eISSN 2005-8330 https://doi.org/10.3348/kjr.2022.0215 Korean J Radiol 2022;23(8):785-793



Magnetic Resonance Imaging of Hidradenitis Suppurativa: A Focus on the Anoperineal Location

Sitthipong Srisajjakul¹, Patcharin Prapaisilp¹, Sirikan Bangchokdee²

¹Department of Radiology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand; ²Department of Internal Medicine, Pratumthani Hospital, Bang Prok, Thailand

Hidradenitis suppurativa (HS) is a chronic inflammatory skin disease involving apocrine-bearing sites. It is characterized by recurrent painful nodules and abscesses that potentially rupture, resulting in sinus tract formation, fistulas, and scarring. HS tends to be found in the intertriginous areas (i.e., the axillary, inguinal, and perianal areas of the body). HS may be uncommon for radiologists because its diagnosis is usually based on clinical assessment. However, diagnosis based solely on clinical manifestations can underestimate the severity of HS. Ultrasonography and MRI play a critical adjunct role in determining the severity and extent of the disease and greatly aid its management. Given that MRI is an effective imaging tool, its role in the analysis of severe and anogenital HS lesions merits considerable attention. Unfortunately, anoperineal HS imposes diagnostic dilemmas. It has multiple symptoms and presentations and often mimics other diseases in the intertriginous areas. Therefore, a thorough understanding of HS is essential to avoid delayed diagnoses. This review highlights the typical MRI imaging features and staging of HS, emphasizing on the anoperineal location. The review also differentiates the disease from mimics to facilitate the prompt delivery of appropriate treatment and improve patients' quality of life.

Keywords: Hidradenitis suppurativa; MRI; Anoperineal disease; Perianal disease; Imaging

INTRODUCTION

Hidradenitis suppurativa (HS) is a complex disease difficult to treat. The exact origin of HS is unclear. It is considered a disease of follicular occlusion rather than an inflammatory or infectious process of the apocrine glands [1]. The plugging of hair follicles with keratin and subsequent expansion and rupture of the follicles lead to spillage of the keratin into the surrounding tissue. This spillage leads to an intense inflammatory response that manifests as painful recurrent nodules and abscesses and, subsequently, the formation of sinus tracts and scarring

Received: April 1, 2022 **Revised:** May 3, 2022 **Accepted:** May 24, 2022

[1,2]. Common locations are the intertriginous areas, such as the axillary region (Fig. 1), groin, and perineal and perianal regions. Several sites may be involved simultaneously, and are usually symmetrical. HS is more common in female; however, anoperineal HS affects more male than female [3]. The hallmark of HS is chronicity, with recurrences occurring in and around the original sites. Among all skin diseases, patients with HS have the highest morbidity and the lowest quality of life [4].

The clinical features of anoperineal HS can overlap with Crohn's disease (CD), which has been reported to coexist in 17% to 40% of patients [5]. Pelvic MRI has gained acceptance as the imaging modality of choice for assessing anoperineal diseases. This article aims to identify MRI findings that can be used as a roadmap to suggest HS when anoperineal diseases are encountered and help differentiate HS from mimics.

Clinical Criteria and Staging

HS typically develops after puberty, with peak onset in the early 20s. The prevalence is estimated at 1% of the

<sup>Corresponding author: Sitthipong Srisajjakul, MD, Department of Radiology, Faculty of Medicine Siriraj Hospital, Mahidol University, 2 Wanglang Road, Bangkoknoi, Bangkok 10700, Thailand.
E-mail: tiam.mahidol@gmail.com</sup>

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (https://creativecommons.org/licenses/by-nc/4.0) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

Korean Journal of Radiology

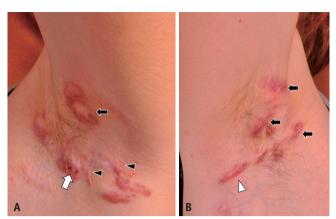


Fig. 1. Bilateral axillary HS.

