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Proceeding report of the Second Symposium on Hidradenitis Suppurativa Advances (SHSA) 2017

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Proceeding report of the Second Symposium on Hidradenitis Suppurativa Advances (SHSA) 2017

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AUTHOR CONTRIBUTIONS

ES, AA, FGB, RGB, MB, RC, SD, JWF, AG, IHH, LKH, JH, JSK, HLT, EM, RM, HN, AN, CN, APM, ZP, VP, MR, BR, CS, GS, AS, JT, XW, MAL performed the research and wrote the paper.All authors have read and approved the final manuscript.

CONFLICT OF INTEREST

AA has received consulting fees from AbbVie, Actelion, Celgene, Galderma, GSK, InflaRx, Janssen, Kyowa Incyte Leo Pharma Novartis Pfizer Regeneron Roche Sanofi2 Genzyme UCB Valeant. FGB has received consulting fees from AbbVie. He is on the advisory Board for AbbVie, Janssen, and is an investigator for AbbVie, Janssen, Novartis and InflaRx. RGB has no conflicts of interest. MB is a Speaker for Astellas, AbbVie, Amgen, Eli-Lilly, La Roche-Posay, LEO Pharma, Janssen, Novartis, Pfizer, Merck, Valeant. He is on the Advisory Board for AbbVie, Amgen, Bausch Health, Eli-Lilly, La Roche-Posay, LEO Pharma, Janssen, Novartis, Pfizer, Merck, Valeant, and he is also an investigator of clinical trials for AbbVie, Amgen, Astellas, Basilea, Biogen, Celgene, Eli-Lilly, Galderma, Janssen, Johnson & Johnson, Janssen, Merck, Novartis, Ortho Biotech, Pfizer. RC has no conflicts of interest. SD is the speaker for AbbVie, and an investigator for Regeneron and InflaRx. JF has no conflicts of interest. AG has received consulting fees from AbbVie, Asana Biosciences, Janssen, Pfizer, UCB; grant support from AbbVie, UCB, National Psoriasis Foundation.IHH is the President of the HS Foundation, in a non-compensated role, an Investigator for; Lenicura (equipment received, no grant), AbbVie (grant paid to institution), Jansen (grant paid to institution), is or has been a consultant with fees received by UCB, Novartis, Incyte, Pfizer. LKH has no conflicts of interest. JH has no conflicts of interest. JSK is a speaker for AbbVie and has received consulting fees from Incyte and ChemoCentryx.HLT is the founder of Dermveda, Inc. and is an investigator for Factor Therapeutics, Molnlycke, BSN, TissuTech, Ilkos and Medline. EM has no conflicts of interest. RM has no conflicts of interest. MAL has received consulting fees from AbbVie, Xbiotech, Janssen, BSN: consultant/ Ad boards, and consultant on clinical trial for Incyte. HBN has received a research grant from Abbvie. AN has received travel grant from AbbVie, MSD. CN has no conflicts of interest. APM is a consultant for non-marketing materials for AbbVie and has received fees, serves or has served on an Advisory Board for AbbVie and has received fees, and serves or has served as a consultant for BSN and has received fees, is the President & Founding Director of Hope for HS in a non-compensated role and is an employee of the HS Foundation. ZP has no conflicts of interest. VP reports receiving educational grants from Abbvie, Celgene, Janssen, Naos, Lilly, Sanofi, Valeant, and non-financial support from La Roche-Posay. VP undertakes personal advisory work with Pfizer, AbbVie, Janssen, UCB, Novartis, Almirall and Celgene. MR has no conflicts of interest. BR has no conflicts of interest. CS is the speaker for Abbvie Inc. and for Norvartis, an investigator for UCB, and serves both as an investigator and consultant for InflaRx. GS in the last 12 months has been a consultant for Medline and has received research grants from Medline, B Braun, Next Science, Avadim, and Smith & Nephew. ES has no conflicts of interest. AS in the last 12 months has received research grants from Dabir Inc, PEMF Inc. and Smith & Nephew. JT was an advisor for Abbvie Inc. XW is the speaker for AbbVie, Janssen-Cilag and has received royalties from Springer.

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Abstract

The 2nd Annual Symposium on Hidradenitis Suppurativa Advances (SHSA) took place on 03–05 November 2017 in Detroit, Michigan, USA. This symposium was a joint meeting of the Hidradenitis Suppurativa Foundation (HSF Inc.) founded in the USA, and the Canadian Hidradenitis Suppurativa Foundation (CHSF). This was the second annual meeting of the SHSA with experts from different disciplines arriving from North America, Europe and Australia, in a joint aim to discuss most recent innovations, practical challenges and potential solutions to issues related in the management and care of Hidradenitis Suppurativa patients. The last session involved clinicians, patients and their families in an effort to educate them more about the disease.

Keywords

hidradenitis suppurativa; symposium

1 | INTRODUCTION

Symposium on Hidradenitis Suppurativa (HS) Advances 03–05 November 2017, Detroit, MI, USA. The 2nd Annual Symposium on Hidradenitis Suppurativa Advances was held in Detroit, Michigan from 3 November 2017 to 5 November 2017. This symposium was a joint meeting of the Hidradenitis Suppurativa Foundation (HSF Inc., Santa Monica, CA, USA), founded in the United States, and the Canadian Hidradenitis Suppurativa Foundation

(CHSF).The conference was opened by Dr. Hamzavi, President of HSF, Dr. Marc Bourcier, president of the CHSF.

The purpose of this meeting was to gather well-known researchers from different disciplines to discuss the most recent innovations, practical challenges and potential solutions to issues related to management and care of HS patients. Furthermore, in this meeting, HS patients were involved and attended a HS School that was on the last day of our meeting.

A range of interactive educational sessions on the epidemiology, economic burden, pathophysiology and management of HS were organized. This inaugural meeting was well received by 125 attendees: 95 from Canada, 26 from United States and 4 internationals.

