

# ASSESSMENT OF SUBCLINICAL ATHEROSCLEROSIS IN PATIENTS WITH HIDRADENITIS SUPPURATIVA AND ITS RELATION TO C-REACTIVE PROTEIN

By

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## ABSTRACT

**Background:** Hidradenitis suppurativa (HS) is a chronic, inflammatory, recurring dermatosis of the hair follicle. Some studies have revealed relation between HS and other diseases as metabolic syndrome, multifaceted disorder associated with increased risk for subclinical atherosclerosis and cardiovascular disease (CVD).

**Objective:** To measure carotid artery intima-media thickness (CIMT) as predictors of cardiovascular risk in HS patients and its relation to inflammatory serum marker especially C-reactive protein (CRP).

**Patients and Method:** This was a prospective case-control performed on 20 patients with HS and compared to 20 healthy volunteers matched in sex, mean of the age and BMI. Participants were recruited from Outpatient clinic of Al-Zahra'a University Hospital during the period from March 2017 to December 2018. The following investigations were done to all the participants including CRP, ESR, neutrophils count, HDL, total cholesterol, triglycerides and glucose. Carotid intima media thickness with B-mode ultrasound.

**Conclusion:** CIMT measurement was not significantly different between patients and controls but differed significantly according to stage of the disease, so HS potentially increased the risk of cardiovascular. The thickness of CIMT related directly to inflammatory serum markers especially CRP, so CRP was considered marker of disease severity and can be used for monitoring the response of treatment.

**Key words:** Hidradenitis suppurativa, subclinical atherosclerosis, cardiovascular disease, carotid intima-media thickness, carotid ultrasonography.

## INTRODUCTION

Hidradenitis suppurativa (HS) is a recurrent chronic inflammatory debilitating skin disease of the hair follicle. It presents with inflamed painful, deep-seated lesions in the apocrine - bearing areas, usually affect the inguinal, anogenital regions and axillae (Nguyen and Damiani, 2021). The prevalence rate

of HS in the general population from 0.05 to 4% (Zouboulis *et al.*, 2015).

Chronic inflammatory diseases such as rheumatoid arthritis, psoriasis and ankylosing spondylitis have been associated with sub clinical atherosclerosis (Gonzalez-Lopez *et al.*, 2016, Arida *et al.*, 2018 and Peluso *et al.*, 2019).

HS is associated with several comorbidities, including subclinical atherosclerosis, cardiovascular disease, metabolic syndrome, diabetes, osteoarthritis or inflammatory bowel disease (*Kohorst et al., 2015* and *Montero-Vilchez et al., 2021*).

Some reported systematic reviews indicates that CVD risk factors appear at significantly higher rate in patients with HS compared with control subjects (*Tzellos et al., 2015*).

There are many non-invasive tools that have been found useful to detect the presence of subclinical atherosclerosis in patients with chronic inflammatory diseases (*Li et al., 2021*). The measurement of CIMT by carotid ultrasound is one of these tools (*Kerekes et al., 2012*).

The present study was done for detection of cardiovascular risk in patients with HS and the relation between them and inflammatory serum markers through evaluation of CIMT by carotid ultrasound and lab measurement of CRP, ESR and neutrophil level.

**The aim of the present work was to** assess the prevalence of subclinical atherosclerosis in HS patients and the relation of C reactive protein to HS severity and incidence of sub-clinical atherosclerosis.

## PATIENTS AND METHODS

This prospective case-control study has been conducted on 20 patients with hidradenitis suppurativa as a patient's group and 20 age and sex-matched healthy volunteers as a control group. Participants were recruited from Outpatient clinic of

Al-Zahra'a University Hospital during the period from March 2017 to December 2018.

The included cases have been clarified about the aim and the procedure of this study, and informed consent approved by the research ethics committee of the Faculty of Medicine, Al-Azhar University Hospitals, was obtained from every participant prior to study initiation.

**Exclusion criteria:** Patients' history of CVD, including ischemic heart disease, heart failure, cerebrovascular accidents, or peripheral arterial disease. Diabetes mellitus patients, obesity and dyslipidemia and chronic kidney disease. Patients with chronic kidney disease, presence of concomitant inflammatory diseases (rheumatoid arthritis and inflammatory bowel disease) or other chronic skin diseases.