A, **B**. A 29-year-old female patient with bilateral axillary HS. Multiple erythematous papules (black arrows) and paired pits from inflamed hair follicles (white arrow) with dermal contractures and rope-like elevation of skin (white arrowhead) and bridging scar (black arrowheads) are shown at both axillae. Bilateral involvement is common in this disease. HS = hidradenitis suppurativa

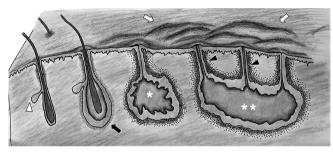


Fig. 2. Step of progression in hidradenitis suppurativa. Normal hair follicle with attached sebaceous gland (white arrowhead) is shown. Follicular occlusion causing widening of the hair follicle is observed (black arrow). Subsequently, there is a nodular or abscess formation (asterisk). Finally, there is interconnection of the abscesses (two asterisks) and then rupture. This can lead to the formation of a complex network of sinus tracts (black arrowheads). Skin thickening and induration (white arrows) develop during the course of the disease.

Table 1. Clinical Criteria for Hidradenitis Suppurativa

able	1. Clinical Criteria for Hiuradenicis Suppurativa
	Clinical Criteria
1	Typical lesions: painful deep-seated nodules, abscesses,
	bridging scars, draining fistulas, and pseudocomedones
2	Typical areas: armpits, inframammary region, intermammary
	folds, groin, perineum, or buttocks
3	Typical chronicity and recurrences: 2 recurrences of the
	lesions during a naried of C months

lesions during a period of 6 months

general European population, increasing to 4% in young adult female. There is a male predominance of anogenital disease, although overall, the condition is twice more common in female [6,7].

The substantial environmental risk factors for the development of HS are smoking, obesity, and a family history of HS. The clinical symptoms and signs of HS are initially characterized by painful subcutaneous nodules with pruritus and hyperhidrosis. The nodules rupture over approximately 7 days to 15 days, leading to deep dermal abscesses. After rupture, the lesions frequently extrude a foul-smelling, purulent discharge. Subsequently, they become draining sinus tracts, scars, or fibrosis (Fig. 2). Three criteria are used to establish a clinical diagnosis (Table 1).

Several classification systems have been proposed to stage HS. The Hurley clinical staging system is the most straightforward and widely implemented. It is also the most relevant system to the European S1 guidelines (Table 2) [8,9]. Most patients have stage I (68%) or stage II disease (28%), with few having stage III disease (4%) [10]. The drawback of the Hurley staging system is that it describes the finding in a particular region rather than the total disease burden for the patient [9].

In contrast, the modified Sartorius scoring system is a more dynamic and quantitative severity assessment tool. It considers the lesion count, number of anatomical regions involved, types of lesions, and distance between two relevant lesions. Nevertheless, it is rarely used in clinical practice as it is time-consuming and complex [11].

Imaging Role

The diagnosis of HS is usually made by clinical observation, and a biopsy is rarely needed. However, compared with diagnosis by imaging, relying solely on a physical examination and using only clinical criteria for diagnosis can lead to underestimations of the stage of the disease and its severity [12]. Visual observation and manual palpation appear to be spatially limited due to inflammation, potentially resulting in incorrect interpretations of fistulas as nodules. Such misdiagnoses

Table 2. Hurley Clinical Staging System for Hidradenitis Suppurativa

Stage	Nodules/Abscesses	Sinus Tracts and Scarring	Extension of Disease
I	Single or multiple	No	Isolated lesions
II	Single or multiple	Yes	Recurrent and widely separated lesions
III	Multiple	Yes	Diffuse involvement across regional area with interconnected lesions



lead to errors in stage assignment, severity assessment, and treatment planning. Histology is often of limited value when HS involvement is deep and widely multiregional [13]. In such instances, a radiological investigation may better determine the subclinical changes (e.g., dermal pseudocysts, widened hair follicles, and subdermal fluid collection), and the severity of the involvement. This information can guide surgical treatment [12,13].