1.1 | Background

Hidradenitis Suppurativa (HS) is a chronic, inflammatory, recurrent, debilitating skin disease (of the terminal hair follicle) that usually presents after puberty with painful, deep-seated, inflamed lesions in the apocrine gland-bearing areas of the body, most commonly the axillary, inguinal and anogenital regions.[1] The disease has female preponderance [1] and has a peak of onset in the second or third decades of life.[1–3] Despite the increased awareness and scholarly attention to HS in the past recent years, HS is still delayed in diagnosis of 7.2 years in average globally.[4] And there is still much more that we need to learn about this disease regarding its pathophysiology, epidemiology and management.

Therefore, more research is required to assess the complexity of this disease and its management.

HS Ultrasound Workshop (pre-conference)

The meeting also included an HS Ultrasound (US) Workshop, that took place in Henry Ford Health Systems in Detroit, Michigan, USA. This US workshop was orchestrated by Dr. Ximena Wortsman, Pontifical Catholic University, Santiago, Chile and Dr. Indermeet Kohli, Henry Ford Hospital, Detroit, MI, USA. This was the first time that the workshop was offered at the SHSA. During the workshop, lectures were given regarding basic ultrasound concepts with an emphasis on ultrasound imaging of the skin in HS. Following didactics, live patients kindly volunteered their time so over 20 trainees could practice what they had learned.

2 | SCIENTIFIC SESSION 1

The conference was opened by Dr. Iltefat Hamzavi, president of HSF, and Dr. Marc Bourcier, president of the CHSF.

2.1 | Epidemiology and comorbidity of Hidradenitis Suppurativa

Dr. Joslyn Kirby from the USA chaired this panel discussion. Other participants were Dr. Marc Bourcier, Dr. Amit Garg, Dr. Afsaneh Alavi, Dr. Vincent Piguet, Dr. Jerry Tan and Dr. Ximena Wortsman.

2.2 | Unmet needs and Research gaps in HS

Marc Bourcier, Canada

Hidradenitis Suppurativa received a lot of attention in the past few years but still has a long way to go. The development of a collaborative framework between healthcare professionals dealing with this chronic and debilitating condition in North America provided a better ground for research in this field. However, unmet needs and research gaps still exist in HS. The main areas for research include early recognition of the disease, recognition of different phenotype, the risk factors for disease progression and eventually management of the disease.

2.3 | Phenotypes, syndromic HS, co-morbidities

Afsaneh Alavi, Canada

HS presents in many ways, the variety of phenotypes including the typical and atypical forms were discussed. Multiple attempts to classify HS phenotypes have been published in the literature from latent class 1–3 and then 6 phenotypic classification such as classic, scaring follicular, syndromic and ectopic type. Apparently not all HS patients are overweight or obese, and BMI is another potential key for classification of HS. Insights into the genetic background of the disease, various clinical presentation of the disease pathophysiology, syndromic forms and highlights of the associated co-morbidities of HS were presented.

2.4 | The incidence of HS in the US

Amit Garg, USA

The true incidence of HS is unknown, as is the utilization of dermatology specialists in the care of HS patients. A retrospective analysis was performed on identified HS patients using electronic health records data. The overall annual incidence of HS in the United States was determined in this study to be 11.4 per 100 000, and more than twice in women than that of men. A rare disease in the United States is defined as a disease that affects less than 200 000 individuals. The conclusion was that HS is an uncommon but not a rare disease.[5]

2.5 | Identifying the True Burden of HS in the UK

Vincent Piguet, Canada

Limited data on the worldwide incidence and prevalence of HS is available, so the true burden of the disease is uncertain. HS patients may be misdiagnosed, a common problem for many patients with HS who often report that they are diagnosed only 5 to 10 years after the start of the condition. Therefore, the group developed algorithms to identify unrecognized “proxy” cases, with at least five Read code records for boils in flexural skin sites. His group performed a UK population-based observational and case-control study on more than 4 million data records. Including “proxy” cases of HS, their results showed prevalence that is closer to 1%. They concluded that this may be a “true” epidemiology of HS in the UK population.[6]

2.6 | Patient oriented outcome measures in hidradenitis suppurativa

Jerry Tan, Canada

This presentation focused on the role of patient reported outcome measures in clinical research including quality-of-life measures. Unlike clinician reported outcome measures which focus on clinical pathologic changes, patient reported outcomes focus on signs, symptoms and impacts important to patients. Appropriate framing of duration for expected improvement is also important in design of outcome measures. Currently available patient reported outcome measures for HS include HSSA, HSIA,[7] HS-QoL[8] and HiSQoL (in development). For each, methods in development and validation where available were reviewed. In an effort to develop a unified quality-of-life instrument, HS-QoL has been amalgamated with HiSQoL which currently consists of 10 domains and 43 items.

2.7 | Role of dermatologic ultrasound in HS

Ximena Wortsman, Chile

The objective of this lecture was to introduce the clinical application and utilization of ultrasound in dermatology for HS. The clinical presentation of HS may be misleading, and on sonography, more advanced signs of involvement may be detected. For example, ultrasound studies may allow visualization of hidden tunnels or fistulous tracts and their connections that the naked eye may not appreciate. Such ultrasonographic findings can modify the staging of the patients; therefore, their management.[9] The presentation of HS cases with clinical-sonographic correlations emphasized these points. This session was also accompanied with a pre-conference workshop on ultrasound.

3 | ORAL PRESENTATION 1: EPIDEMIOLOGY, CO-MORBIDITIES

Dr. Amit Garg from the USA chaired this session

3.1 | Accurate longitudinal lesion mapping in HS: Development of a standardized mapping system for research and clinical use

John W. Frew, Australia

Current scoring systems of HS provide only a snapshot of the amount and variability of lesion types at one point in time. Dr. Frew and his team have proposed a mapping approach for HS lesions. They have developed a system that involves identification of anatomical landmarks that may be documented in three dimensions utilizing ultrasound. This longitudinal lesion mapping system for HS may be applicable for future use both clinically and for research proposes.