### All cases were subjected to:

Complete history taking: Age of onset, duration of the disease, family history of HS and risk factors. History of chronic heart disease, diabetes mellitus or chronic kidney diseases or any other chronic disease

Weight, height, and abdominal circumference of participants have been measured, and their body mass index (BMI) have been calculated according to the following formula (weight in kg/ height in m<sup>2</sup>) (*MacKay 2010*).

**Dermatological examination:** To determine the presence of other skin diseases and to score the degree of severity of the disease was according to; Hurley staging system (*Zouboulis et al., 2015*).

**Blood sampling:** for assessment of Neutrophils count, C-Reactive Protein (CRP) level, erythrocyte sedimentation rate (ESR), FBG, high density lipoprotein, triglycerides, and serum creatinine.

**Radiological investigations:** Measurement of carotid artery intima-media thickness (CIMT) by Doppler. All patients were underwent to Dupler examination at Radiology Department of Al-Zahraa University Hospital using Esaote My Lab™50 machine, (made in Italy) a high-frequency linear array probe 7 MHz is used.

The patient was examined in a supine position and the head extended and inclined by about 30 with a pillow under it. The intima-media complex (IMC) composed of two layers, the hyperechoic layer closer to the vascular lumen and the hypoechoic layer. Depiction of IMC was taken in B-mode gray scale along the posterior wall of CCA and the minimum unit of IMT measurement was 0.1 mm.

Mean IMT measurement of mean IMT was performed on the right and left

common carotid artery. The cut points for intima-media thickness between normal and abnormal populations have varied among reported studies and, therefore, it was difficult to establish a single cut point that defines abnormality. CIMT of 0.9 mm or more was abnormal and likely to be associated with sonographically plaque.

#### **Statistical design:**

Quantitative data were expressed in term of mean  $\pm$ (SD) and median, independent T-test and ANOVA were used to study the difference in parametric continuous variables while, Mann Whitney and Kruskal Wallis tests were used for non-parametric data. Qualitative data were expressed in number and percentage. Chi square-test, Fisher's Exact or Monte Carlo corrections were used for comparison between groups. Pearson correlation test was done to assess relation between CIMT with duration and ESR in patients' group. Significance level was taken at a P-value  $\leq$ 0.05. SPSS, version 20 was used for data analysis.

## **RESULTS**

Descriptive data of the studied groups in our study showed that the age of patients and control ranged from 20 to 55 years (mean  $\pm$  SD: 38.45 $\pm$ 10.78years) with no statistically significant difference. There was no statically significant difference regard, sex and BMI both patient and control group.

As regard smoking 7 out of 20 patients (35%) were smokers and 13 (65%) were nonsmokers. while the controls were nonsmokers (P=0.008).

In patient group 5 patients (25%) had positive family history of HS, while the

control group showed negative family history for HS (P =0.047).

Regarding serum creatinine, serum lipid profile and fasting blood glucose there was no statically significant difference between patient and control groups.

There was no statically significance difference between patients and control regarding TG, HDL, LDH and Total cholesterol level (**Table 1**).

It was noticed that neutrophils showed statically significant difference between patient and control groups (**Table 1**).