Ultrasonography is most frequently employed as the

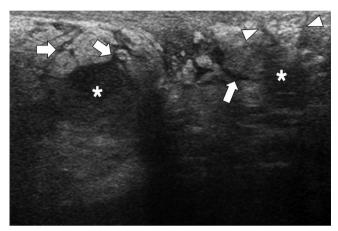


Fig. 3. Perineal hidradenitis suppurativa. A 45-year-old male patient with tender and itching at perineum. Ultrasonography of the anterior perineum shows a few hypoechoic fluid pockets (asterisks). Widened dermal hair follicles connecting to fluid collection are seen (arrowheads). Several areas of hypodermal sinus tracts emanating from fluid collections are noted (arrows).

first line of investigation in HS, and it is an excellent technique for studying skin layers (Fig. 3). Nevertheless, MRI is gaining in popularity. While MRI is mainly used for extensive HS and deep anoperineal lesions [14], some physicians employ it even for milder stages of the disease. It is vital to obtain high-resolution images to readily identify the sinus tracts and a dermal or subdermal abscess and assess its extent. The MRI sequences include T2weighted acquisitions, even with the short tau inversion recovery (STIR) technique, to emphasize the signal of fluid components. Post gadolinium and diffusion-weighted images may also help detect active disease and aid in diagnosing complex cases that previously underwent surgery. The MRI protocol and imaging parameters for anoperineal HS are detailed in Table 3.

MRI Findings According to Disease Stage

Hurley Stage 1

The disease at this initial stage is confined to the skin and subcutaneous layer. MRI findings include dermal thickening and subdermal induration, which show high signal intensity on T2-weighted images, restricted diffusion, and enhancement (Fig. 4). Solitary or multiple isolated abscess formation is evident (Fig. 5). However, no scarring or sinus tract is present.

Table 3. Pelvic MRI Protocol for Hidradenitis Suppurativa Evaluation at Our Institute

Imaging Sequence and Orientation	FOV (mm)	TR/TE (ms)	Thickness (mm)	Gap (mm)	NSA	Matrix Size	S/N Ratio
T2W SSFSE coronal	350 x 330	1200/89	5	1	1	358 x 356	1
T2W SSFSE axial	350 x 285	2000/114	6	1.2	1	293 x 400	1
T2W STIR axial	350 x 295	2860/78	6	1.2	1	227 x 384	1
T2W STIR sagittal	170 x 180	7040/95	3	0	2	286 x 336	1
T2W STIR axial	170 x 170	9550 x 88	3	0	2	235 x 336	1
T2W STIR coronal	170 x 180	5890 x 96	3	0	2	269 x 236	1
DWI axial b value 50, 800, 1000 s/mm²	170 x 170	9200 x 67	3	0	b50 = 1 b800 = 7 b1000 = 10	110 x 110	1
T1W 3D axial FS without GD	350 x 306	3/1.19	3	0	1	231 x 352	1
T1W 3D axial FS with GD	350 x 306	3/1.19	3	0	1	231 x 352	1
T1W 3D coronal FS with GD	350 x 330	4/1.23	3	0	1	204 x 256	1
T1W 3D sagittal FS with GD	270 x 330	4/1.23	3	0	1	156 x 256	1

All parameters presented are taken from a protocol designed for a 1.5T MR scanner. It is very important to cover the whole buttock and perineum because the fistulous pathways extend and open at the skin surface. Do not place pillows under the legs because doing so risks lifting the bottom of the pelvis away from the coil. A 3T MR scanner can be used with a high S/N ratio and spatial resolution with theoretically high conspicuity to identify minor fistulous pathways. Drawbacks include increased susceptibility artifacts, magnetic field inhomogeneity, and specific absorption rates. DWI = diffusion-weighted image, FOV = field of view, FS = fat suppressed, GD = gadolinium, NSA = number of signal coverage, SSFSE = single shot fast spin echo, STIR = short tau inversion recovery, S/N = signal to noise, T = Tesla, TE = echo time, TR = repetition time, T1W = T1-weighted, T2W = T2 weighted, 3D = three-dimensional



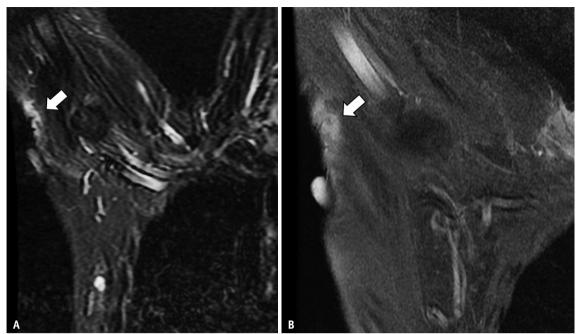


Fig. 4. Stage I hidradenitis suppurativa. A 35-year-old female patient with painful lumps at right axilla.
A. Coronal short tau inversion recovery image shows skin thickening or induration with evidence of subdermal high signal intensity (arrow).
B. Coronal T1-weighted post gadolinium image shows area of enhancement extending to dermal surface at right axilla (arrow). Body marker was seen in both images.