3.2 | The Canadian Population-based study of surgically managed HS

Morteza Bashash, Ali Mehdizadeh, Laura Rosella, Afsaneh Alavi (presenter, Canada), R. Gary Sibbald, Delaram Farzanfar, Audrey Laporte, Howard Hu

HS is a chronic debilitating disease that poses also an economic burden to health systems. However, limited studies have appraised the financial burden of this disease. This study was

a population-based study of surgically managed HS in Canada based on international classification of diseases (ICD-10) codes and Ontario Health Insurance Plan (OHIP) billing codes between the years 2002 and 2013. A total of 6244 cases were included in this study. Since the province of Ontario represents ethnically diverse population, the results may be generalizable to other populations outside Canada. Two-thirds of the cases were between 30 and 64 years of age, and 66% were females; 76% of patients over 20 years of age had a high school diploma, and a small subset (9%) lived in the rural neighbourhoods. 94% of patients had moderate to high levels of morbidity, with more than half categorized as moderate. In general, patients with HS impose higher costs at year 1. The first year includes the surgical intervention and its associated costs, which also account for higher costs among men over 60 years of age. There are observed declines in the costs following the index date for both men and women across all ages. For male patients, the annual costs ranged from 4570 to 23 118 CAD for patients under the age of 30 years and older than 65 years. The annual costs for female patients ranged between 3976 to 14 520 CAD for patients under the age of 30 years and older than 65 years.[10] So, the conclusion that can be drawn from this study is that administrative population-based databases may provide essential information to assess the burden of chronic diseases and may shed a light on factors associated with higher cost.[10]

3.3 | Effects of Resilience on depression and health-related quality of life for patients with HS Coping with Hidradenitis

Melissa Butt, USA

Investigation on the coping strategies used by patients with HS to mitigate the effect of HS and depression on quality of life. This was a multi-institutional cross-sectional survey of HS patients at four international sites. Coping styles such as problem-focused and avoidant styles were utilized more by those with a lower quality of life, suggesting that coping styles plays an active role in HS quality of life. The conclusions were to focus on copying styles in future studies, and to teach patients these techniques with the goal of increasing their quality of life.[11]

3.4 | An Assessment of the relative impact of skin disease vs obesity on quality of life in patients with HS

Nicole Mona Golbari (Presenter, USA), MA Storer, ML Porter, AB Kimball

A survey was conducted regarding the impact of obesity versus the skin disease itself on the quality of life for HS patients. The study included 31 participants with the mean BMI of 39, utilizing DLQI relevant to both conditions: The Hurley Stage distribution was I: 3%, II: 58%, III: 39%. The mean total impact for HS versus weight was 16.5 vs 10.3 (maximum of 24), respectively. When comparing individual questions relevant to HS versus weight, QoL scores were all significantly higher for skin disease. The results showed that HS skin disease itself conferred a greater impact on quality of life rather than the obesity.

3.5 | The link between depression and inflammation in HS

Delaram Frazanfar, Yekta Dowlati, Lars French, Michelle Lowes, Afsaneh Alavi (presenter, Canada)

The prevalence of affective disorders such as depression and anxiety is particularly high in autoimmune diseases including inflammatory dermatological conditions such as HS. A literature review spanning across the fields of psychology, neuroscience and dermatology was conducted to gather evidence for the role of inflammation in precipitation of affective disorders in HS patients. Elevated levels of circulating cytokines such as interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-alpha) may exhibit a dose-response pattern with severity of depression.[12]

3.6 | HS: The relationship between pain, depression, and quality of life

Zarine S. Patel (presenter, USA), Elizabeth K. Seng, Steven R. Cohen, Michelle A. Lowes

Pain and distress are core components of the HS experience.[13] This issue was investigated in 201 patients from the Montefiore HS Treatment Center utilizing validated questionnaires. Higher DLQI scores were strongly associated with lower SF-36 pain subscale scores Higher DLQI scores, indicating greater impact on quality of life, were strongly associated with lower SF-36 pain sub scale scores indicating below average health status related to bodily pain and pain interference. The conclusion was that pain and depression are central to the poor quality of life associated with HS.

4 | SESSION 2: PATHOPHYSIOLOGY OF HS

Dr. John W. Frew from Australia chaired this session.

4.1 | Pathophysiology of Hidradenitis Suppurativa

Michelle A. Lowes, USA

The pathogenesis of HS is complex and not well understood.[14] This was an introduction of what we currently know about the role of genes, cytokines, biofilms on pathogenesis and their impact on disease activity. Environmental factors such as obesity and stress may also promote and exacerbate the disease. The resultant chronic cutaneous suppurative inflammation then recruits the systemic immune system. The primary cytokine signature of HS is not yet known. However, IL-6 is a cytokine that may explain some of the systemic manifestations of HS, including acute phase reactants, immunoglobulins, neutrophilia, fever, as well as pain, depression and inflammatory arthritis. This is a unifying hypothesis to integrate cutaneous and systemic inflammation in HS, but further studies are needed to evaluate this concept.

4.2 | Genetics of HS: A systemic review and critical evaluation of reported sequence variants in HS

John W. Frew, Australia

A systematic review of the literature was conducted for studies pertaining to genetic investigations on the background of HS.[15] The large number of mutations of “uncertain significance” is largely due to the variable number of functional studies.[15]

4.3 | Update on the role of bacteria and the skin microbiome in HS

Haley Naik, USA

In recent years, sequence-based genomic approaches have been introduced to provide an unbiased assessment of the microorganisms existing on the skin and mucosal surfaces in health and disease. Microbes are believed to play an important role in initiating and perpetuating HS. While conventional culture-based methods have been used to identify microorganisms in HS lesions, they have failed to isolate organisms in 50% of HS lesions because less than 1% of bacterial species are cultivatable under standard laboratory conditions.

