriasis which is morphologically and clinically mimicking tinea cruris.

A 22-year-old male with 2-year history of wax and wane skin lesion visited our clinic. We could find erythematous patches with minimal scale on both inguinal areas. Our first impression was confusing but histopathological examination revealed it to be psoriasis. This case can be an another example of variable clinical features of psoriasis.

PSORIASIS ASSOCIATED WITH ULCERATIVE COLITIS

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Ulcerative colitis is an inflammatory bowel disease which causes chronic inflammation of digestive tract. Although skin lesions are not common in patients with ulcerative colitis, various mucocutaneous lesions have been reported including erythema multiforme, pyoderma gangrenosum, erythema multiforme and psoriasis. The prevalence of psoriasis in ulcerative colitis is significantly greater than in normal population, which suggests that there is a relationship between psoriasis and ulcerative colitis. Also, in the study of histocompatibility antigen linkage, HLA-B27 is strongly associated with ankylosing spondylitis, inflammatory bowel disease and psoriasis.

Authors report of a 50 year old female who showed signs of psoriasis and ulcerative colitis. She has been treated under diagnosis of psoriasis vulgaris since she was 23 years old. When she was 46 years old, she suffered form persistent bloody diarrhea. Colonoscopic examination revealed multiple hemorrhage and ulcerations, and ulcerative colitis was confirmed by mucosal biopsy. HLA-B27 was negative in laboratory findings. She had been taken treatment with sulfasalazine and acitretin.

TACROLIMUS OINTMENT FOR THE TREATMENT OF FACIAL PSORIASIS

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Psoriasis on the face gives rise to considerable concern and psychosocial stress because of its cosmetic problems. Facial involvement of psoriasis requires a different approach than that used for typical plaque psoriasis on other skin areas. The most common treatment for psoriasis is topical corticosteroids. However, facial skin is susceptible to corticosteroid-induced atrophy because of higher percutaneous absorption in this area. Tacrolimus is an immunosuppressive drug that has proved effective in the treatment of psoriasis when administered systemically. Recently, tacrolimus ointment has been suggested to be effective and well tolerated treatment in patients with facial psoriasis.

Hereby we report seven cases of facial psoriasis improved with topical tacrolimus ointment. Included in the cases were the patients with a long-standing history of facial psoriasis, partially controlled with classical topical therapy. 0.1% tacrolimus ointment was applied twice a day without occlusion. Within the first 8 weeks of treatment, every patient achieved good improvement with the use of tacrolimus ointment. The adverse event was mild pruritus in two patients. None of the patients had atrophy, telangiectasia, or striae develop during the treatment. We suggest that tacrolimus ointment may be an alternative to classical options for the treatment of facial psoriasis.

대한건선학회 임원 및 평의원

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