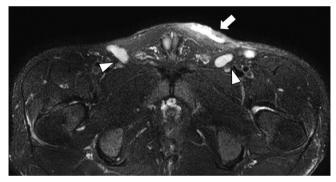


Fig. 5. Stage I hidradenitis suppurativa. A 51-year-old male patient with painful left groin. Axial short tau inversion recovery image shows skin thickening with subcutaneous abscess formation at left anterior perineum (arrow). Enlarged enhanced reactive bilateral inguinal nodes are identified (arrowheads).

Hurley Stage 2

If the disease does not heal with mild scarring, recurrent involvement can develop, with multiple, raised, subdermal pockets of rim-enhancing pus forming. These pockets eventually rupture to form sinus tracts. MRI findings show a combination of the stage I findings and widely separated, high signal intensity, sinus or fistulous tracts on STIR or post gadolinium images (Figs. 6, 7).

Hurley Stage 3

Stage 3 often includes refractory or intractable cases with

extensive involvement across a regional area. MRI shows multiple interconnecting sinus tracts and fistulas draining through the skin. Chronicity can be observed as thick sinus tract walls with low signal intensity during STIR imaging due to scar formation (Fig. 8). Occasionally, the disease extends beyond the dermis and subcutaneous fat to involve the deeper fascia and muscle, causing local deformities [15].

Enlarged inguinal nodes may be observed at all stages. Fistula communication with the bladder, urethra, and rectum is extremely rare. MRI has been used preoperatively to evaluate the borders of lesions before wide surgical excisions of HS and aid assess treatment responses. Griffin et al. [14] found that a reduction in the number of sinus tracts and their signal intensity on follow-up STIR MRI signified decreased disease activity, and loss of high T2 signal preceded a lack of enhancement on MRI.

The Link between HS and CD

Currently, the pathogenesis of HS and CD remains controversial. Studies have illustrated that HS and CD share similar risk factors, immune dysregulation mechanisms, genetic loci, and biochemical changes. It is well recognized that smoking increases the risk of CD and HS. Smoking in HS has pathological effects on the development of epidermal

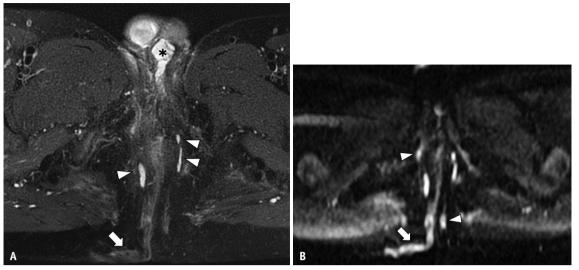


Fig. 6. Stage II hidradenitis suppurativa. A 35-year-old male patient with purulent discharge at perianal area. **A.** Axial short tau inversion recovery shows abscess pocket at left scrotum (asterisk) and multiple subdermal sinus tracts along right and left sided intergluteal cleft (arrowheads). Thickening of dermal plane and subcutaneous tissue of right gluteal region with increased signal intensity (arrow). **B.** DWI accentuates depiction of more subdermal sinus tracts (arrowheads) than axial short tau inversion recovery in **(A)**. Skin and subcutaneous induration at right gluteal region is more conspicuous on DWI (arrow). DWI = diffusion-weighted image

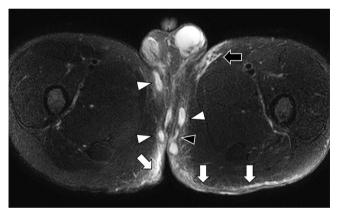


Fig. 7. Stage II HS. A 31-year-old male patient with grade II HS. Axial short tau inversion recovery shows multiple abscesses along right and left side of intergluteal cleft (white arrowheads) and sinus tract between abscesses at left side of intergluteal cleft (black arrowhead). Another branching sinus tract is in the left groin region (black arrow) with skin thickening and subcutaneous edema in the bilateral gluteal regions (white arrows). HS = hidradenitis suppurativa

hyperplasia and follicular plugging [16]. Some studies found that CD and HS had identical immune dysregulation mechanisms, such as significant increases in interleukin-1 (IL-1), IL-6, IL-17, IL-23, and tumor necrosis factor [16,17]. Both CD and HS histopathologically feature lymphatic follicles and granulomas.