4.4 | Biofilms in Hidradenitis Suppurativa

Gregory Schultz, USA

Recent data strongly indicate that bacterial biofilms play a major role in stimulating the chronic inflammation that characterizes acute and chronic HS lesions. Commensal organisms have been identified in tissues in either prolonged culture methods or in RNA metagenomics sequencing. Biofilm bacterial structures have been histologically detected in a high percentage of chronic lesions and in perilesional samples.[16]

4.5 | The Critical role of alcohol in the pathogenesis of HS

Giovanni Damiani (presenter, Italy), Elena Pezzolo, Ilaria Coati, Nicola Milanesi, Simone Ribero, Michela Iannone, Stefano Veraldi, Emilio Berti

The objective of this study was to determine the role of alcohol in HS patients' lives. This was a prospective observational study that enrolled HS patients for 3 months. The study was conducted via questionnaires that included the Alcohol Use Disorders Identification Test (AUDIT), DLQI and the Autoinflammatory Disease Damage Index (ADDI) and the Sartorius Score. Participants in the study were shown a 10 minutes video that exhibited the deleterious effects of alcohol. The results showed younger patients had higher AUDIT index and lower ADDI and Sartorius scores that were mild to moderate. However, further studies are required to assess whether alcohol plays any role in HS disease.

4.6 | Nicastrin Haploinsufficiency increases inflammatory responsiveness in Keratinocytes cell lines

Elisha D.O. Roberson, USA

In some rare families with autosomal dominant HS, mutations in components of the gamma-secretase complex have been previously identified. The most prevalent mechanism is presumed to be loss-of-function of the Nicastrin subunit. ShRNA knockdown constructs were developed for Nicastrin and their effects investigated on embryonic kidney cells and immortalized keratinocytes. Non-radioactive proliferation assay was performed, and cells that had less Nicastrin showed increased cell death. Furthermore, transcriptome profiling with illumine bead arrays demonstrated significant keratinocyte alterations with Nicastrin knockdown.

4.7 | Biomarkers in Hidradenitis Suppurativa

Lauren K Hoffman (presenter, USA), L. Tomalin, O. Lukatskaya, M. Suarez-Farinas, MA Lowes

No diagnostic test exists for HS, nor have biomarkers been identified that reliably describe disease activity. A literature review was conducted to assess the type of serum biomarkers available in HS.[17] The results failed to exhibit a unique serum diagnostic biomarker for HS. However, many mediators are associated with HS, including IL-32, IL-6, calprotectin (S100A8/A9) and CRP. Hepcidin showed a 3-fold increase in HS patients and is also involved in the pathogenesis of anaemia, which is also interesting since many HS patients were found to suffer from chronic anaemia. Future studies should search deeper into the pathogenesis of HS to reveal more about the disease and its biomarkers.

4.8 | Epigenetic alteration by DNA hypermethylation of genes associated with HS

Uppala Radhakrishna, USA

HS displays genetic heterogeneity that may be traced to specific gamma-secretase gene mutations in some families, whilst these gene mutations are not present in other sporadic cases.[18] A genome-wide DNA methylation scan was performed in a cohort of 24 HS subjects previously excluded from gamma-secretase mutations and 24 controls. A significant CpG hypermethylation was detected at 304 genes in HS subjects.

4.9 | Drug-Induced HS: A systematic review of case reports

John W. Frew, Australia

Rare reports of drug-induced HS do exist. In this study, all published cases were evaluated using the Naranjo criteria. 18 publications reporting 48 individual case reports of HS were identified. The most common incriminating drugs in decreasing order were Adalimumab, Infliximab, Lithium, Etanercept, Rituximab, Vemurafenib and Tocilizumab. The overall incidence for drug-induced HS was low, and the average Naranjo probability scores were only “possible,” indicating the majority of cases had an alternative explanation for the development of HS based upon known risk factors apart from the suggested medication.[19]

4.10 | From Bank to bedside: Establishment of a fresh tissue bank for HS

Ginette A. Okoye, Angel S. Byrd (presenter, USA), Michelle L. Kerns, Carmelo Carmona-Rivera, Philip M. Carlucci, Julie A. Caffrey, Stephen M. Milner, Justin M. Sacks, Oluseyi Aliu, Lloyd S. Miller, Mariana J. Kaplan

This presentation described the establishment of a fresh tissue bank for HS. Tissue resections can be harvested in the operation room as well as biopsies from patients in the outpatient clinic. Tissue sections may be utilized for immunohistochemistry or immunofluorescence, as well as a variety of other studies. The methods outlined in this presentation are available in a companion online supplement, Specimen Collection for Translational Studies in Hidradenitis Suppurativa. An example of utilization of tissues from

the tissue bank is now published, titled, Collagen deposition in chronic Hidradenitis Suppurativa: Potential role for $CD163^+$ macrophages.[20]

5 | SESSION 3: MEDICAL TREATMENTS OF HS

Dr. Marc Bourcier from Canada chaired this session.

5.1 | HS Treatment algorithm (focusing on medical treatment)

Dr. Joslyn Kirby, USA

A current HS treatment algorithm was presented that included a stage-based approach to therapy that utilizes multimodal therapy.

5.2 | Targeted antibiotic therapy combined with surgery for remission of HS: How and why

Aude Nassif, France

Previous studies showed that broad spectrum antibiotics may induce long-term remission for HS patients.[20,21] Targeted antibiotic therapy has been combined with surgery for remission of HS. This study was performed by prospective evaluation of the bacteriology of HS lesions based on Hurley severity score. Antibiotics were provided based on the results of the isolated flora and clinical response observed. The results indicate relapses occurred without appropriate maintenance therapy. Surgery was performed after “cooling down” the flares with targeted antibiotics. The conclusion from this study was that targeted antibiotic therapy can obtain remission in HS, but relapses demonstrate the need for subsequent surgery.