**Table (1): Comparison between the two studied groups according to laboratory investigations**

Parameters	Cases (n = 20)		Control (n =20)		P
	No.	%	No.	%	
<b>Serum Creatinine (mg/dl)</b>					
Less than 1.3	20	100.0	20		
More than 1.3	0	0.0	0	-	-
Min. -Max.	0.60 - 1.20		0.50 - 1.20		0.886
Mean $\pm$ SD.	0.89 $\pm$ 0.18		0.90 $\pm$ 0.25		
Median	0.85		1.0		
<b>Triglycerides (mg/dl)</b>					
Normal	20	100.0	20		
Abnormal	0	0.0	0	-	-
Min. -Max.	89.0 - 130.0		95.0 - 138.0		0.138
Mean $\pm$ SD.	110.10 $\pm$ 11.19		115.80 $\pm$ 12.58		
Median	111.0		116.50		
Mean rank	23.9		25.1		
<b>HDL</b>					
Min. - Max.	45.0 - 90.0		48.0 - 80.0		0.830
Mean $\pm$ SD.	64.80 $\pm$ 13.07		64.0 $\pm$ 10.11		
Median	63.50		61.0		
<b>LDL</b>					
Min. - Max.	55.0 - 95.0		55.0 - 91.0		0.885
Mean $\pm$ SD.	73.60 $\pm$ 12.47		74.15 $\pm$ 11.46		
Median	70.0		71.50		
<b>Total Cholesterol (mg/dl)</b>					
Normal	20	100.0	20	-	-
Abnormal	0	0.0	0		
Min. -Max.	100.0 - 170.0		103.0 - 170.0		0.994
Mean $\pm$ SD.	138.40 $\pm$ 20.69		138.35 $\pm$ 17.88		
Median	140.0		140.50		
<b>FBG (mg/dl)</b>					
Normal	16	80.0	20		0.106
Abnormal	4	20.0	0	0.106	
Min. -Max.	70.0 - 110.0		77.0 - 93.0		0.140
Mean $\pm$ SD.	91.20 $\pm$ 12.50		86.55 $\pm$ 5.50		
Median	90.0		88.0		
<b>Neutrophils</b>					
Not	14	70.0	20		0.020
Elevated	6	30.0	0	0.020*	
Min. -Max.	0.55 - 0.85		0.55 - 0.67		0.014
Mean $\pm$ SD.	0.67 $\pm$ 0.10		0.60 $\pm$ 0.04		
Median	0.62		0.60		

\*: Statistically significant

**Measurement of CIMT (considered thick if  $\geq 1$  mm):**

In patient group: 3 patients with CIMT  $\geq 1$  mm (15%) and 17 (85%) patients with CIMT  $< 1$ mm. Measurement of CIMT ranged between 0.6 to 1.2 mm (Mean  $\pm$  SD: 0.78  $\pm$  0.17).

In control group: all patients measurement was  $< 1$ mm. The measurement ranged between 0.4 to 0.8 mm with (Mean  $\pm$  SD: 0.68  $\pm$  0.09).

There was no statistically significant difference between patient and control regarding CIMT (p=0.231) (Table 2).

**Concerning C reactive protein:**

In the patient group: Six patient (30%) showed elevated CRP (Mean  $\pm$  SD: 7.33  $\pm$  1.63).

In control group: All of them showed normal CRP. with a statistically clinical significance p=0.020 (Table 2).

There was a statistically significant difference between patient and control regarding elevated CRP (p=.020) (Table 2).

**Erythrocyte sedimentation rate (ESR):**

In patient group: ESR ranged from 4 to 55 (Mean  $\pm$  SD: 24.10  $\pm$  17.53).

In controls: It ranged from 3 to 9 (Mean  $\pm$ SD: 6.15  $\pm$  1.87).

There was a statistically significant difference between patient and control regarding ESR level (p<0.001) (Table 2).

**Table (2): Comparison between the two studied groups according to CIMT, CRP and ESR**

Parameters	Cases (n = 20)		Control (n = 20)		P
	No.	%	No.	%	
<b>Carotid artery intima median thickness CIMT (mm)</b>					
Absent ( $< 0.8$ )	14	70.0	18	90.0	0.235
Present ( $\geq 0.8$ )	6	30.0	2	10.0	
Absent ( $< 1$ )	17	85.0	20	100.0	0.231
Present ( $> 1$ )	3	15.0	0	0.0	
Min. - Max.	0.60 - 1.20		0.40 - 0.80		0.025*
Mean $\pm$ SD.	0.78 $\pm$ 0.17		0.68 $\pm$ 0.09		
Median	0.70		0.70		
<b>CRP</b>					
Normal	14	70.0	20	100.0	0.020*
Elevated	6	30.0	0	0.0	
Min. - Max.	5.0 - 9.0		---		-
Mean $\pm$ SD	7.33 $\pm$ 1.63				
Median	7.50				
<b>ESR</b>					
Min. - Max.	4.0 - 55.0		3.0 - 9.0		P=0.000*
Mean $\pm$ SD	24.10 $\pm$ 17.53		6.15 $\pm$ 1.87		
Median	20.0		6.0		
Mean rank	27.8		13.2		

\*: Statistically significant

There was a statistically significant relation between Hulry staging and age, duration, smoking, family history, and

BMI in the patient group, while there was no statistically significant relation between Hulry stage and sex (**Table 3**).