Differential Diagnosis

CD

Making a differential diagnosis between anoperineal HS

and CD is challenging as they can present with the same tissue changes. In addition, it is common for both disorders to coexist (Fig. 9), with signs of one overlapping with those of the other [5,18]. T2-weighted acquisitions and the STIR technique are the key imaging sequences used to identify abscesses and fistulous pathways in both diseases. The site, distribution, and characteristics of the lesions and associated findings are clues facilitating a differential diagnosis on MRI.

In HS, the lesions often affect the anterior inguinal area, intergluteal fold, and the posterior sacral or gluteal region. Most fistulas do not reach the dentate line of the anal canal due to the absence of apocrine glands at and above that point. It is common to observe diffuse subcutaneous edema and skin thickening with bilateral involvement and less sphincter involvement. Involvement of the axillary region or another HS-specific location is another valuable clue [19,20].

In CD, lesions are more prevalent in the perianal location. Fistulous tracts or abscesses usually involve the anal sphincter complex, extending to the ischiorectal fossa and supralevator space [19,20]. Coexisting rectal wall thickening and concomitant gastrointestinal symptoms are more commonly observed with CD.

In addition, inflammatory granulomas or abscesses are more frequently found in HS and tend to be small and coalescent. In contrast, fistulous tracts seem to be more frequent in patients with CD than those with HS.



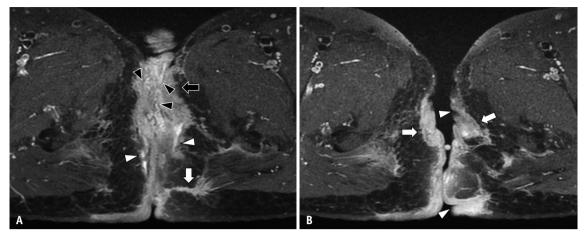


Fig. 8. Stage III hidradenitis suppurativa. A 37-year-old male patient with tender and foul discharge at perianal region. **A.** Axial short tau inversion recovery shows multiple subdermal sinus tracts along either side of the intergluteal cleft (white arrowheads). There is some intercommunication (black arrowheads) and marked subcutaneous thickening (black arrow) and a deep scar (white arrow). **B.** Axial short tau inversion recovery lower than **(A)** shows marked skin thickening (arrows) with scars and retracted skin surface at upper inner thighs (arrowheads). Significant scarring is very common in severe disease.

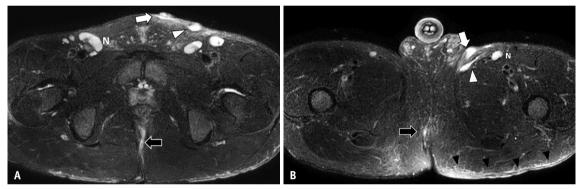


Fig. 9. Crohn's disease and HS.

A. A 36-year-old male patient with Crohn's disease and HS. Axial short tau inversion recovery shows subcutaneous abscess (arrowhead), subcutaneous induration with edema (white arrow), and enlarged inguinal nodes (N) consistent with HS. There is evidence of a perianal fistula in Crohn's disease (black arrow). **B.** A 31-year-old male patient with Crohn's disease and HS. Axial short tau inversion recovery shows subcutaneous abscess (white arrow) and sinus tract at left groin (white arrowhead), subcutaneous and skin thickening at left gluteal region (black arrowheads) and enlarged inguinal nodes consistent with HS (N) and associated perianal Crohn's disease (black arrow). HS = hidradenitis suppurativa

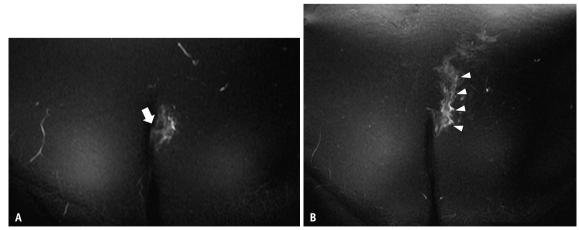


Fig. 10. Pilonidal abscess.