5.3 | Antibiotic stewardship in HS

Mayur Ramesh, USA

A review of the role of bacteria in the pathogenesis of HS, the role of microbiota and biofilm in HS, as well as potential adverse effects of long-term antibiotic use in HS.

This session was followed by a debate session.

Debate on the role of bacteria in HS

The pathophysiology of hidradenitis suppurativa (HS) is poorly understood, including the role and contribution of microbes to HS pathogenesis. A debate was hosted focused on the question of whether bacteria are infectious pathogens in HS. In general, the panelists agreed that bacteria are pathogenic (defined as playing a role in pathogenesis), but there was interesting discussion whether bacteria are causing HS as an infectious disease as we classically understand it or whether bacteria might play a different role in HS pathogenesis.

5.4 | TNF inhibitors in HS

Robert Micheletti, USA

In this presentation, the available evidence supporting the use of TNFα inhibitors for treatment of HS was reviewed. Advanced treatment pearls relevant to the use of anti-TNFα therapy were also discussed. Infliximab and Adalimumab have shown the most promise especially when administered in high dosage according to a systematic review from 2013. Adalimumab was FDA approved based on the PIONEER study which was a breakthrough in the management of HS.[22] Waning efficacy may be caused by the presence of anti-drug antibodies. This phenomenon should be remembered and may be noticed with longer term usage of this drug.

5.5 | Hidradenitis Suppurativa in pregnancy

Jenny Hsaio, USA

HS commonly affects women of childbearing age. Pregnancy may either improve or worsen HS symptoms. Pregnant women should receive the same lifestyle modification advice as other HS patients such as smoking cessation and weight management. Most importantly, medical treatment should obviously have minimal hazardous effects on the foetus. Fortunately, many medications including biologic drugs are FDA pregnancy category B. Medications that must be avoided during pregnancy in HS patients include retinoids, tetracycline antibiotics and hormonal therapy (spironolactone and oral contraceptives). Overall, the session focus on the close attention to the pregnant patient's overall well-being, as pregnancy can exacerbate conditions that have a higher incidence in HS patients, including mood disorders and metabolic syndrome.

5.6 | Emerging therapies in Hidradenitis Suppurativa

Steven Daveluy, USA present recent insights on emerging therapies in HS.

An increased scientific interest on HS, and increased annual number of publications, was marked since the 1st International Conference on HS research, held in 2006 in Dessau, Germany. This increased scientific interest together with activities of the HSF and the EHSF, may have triggered much pharmaceutical interest, that was noticed for example in the publication of the PIONEER study,[23] more studies are being published every year. Break through publications with large numbers of patients have currently been published originating from Europe and Israel, mostly under the auspices of the EHSF, reporting on diagnostic criteria, the dynamic classification and treatment effectiveness evaluation score IHS4, disease course, delay of diagnosis, stratification of obese vs. lean patients and several others.[24–31] However, ongoing search for novel therapy is performed, examples of new therapies for HS with positive results in small sample size studies include Ustekinumab (IL-12/23 inhibitor), Secukinumab (IL-17A inhibitor), Anakinra (IL-1 inhibitor), Liraglutide (a Glucagon-like peptide agonist) was published in a case report, Apremilast (phosphodiesterase 4 inhibitor) was reported in a case series, and IFX1 (anti-C5a monoclonal antibody). Overall, these agents show promising results, but larger randomized controlled studies are required to further asses these novel therapies.

6 | ORAL PRESENTATION 3: MEDICAL TREATMENT OF HS

Dr. Robert Micheletti from the USA chaired this session.

6.1 | The effect of chlorhexidine wash on antibacterial resistance in HS lesions: a retrospective analysis

Dr. Paul Leiphart from the USA

The effects of chlorhexidine wash on antibacterial resistance in HS lesions were studied in a cross-sectional analysis. A cross-sectional analysis was done on patients with HS at Penn State Health from 2005 to 2017. The patients using a concomitant chlorhexidine wash while taking antibiotics did have lower rates of resistance to macrolides (33.33% vs 50.00%), clindamycin (0.00% vs 20.00%), cephalosporins (13.33% vs 44.44%), fluoroquinolones (0.00% vs 5.71%), aminogly-cosides (0.00% vs 5.71%) and daptomycin (0.00% vs 10.00%) than patients who were taking antibiotics without a concomitant chlorhexidine wash. However, none of the differences in the patterns of antibacterial resistance were statistically significant, regardless of the antibiotic class. Using a concomitant chlorhexidine cleanser might decrease the rates of antibiotic resistance in HS lesions, however, we did not detect a significant difference in the patterns of antibiotic resistance with or without the use of a concomitant chlorhexidine cleanser.

6.2 | Gender disparities in the onset of HS in children

Amanda F. Nahhas (presenter, USA), Cynthia L. Nicholson, Angela ParksMiller, Lauren Gold, Iltefat Hamzavi

A retrospective chart review of 33 HS children was divided into 2 groups based on timing of HS onset; prepubescent (n = 12) and post-pubescent (n = 21). In the younger group, there were less females than males and the opposite was found in the older group. Hurley staging and family history of HS did not appear to show significant differences when comparing between prepubescent and postpubescent children with HS in this study.[32]

6.3 | Anti-inflammatory Benefit of Levofloxacin-Metronidazole-Rifampicin in the treatment of HS

Lauren K. Hoffman (presenter, USA), YS Soliman, and SR Cohen

The efficacy of oral antibiotics alone or in combinations as treatment for HS has been shown in previous studies. However, it is still uncertain whether this is due to anti-inflammatory properties or antimicrobial effect or both. A retrospective chart review study of 19 HS patients treated with the “triple antibiotic regimen” (Levofloxadn-Metronidazole-Rifampicin) was performed. The duration of therapy was between 6 and 24 months. The results showed nearly half of the patients experienced an improvement in pain and drainage and 90% reported fewer HS flares.