**Table (3): Relation between Hulry stage and different parameters in patients' group (n= 20)**

Hulry staging Parameters	I (n = 7)		II (n = 7)		III (n = 6)		
	No.	%	No.	%	No.	%	
<b>Sex</b>							
Male	5	71.4	5	71.4	1.000	83.3	1.000
Female	2	28.6	2	28.6		16.7	
<b>Age (years)</b>							
Min. - Max.	20.0 - 43.0		25.0 - 51.0		42.0 - 55.0		P=0.001
Mean $\pm$ SD.	29.0 $\pm$ 8.35		39.14 $\pm$ 8.03		48.67 $\pm$ 5.43		
Median	27.0		39.0		50.50		
<b>Duration (years)</b>							
Min. - Max.	2.0 - 7.0		6.0 - 11.0		13.0 - 18.0		<0.001
Mean $\pm$ SD.	4.86 $\pm$ 1.95		8.29 $\pm$ 1.60		16.0 $\pm$ 2.0		
Median	5.0		8.0		16.0		
<b>Smoking</b>							
Negative	7	100.0	5	71.4	0.005*	16.7	0.005
Positive	0	0.0	2	28.6		83.3	
<b>Family history</b>							
Negative	7	100.0	7	100.0	<0.001*	16.7	<0.001
Positive	0	0.0	0	0.0		83.3	
<b>BMI</b>							
Min. - Max.	19.0 - 25.0		21.0 - 27.0		23.0 - 30.0		0.030
Mean $\pm$ SD.	21.57 $\pm$ 2.23		23.86 $\pm$ 2.54		25.83 $\pm$ 3.06		
Median	21.0		24.0		25.0		

By post hoc test: regarding age: Stage I is statistically significant with stage III (sig=0.001) but not stage II (sig=0.1). Stage II is not statistically

There was a statistically significant relation between CIMT and Hulry stage, duration of HS, family history, smoking, CRP, neutrophils and ESR (**Table 4**).

**Table (4): Relation between CIMT and different parameters in patients' group (n= 20)**

Parameters \ CIMT	Absent (<1) (n = 17)		Present (>=1) (n = 3)		
	No.	%	No.	%	
<b>Hulry stage</b>					
I	7	41.2	0	0.0	0.015
II	3	17.6	3	100.0	
III					
<b>CRP</b>					
Negative	14	82.4	0	0.0	0.018
Positive	3	17.6	3	100.0	
<b>Neutrophils</b>					
Not Elevated	14	82.4	0	0.0	0.018
Elevated	3	17.6	3	100.0	
<b>Smoking</b>					
Negative	13	76.5	0	0.0	0.031
Positive	4	23.5	3	100.0	
<b>Family history</b>					
Negative	15	88.2	0	0.0	0.009
Positive	2	11.8	3	100.0	
<b>Duration (years)</b>					
Min. - Max.	2.0 - 18.0		15.0 - 18.0		0.017
Mean ± SD.	8.12 ± 4.20		16.67 ± 1.53		
Median	7.0		17.0		
<b>ESR</b>					
Min. - Max.	4.0 - 53.0		45.0 - 55.0		0.013
Mean ± SD.	19.53 ± 14.62		50.0 ± 5.0		
Median	18.0		50.0		

There was a positive correlation between CIMT with both duration of HS, and the increase of ESR level (**Table 5**).

**Table (5): Correlation between CIMT with duration and ESR in patients' group (n= 20)**

Parameters \ CIMT	R	P
<b>Duration (years)</b>	0.719	<0.001
<b>ESR</b>	0.750	<0.001

r: Pearson coefficient

## DISCUSSION

There was no statically significant difference between patient and control group regarding age, sex and BMI as both groups were cross matched.

Positive family history in patients with HS was significantly higher in patients than control; it suggested that genetic factors play a role in causing HS. Different mutations located at the  $\gamma$ -secretase gene have been identified in six Han-Chinese HS families, with all afflicted members of the families carrying the mutation in agreement with studies done by (Wang *et al.*, 2010, Jemec *et al.*, 2012 and Pink *et al.*, 2013).

There was no clinical difference between patient and control as regard serum lipid profile in contrast to study done by *Gonzalez Lopez et al. (2016)* who found a relation between them. This explained by that in our study we excluded any member with dyslipidemia to detect the relation between CIMT and inflammatory serum markers with HS as dyslipidemia, if present, may be the cause of subclinical atherosclerosis and not the inflammatory state of the disease.