A, **B**. A 37-year-old male patient with cutaneous discharge at mid upper buttock. Coronal short tau inversion recovery shows small focal high signal intensity at higher level of the intergluteal cleft (arrow) with elongated tract (arrowheads) in the left buttock and surrounding inflammatory change. There is no communication with the anal sphincteric area.



Pilonidal Abscess

Classically, pilonidal sinuses and abscesses arise at the natal cleft (the primary opening) and chiefly affect young male. Their clinical presentations vary. They may present as a simple intergluteal skin pit with minimal symptoms. Alternatively, they may manifest as a complex infection arising in the subcutaneous tissue of the sacrococcygeal area, with multiple sinuses and secondary openings located off the midline (Fig. 10). In cases where the pilonidal sinus runs caudally, the secondary opening may resemble a perianal fistula. HS is frequently mistaken as recurrent pilonidal sinus and is strongly associated with and exacerbated by smoking and obesity. However, recurrent painful suppurative abscesses, subcutaneous nodules, sinus

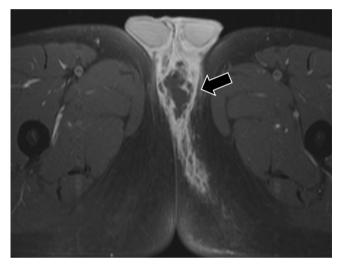


Fig. 11. Perineal actinomycosis. A 32-year-old male patient with a history of penetrating trauma by wooden stick at left buttock. Axial T1-weighted post gadolinium image shows tract of penetrating trauma at medial aspect of left buttock. There is scrotal wall thickening with a rim enhancing abscess at the perineum (arrow). Fluid analysis of this abscess found actinomycosis.

tracts, or fistulas with chronic scarring and opened pits serve as diagnostic clues [8].

Actinomycosis

Pelvic actinomycosis may occur because of penetrating trauma, abdominal surgery, or long-standing use of intrauterine devices. It usually presents abscess formation, dense fibrosis, and draining sinuses (Fig. 11). The condition can spread across tissue planes. Its infiltrative nature might stem from the action of proteolytic enzymes released by the bacterium *Actinomycosis israelii*. Pelvic actinomycosis is frequently confused with malignant diseases [21].

Carbuncles, Cellulitis, and Erysipelas

HS may not be differentiated from carbuncles, cellulitis, and erysipelas if there is no sinus tract or fistula formation. The typical locations and chronicity of HS are clues for differentiation.

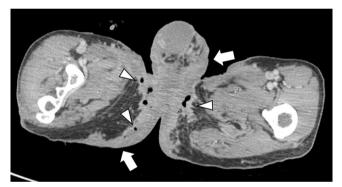


Fig. 12. Fournier's gangrene. A 49-year-old male patient presented at the emergency unit with fever and tenderness of the genitalia. Axial enhanced CT shows soft-tissue edema and fascial thickening at scrotum and medial aspect of both upper thighs (arrows) and multiple soft-tissue gas (arrowheads). CT is far better than MRI for detecting air bubbles.

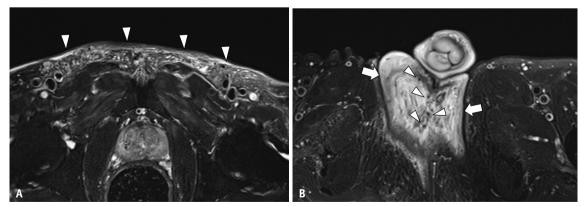


Fig. 13. Fournier's gangrene. A 52-year-old male patient with sepsis and multiple organ failure with crepitus at scrotal region. **A.** Axial fat-suppressed T2-weighted image shows skin thickening and subcutaneous edema at anterior perineal region (arrowheads). **B.** Axial fat-suppressed T2-weighted image shows marked scrotal wall thickening (arrows) and multiple tiny air bubbles at right scrotal sac (arrowheads).



Fournier's Gangrene

Fournier's gangrene is a urological emergency. It is defined as rapid and progressive infective necrotizing fasciitis of the perineal or anoperineal region caused by polymicrobial infection. The condition typically affects middle-aged male, and the most frequently associated factors are diabetes and chronic alcohol consumption [22]. Soft-tissue inflammation and edema cause a suppurative infection and obliterative endarteritis of the dermal and hypodermal arteries leading to necrosis. Crepitus can be discovered through physical examination due to soft-tissue gas. The rate of fascial necrosis has been reported to be as high as 2 to 3 cm per hours, and patients can succumb to severe sepsis. Early diagnosis is therefore essential for patient survival [23].