6.4 | A proof of concept study of the role of topical dapsone in patients with HS

Afsaneh Alavi, Jeannine A, Archer, Dalal Almutairi, Sharon A. Whitty, Monica Grewal, Divine Joyce Briones and Eran Shavit (presenter, Canada)

Dapsone has been used in medicine for a myriad of conditions including HS. Topical treatments in patients with HS have been a challenge and bacterial resistance is an issue with

the commonly used topical clindamycin.[33] Topical dapsone is an FDA-approved application for acne vulgaris.[34] A retrospective chart review of ten HS patients treated with topical dapsone gel 5% was performed. The study was evaluated with patient satisfaction questionnaires and dermatology life quality index (DLQI). This is a preliminary study as recruitment is on-going. The initial results point to improved pain and overall satisfaction with the use of topical dapsone gel. There is a need for randomized trial comparing the effect of topical dapsone to placebo or standard treatment.

6.5 | Intralesional Triamcinolone, a standard of care treatment for Acute HS, does not prove to be superior to placebo

Kristen D. Fajgenbaum (presenter, USA), Chris J. Sayed

Clinical experience and a single cohort study suggest that intralesional triamcinolone may be useful for HS, but its effectiveness has not been extensively assessed. This aim of this study was to assess the efficacy of intralesional triamcinolone at resolving lesions and decreasing pain during acute flares. The study was designed as a randomized, double-blind, placebo-controlled trial comparing intralesional triamcinolone 10 mg/mL, triamcinolone 40 mg/mL, and normal saline as a placebo control. A total of 58 patients were included in this study. The results showed no significant difference between normal saline and intralesional triamcinolone in both dosages in treating acute HS lesions.

6.6 | A retrospective study of 39 HS patients treated with dapsone

Rashie Brar (presenter, Canada), Rosilene Lanzini, Hermenio Lima

The objectives of this study were to evaluate the effectiveness and adverse effects of systemic dapsone as a single treatment or in combination with other regimens for HS. A retrospective review of 39 HS patients was performed. Oral Dapsone was given either as monotherapy (56%) or in combination with adalimumab (41%) and isotretinoin (2.6%). The number of participants achieving clinical response using the reduction of the Hurley Stage from the base line before treatment compared to the Hurley stage after treatment (min 6 months) was obtained. The improvement measurement was defined as at least reduction of <=1 of the Hurley Stage in >=1 affected anatomic region with a minimum of 6 months of therapy. Number of participants worsening, or no response was calculated using the increase the Hurley Stage from the baseline before treatment compared to the Hurley stage after treatment (min 6 months) or no change in the Hurley stage, respectively. The worsening of HS was defined as at least increase by >= 1 Hurley Stage in >= 1 affected anatomic region (minimum 6 months of therapy). Descriptive analysis was done using JMP® v.14. Improvement was seen in all patients using dapsone for over 6 months. Larger studies are required to assess the role of systemic dapsone, especially as monotherapy.

7 | SCIENTIFIC SESSION 4: MANAGEMENT OF HS

Dr. Michelle Lowes from the USA chaired this session.

7.1 | US evidence-based guidelines for HS treatment

Christopher Sayed, USA

Following the existing European guidelines for the management of HS,[35] the HS Foundation and the Canadian HS Foundation joined forces to develop and publish the North American guidelines for the management of HS. This work is ongoing with the goal of publication in 2018.

7.2 | Wound healing in HS

Hadar Lev-Tov, USA

HS is a disease that involves drainage and odour from the wounds. The daily management of the wounds are essential to patients' satisfaction and therefore to treatment success. What kind of dressing should be utilized? How often should they be changed? For these and some more unanswered questions, some practical pearls were presented for the everyday management of chronic wounds in HS patients.

7.3 | IDEOM: development of the core outcome set in HS

Dr. Amit Garg from the USA

Five e-Delphi rounds and four face-to-face consensus meetings were conducted to yield a final core domain set which included; pain, physical signs, HS specific quality of life, global assessment and progression of course.[36,37] Routine adoption of the core domains in future HS trials should ensure that outcomes of importance to both patients and other relevant stakeholders will be collected and will facilitate comparison of outcomes across trials.

7.4 | NIAM: HS research

Dr. Ricardo Cibotti

In this session, Dr Cibotti, the Program Director of Immunobiology and Immune Diseases of Skin Program in NIAM (National Institute of Arthritis and Musculoskeletal and Skin Diseases) discussed the potential resources for skin research particularly HS.

7.5 | HS Foundation (HSF) and research road map

Michelle Lowes, USA

Dr Michelle Lowes extensively discussed the road map of research planned by HSF. She also discussed the unmet need to develop a North American HS consortium.

8 | SCIENTIFIC SESSION 5: SURGICAL TREATMENT OF HS

Dr. Iltefat Hamzavi from the USA chaired this session.

8.1 | Office procedures for HS & the medical dermatologist

Christopher Sayed, USA

HS unlike many other inflammatory dermatologic diseases is often surgically managed. The patients are commonly referred to surgeons for interventions. However, some surgical procedures for HS patients may be accomplished in office-based settings. The focus was on deroofing and excisional procedures. Ideally, dermatologists' will gain comfort approaching certain procedures in selected cases for the benefit of the patients.

8.2 | Where Mohs surgery meets HS

Richard Bennett, USA

Mohs surgery was developed and mainly utilized for the management of cutaneous malignancy, particularly on the face region. This presentation was meant to introduce the application of Mohs surgery in HS disease.

8.3 | Complex surgical situations in the perianal and genital area

Falk Bechara, Germany

HS often requires surgical intervention for advance disease when the disease is under medical control. The perianal and genital areas offer many challenges due to the anatomical locations involved. Dr. Bechara shared his experience of some complex surgical situations in these regions and has provided some important tips on how he was able to overcome these obstacles.[38]

8.4 | Plastic surgery for Hidradenitis Suppurativa

Aamir Siddiqui, USA

The role of plastic surgery for HS was presented through a retrospective review study on 254 patients. Reconstructive efforts were undertaken in 17% of procedures and represented skin grafts or local flaps. For patients undergoing surgery while maintaining participation in the multidisciplinary clinic, 87% of patients were healed at 1-year post surgery, whereas patients who did not maintain follow-up in the multidisciplinary clinic only healed in 45%.

8.5 | Lasers and light Therapies for the treatment of Hidradenitis Suppurativa

Iltefat Hamzavi, USA

Light and laser therapies have been utilized in the field of dermatology to treat a variety of skin conditions, but only recently were studied in the management of HS. Laser and Light therapies that are utilized for the treatment of HS offer several advantages including low risk of complications in comparison to the traditional cold knife surgical procedures. As a preventive and treatment option, laser-assisted hair reduction may be utilized for HS as well. Also, photodynamic therapy (PDT) was introduced in the past few years for the management of HS with promising results.[39] A surgical option includes tissue debulking with the CO2 laser (10 600nm wavelength).

8.6 | Carbon Dioxide Laser surgery for Hidradenitis Suppurativa

Barry Resnik, USA

Presented his experience with Carbon Dioxide (CO_2) Laser excision for HS patients. In his experience, this procedure was less painful, had a lower recurrence rate and resulted in scars that were cosmetically pleasing for the patients.

HS School—The 2nd Symposium on Hidradenitis Suppurativa Advances (SHSA) held an inaugural educational session especially for HS patients, called HS School.

HS School is an opportunity for patients and their caretakers or relatives to hear about HS from experts, in language that the public can understand. There was also a Question and Answer segment at the end of the session where patients can ask our panel general questions about HS.

REFERENCES

[1]. Kurzen H, Kurokawa I, Jemec GBE, Emtestam L, Sellheyer K, Giamarellos-Bourboulis EJ, et al., Exp. Dermatol. 2008, 17, 455. [PubMed: 18400064]

[2]. Beltrame A, Staffolani S, N. Engl. J. Med. 2017;377(25), 2474. [PubMed: 29262280]

[3]. van der Zee HH, Jemec GB, J. Am. Acad. Dermatol. 2015, 73(5 Suppl 1), S23. [PubMed: 26470610]

[4]. Saunte DM, Boer J, Stratigos A, Szepietowski JC, Hamzavi I, Kim KH, et al., Br. J. Dermatol. 2015, 173(6), 1546. [PubMed: 26198191]

[5]. Garg A, Lavian J, Lin G, Strunk A, Alloo A, J. Am. Acad. Dermatol. 2017, 77(1), 118. [PubMed: 28285782]

[6]. Ingram JR, Jenkins-Jones S, Knipe DW, Morgan CLI, Cannings-John R, Piguet V, Br. J. Dermatol. 2018, 178(4), 917. [PubMed: 29094346]

[7]. Kimball AB, Sundaram M, Banderas B, Foley C, Shields AL, J Dermatolog Treat. 2018, 29(2), 152. [PubMed: 28608738]

[8]. McLellan C, Sisic M, Oon HH, Tan J, J Cutan Med Surg. 2017;22, 142. [PubMed: 28992738]

[9]. Wortsman X, JAMA 2018, 319(15), 1617. [PubMed: 29677295]

[10]. Mehdizadeh A, Rosella L, Alavi A, Sibbald G, Farzanfar D, Hazrati A, et al., J Cutan Med Surg. 2018;22(3), 312. [PubMed: 29528753]

[11]. Kirby JS, Butt M, Esmann S, Jemec GBE, JAMA Dermatol. 2017, 153(12), 1263. [PubMed: 29117300]

[12]. Farzanfar D, Dowlati Y, French LE, Lowes MA, Alavi A, Skin Pharmacol Physiol. 2018;31(5), 246. [PubMed: 29953999]

[13]. Patel ZS, Hoffman LK, Buse DC, Grinberg AS, Afifi L, Cohen SR, et al., Curr. Pain Headache Rep. 2017, 21(12), 49. [PubMed: 29094219]

[14]. Hoffman LK, Ghias MH, Lowes MA, Semin. Cutan. Med. Surg. 2017;36(2), 47. [PubMed: 28538743]

[15]. Frew JW, Vekic DA, Woods J, Cains GD, Br. J. Dermatol. 2017, 177(4), 987. [PubMed: 28278367]

[16]. Omar A, Wright JB, Schultz G, Burrell R, Nadworny P, Microorganisms. 2017;5(1), pii: E9.

[17]. Blok JL, Li K, Brodmerkel C, Horvátovich P, Jonkman MF, Horváth B, Br. J. Dermatol. 2016, 174(4), 839. [PubMed: 26641739]

[18]. Ratnamala U, Jhala D, Jain NK, Saiyed NM, Raveendrababu M, Rao MV, et al., Exp. Dermatol. 2016, 25(4), 314. [PubMed: 26663538]

[19]. Frew JW, Vekic DA, Woods JA, Cains GD, J. Am. Acad. Dermatol. 2018, 78(1), 217. [PubMed: 29241794]

[20]. Byrd AS, Kerns ML, Williams DW, Zarif JC, Rosenberg AZ, Delsante M, et al., Br. J. Dermatol. 2018;179, 792. [PubMed: 29603182]

[21]. Join-Lambert O, Coignard H, Jais JP, Guet-Revillet H, Poirée S, Fraitag S, et al., Dermatology 2011, 222(1), 49. [PubMed: 21109728]