CT is beneficial and outperforms plain radiography and ultrasonography. Computed tomography delineates fascial and skin thickening, fat stranding, and gas extension in soft tissue, and it can occasionally identify the starting point of Fournier's gangrene (Fig. 12). MRI can readily detect soft-tissue edema and fascial thickening in fluid-sensitive sequences. However, it may sometimes prove challenging to differentiate soft-tissue gas from the signal void of vascular flow or phlebolith (Fig. 13) [24].

Complications

Complications may be local, systemic, physical, or (in cases of long-term uncontrolled disease) psychological. Infection may follow, in turn causing septicemia. Fibrosis and scarring result in contractures and impaired joint mobility. Arthropathy associated with HS manifests with variable clinical findings, and its activity is related to the disease activity of the HS [25]. Rarely, squamous cell carcinoma may develop on top of the scars or chronic inflammation in long-standing HS cases [26].

Management

Topical and systemic antibiotics, retinoids, hormones, steroids, and anti-tumor necrosis factor therapy coupled with local cleansing and warm compression are usually used for every clinical stage (Hurley stages I–III) [27]. If the disease is recurrent or advanced (Hurley stages II–III), incisional drainage or radical excision of all apocrine glands in the affected areas may be needed [28]. Surgery is often required to treat the tunnels and scars associated with chronic HS [26,28,29].

CONCLUSIONS

Although HS is mainly clinically diagnosed, imaging tools, especially MRI, are beneficial and improve staging and characterization of lesions. The imaging tools can also guide management and reduce recurrent disease.

Availability of Data and Material

The datasets generated or analyzed during the study are available from the corresponding author on reasonable request.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

Author Contributions

Conceptualization: all authors. Data curation: all authors. Investigation: all authors. Resources: all authors. Validation: all authors. Visualization: all authors. Writing—original draft: all authors. Writing—review & editing: all authors.

ORCID iDs

Sitthipong Srisajjakul https://orcid.org/0000-0002-2598-3037 Patcharin Prapaisilp https://orcid.org/0000-0001-9516-067X Sirikan Bangchokdee https://orcid.org/0000-0002-6031-447X

Funding Statement

None

REFERENCES

- Alikhan A, Lynch PJ, Eisen DB. Hidradenitis suppurativa: a comprehensive review. J Am Acad Dermatol 2009;60:539-561; quiz 562-563
- Kurzen H, Kurokawa I, Jemec GB, Emtestam L, Sellheyer K, Giamarellos-Bourboulis EJ, et al. What causes hidradenitis suppurativa? *Exp Dermatol* 2008;17:455-456; discussion 457-472
- 3. Kimball AB, Kerdel F, Adams D, Mrowietz U, Gelfand JM, Gniadecki R, et al. Adalimumab for the treatment of moderate to severe hidradenitis suppurativa: a parallel randomized trial. *Ann Intern Med* 2012;157:846-855