[22]. Join-Lambert O, Coignard-Biehler H, Jais JP, Delage M, Guet-Revillet H, Poirée S, et al., J. Antimicrob. Chemother. 2016, 71(2), 513. [PubMed: 26565016]

[23]. Kimball AB, Okun MM, Williams DA, Gottlieb AB, Papp KA, Zouboulis CC, et al., N. Engl. J. Med. 2016, 375(5), 422. [PubMed: 27518661]

[24]. Zouboulis CC, del Marmol V, Mrowietz U, Prens E, Tzellos T, Jemec GBE, Dermatology 2015, 231, 184. [PubMed: 26139027]

[25]. Zouboulis CC, Tzellos T, Kyrgidis A, Jemec GBE, Bechara FG, Giamarellos-Bourboulis EJ, Ingram JR, Kanni T, Karagiannidis I, Martorell A, Matusiak L, Pinter A, Prens EP, Presser D, Schneider-Burrus S, von Stebut E, Szepietowski JC, van der Zee HH, Wilden SM, Sabat R; on behalf of the EHSF Investigator Group. Br. J. Dermatol. 2017;177, 1401. [PubMed: 28636793]

[26]. Shalom G, Freud T, Ben Yakov G, Khoury R, Dreiher J, Vardy DA, Comaneshter D, Cohen AD, J Invest Dermatol 2016, 136, 1716. [PubMed: 27094593]

[27]. Shalom G, Freud T, Harman-Boehm I, Polishchuk I, Cohen AD, Br. J. Dermatol. 2015, 173, 464. [PubMed: 25760289]

[28]. Shalom G, Babaev M, Freud T, Tiosano S, Pam N, Horev A, Dreiher J, Vardy DA, Comaneshter D, Cohen AD, J. Am. Acad. Dermatol. 2017, 77(1047–1052), e2.

[29]. Saunte DM, Boer J, Stratigos A, Szepietowski JC, Hamzavi L, Kim KH, Zarchi K, Antoniou C, Matusiak L, Lim HW, Williams M, Kwon HH, Gürer MA, Mammadova F, Kaminsky A, Prens E, van der Zee HH, Bettoli V, Zauli S, Hafner J, Lauchli S, French LE, Riad H, El-Domyati M, Abdel-Wahab H, Kirby B, Kelly G, Calderon P, del Marmol V, Benhadou F, Revuz J, Zouboulis CC, Karagiannidis I, Sartorius K, Hagströmer L, McMeniman E, Ong N, Dolenc-Voljc M, Mokos ZB, Borradori L, Hunger RE, Sladden C, Scheinfeld N, Moftah N, Emtestam L, Lapins J, Doss N, Kurokawa I, Jemec GBE, Br. J. Dermatol. 2015;173, 1546. [PubMed: 26198191]

[30]. Thorlacius L, Garg A, Ingram JR, Villumsen B, Theut Riis P, Gottlieb AB, Merola JF, Dellavalle R, Ardon C, Baba R, Bechara FG, Cohen AD, Daham N, Davis M, Emtestam L, Fernández-Peñas P, Filippelli M, Gibbons A, Grant T, Guilbault S, Gulliver S, Harris C, Harvent C, Houston K, Kirby JS, Matusiak L, Mehdizadeh A, Mojica T, Okun M, Orgill D, Pallack L, Parks-Miller A, Prens EP, Randell S, Rogers C, Rosen CF, Choon SE, van der Zee HH, Christensen R, Jemec GBE, Br. J. Dermatol. 2018, 178, 715. [PubMed: 29080368]

[31]. Riis PT, Saunte DM, Benhadou F, del Marmol V, Guillem P, El-Domyati M, Abdel-Wahab H, Antoniou C, Dessinioti C, Ali Gürer M, Beksaç B, Szepietowski JC, Matusiak L, Emtestam L, Lapins J, Riad H, Doss N, Massa AF, Hamzavi I, Nicholson C, Dolenc-Voljc M, Kim KH, Ohn J, Zouboulis CC, Karagiannidis I, Durienc P, Bukvic Mokos Z, Jemec GBE, J. Eur. Acad. Dermatol. Venereol. 2018, 32, 307. [PubMed: 28940801]

[32]. Braunberger TL, Nicholson CL, Gold L, Nahhas AF, Jacobsen G, Parks-Miller A, et al., Pediatr. Dermatol. 2018, 35(3), 370. [PubMed: 29575194]

[33]. Fischer AH, Haskin A, Okoye GA, J. Am. Acad. Dermatol. 2017, 76(2), 309. [PubMed: 27742173]

[34]. Draelos ZD, Carter E, Maloney JM, Elewski B, Poulin Y, Lynde C, et al. J. Am. Acad. Dermatol. 2007;56, 439. [PubMed: 17208334]

[35]. Zouboulis CC, Desai N, Emtestam L, Hunger RE, Ioannides D, Juhász I, et al., J. Eur. Acad. Dermatol. Venereol. 2015, 29(4), 619. [PubMed: 25640693]

[36]. Thorlacius L, Garg A, Ingram JR, Villumsen B, Theut Riis P, Gottlieb AB, et al. Br. J. Dermatol. 2018;178(3), 715. [PubMed: 29080368]

[37]. Tholacius L, Ingram JR, Villumsen B, Esmann S, Kirby JS, Gottlieb AB, et al. Br. J. Dermatol. 2018;179, 642. [PubMed: 29654696]

[38]. Scholl L, Hessam S, Bergmann U, Bechara FG, J Cutan Med Surg. 2018;22(2), 239. [PubMed: 29587516]

[39]. Hamzavi IH, Griffith JL, Riyaz F, Hessam S, Bechara FG, J. Am. Acad. Dermatol. 2015, 73(5 Suppl 1), S78. [PubMed: 26470622]