- 4. von der Werth JM, Jemec GB. Morbidity in patients with hidradenitis suppurativa. *Br J Dermatol* 2001;144:809-813
- van der Zee HH, van der Woude CJ, Florencia EF, Prens EP. Hidradenitis suppurativa and inflammatory bowel disease: are they associated? Results of a pilot study. *Br J Dermatol* 2010;162:195-197
- Revuz JE, Canoui-Poitrine F, Wolkenstein P, Viallette C, Gabison G, Pouget F, et al. Prevalence and factors associated with hidradenitis suppurativa: results from two case-control studies. J Am Acad Dermatol 2008;59:596-601
- Jemec GB, Heidenheim M, Nielsen NH. The prevalence of hidradenitis suppurativa and its potential precursor lesions. J Am Acad Dermatol 1996;35(2 Pt 1):191-194
- 8. Zouboulis CC, Desai N, Emtestam L, Hunger RE, Ioannides D, Juhász I, et al. European S1 guideline for the treatment of hidradenitis suppurativa/acne inversa. *J Eur Acad Dermatol Venereol* 2015;29:619-644
- 9. Hurley H. *Dermatologic surgery, principles and practice*. New York: Marcel Dekker, 1989:729-739
- 10. Canoui-Poitrine F, Revuz JE, Wolkenstein P, Viallette C, Gabison G, Pouget F, et al. Clinical characteristics of a series of 302 French patients with hidradenitis suppurativa, with an analysis of factors associated with disease severity. *J Am Acad Dermatol* 2009;61:51-57
- Sartorius K, Emtestam L, Jemec GB, Lapins J. Objective scoring of hidradenitis suppurativa reflecting the role of tobacco smoking and obesity. *Br J Dermatol* 2009;161:831-839
- Wortsman X, Moreno C, Soto R, Arellano J, Pezo C, Wortsman J. Ultrasound in-depth characterization and staging of hidradenitis suppurativa. *Dermatol Surg* 2013;39:1835-1842
- 13. Wortsman X. Imaging of hidradenitis suppurativa. *Dermatol Clin* 2016;34:59-68
- 14. Griffin N, Williams AB, Anderson S, Irving PM, Sanderson J, Desai N, et al. Hidradenitis suppurativa: MRI features in anogenital disease. *Dis Colon Rectum* 2014;57:762-771
- Aduful H, Paintsil A. Extensive groin and perineal hidradenitis suppurativa complicated by high fistula in ano. *Ghana Med J* 2007;41:30-32
- Hana A, Booken D, Henrich C, Gratchev A, Maas-Szabowski N, Goerdt S, et al. Functional significance of non-neuronal acetylcholine in skin epithelia. *Life Sci* 2007;80:2214-2220

- 17. Schlapbach C, Hänni T, Yawalkar N, Hunger RE. Expression of the IL-23/Th17 pathway in lesions of hidradenitis suppurativa. *J Am Acad Dermatol* 2011;65:790-798
- Yazdanyar S, Miller IM, Jemec GB. Hidradenitis suppurativa and Crohn's disease: two cases that support an association. *Acta Dermatovenerol Alp Pannonica Adriat* 2010;19:23-25
- Haggett PJ, Moore NR, Shearman JD, Travis SP, Jewell DP, Mortensen NJ. Pelvic and perineal complications of Crohn's disease: assessment using magnetic resonance imaging. *Gut* 1995;36:407-410
- Monnier L, Dohan A, Amara N, Zagdanski AM, Drame M, Soyer P, et al. Anoperineal disease in hidradenitis suppurativa : MR imaging distinction from perianal Crohn's disease. *Eur Radiol* 2017;27:4100-4109
- 21. Heo SH, Shin SS, Kim JW, Lim HS, Seon HJ, Jung SI, et al. Imaging of actinomycosis in various organs: a comprehensive review. *Radiographics* 2014;34:19-33
- Safioleas M, Stamatakos M, Mouzopoulos G, Diab A, Kontzoglou K, Papachristodoulou A. Fournier's gangrene: exists and it is still lethal. *Int Urol Nephrol* 2006;38:653-657
- 23. Levenson RB, Singh AK, Novelline RA. Fournier gangrene: role of imaging. *Radiographics* 2008;28:519-528
- 24. Lin MP, Chen YL, Cheng JT, Lu CL. Early signs of Fournier gangrene: radiological characteristics. *Radiol Diagnos Imaging* 2018;2:1-6
- 25. Hamoir XL, François RJ, Van den Haute V, Van Campenhoudt M. Arthritis and hidradenitis suppurativa diagnosed in a 48-yearold man. *Skeletal Radiol* 1999;28:453-456
- 26. Slade DE, Powell BW, Mortimer PS. Hidradenitis suppurativa: pathogenesis and management. *Br J Plast Surg* 2003;56:451-461
- Boer J, van Gemert MJ. Long-term results of isotretinoin in the treatment of 68 patients with hidradenitis suppurativa. J Am Acad Dermatol 1999;40:73-76
- 28. van der Zee HH, Prens EP, Boer J. Deroofing: a tissue-saving surgical technique for the treatment of mild to moderate hidradenitis suppurativa lesions. J Am Acad Dermatol 2010;63:475-480
- 29. Kelly AM, Cronin P. MRI features of hidradenitis suppurativa and review of the literature. *AJR Am J Roentgenol* 2005;185:1201-1204