Regarding serum creatinine, serum lipid profile and fasting blood glucose, there was no statistically significant difference between patient and control groups as any member with abnormal measurement was excluded.

This study showed no significant difference between patient and control group as regard CIMT measurement in contrast to study done by *Gonzalez Lopez et al. (2016)* in which patients had greater carotid intima-media thickness values than control subjects. Despite this, the CIMT

differed significantly according to Hurly staging, the thickness increased with the increase of the stage of HS indicating that HS may considered a risk factor for development of atherosclerosis in the future.

Our study CRP and ESR significantly higher in patient with HS. This agreed with *Jiménez-Gallo et al. (2017)* study. *Yayan (2012)* observed increased CRP in one third of patients with acute coronary heart disease which indicate that CRP could be a strong predictor of increased risk cardiovascular in healthy individuals.

CRP and ESR considered early biomarkers that indicate the onset of systemic inflammation in HS (*Jiménez-Gallo et al., 2017*).

This study showed that there is relation between CIMT and CRP this agreed with study done by *Tkhashi et al. (2014)* that detected increase serum CRP level in patients with psoriasis (HS and psoriasis both are auto inflammatory chronic skin diseases).

In our study neutrophilic counts significantly higher in patient with HS. This increased neutrophilic counts and activation could consider HS a neutrophilic disorder (*Jiménez-Gallo et al., 2017*).

CIMT thickness in HS related significantly with elevated level of neutrophils. *Baetta et al. (2010)* postulate that neutrophils have causative role in atherogenesis and its progression.

CIMT correlated significantly with the duration of the disease increasing duration associated with increase of systemic inflammatory load. *Gonzalez et al. (2016)*

suggested that ESR and CRP play role in both HS and CVD.

CIMT significant relation with the incidence of smoking and hidradenitis suppurativa. This is explained by that smoking is considered not only a risk factor for CVD but also for HS. This agreed with *Micheletti et al. (2014)* who stated that many factors associated with HS, which includes smoking.

There was a significant relation between CIMT and family history of hidradenitis suppurativa which can be explained by genetics role in pathogenesis of both (*Gonzalez Lopez et al., 2016*).

In this study there was a relation between Hulry staging with age of the patient and duration of the HS which could be attributed to increase disease activity and inflammatory load with the increase of duration of the disease.

No change in the disease severity was detected according to sex of the patients which mean that sex hormones do not play role in disease severity in contrast to study done by *Vazquez et al. (2013)*, *Jørgensen et al. (2020)* and *Kirby et al. (2021)* this difference between both studies can be explained by limited number of our study participants.

In this study there is significant relation between disease severity and smoking in agreement with study done by *Vazquez et al. (2013)* and *Kohorst et al. (2015)*. Smoking is considered an important external risk factor for HS. Nicotine causes hyperplasia of infundibular keratinocytes and activation of a many of immune cells. The immunological response led to production of pro-inflammatory cytokines includes

interleukin IL-1, IL-8, tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) and stimulation of the Th17 cell (*Prens et al., 2015*).

The groups in our study according to inflammatory serum markers (CRP, ESR and neutrophils) showed significant difference which denoted that inflammatory serum makers especially CRP increased in patient with HS disease. Interestingly CRP, ESR and neutrophils increased significantly with the increase of the severity of hidradenitis suppurativa in agreement with study done by *Hesham et al. (2015)* with significant positive correlations among CRP and neutrophil count with HS Score.

## CONCLUSION

CIMT measurement was not significantly different between patients and controls but differed significantly with the severity of the disease. Inflammatory serum markers (CRP, ESR and neutrophils) were significantly different according to the stage of the disease. So, these serum markers can be used as indicator for the severity of the disease, and for follow up of the response to treatment.

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## معدل تصلب الشرايين تحت اكلينيكي بين المرضى بالتهاب الغدد العرقية القيحي وعلاقته بالبروتين المتفاعل - سي سارة احمد جلال، اسلام محمود محمد عبد المحسن، عيبر مصطفى كامل، هالة مغربي مغربي\*

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**البحث:** التهاب الغدد العرقية القيحي هو مرض جلدي التهابي مزمن يصيب بصيالات الشعر. كشفت بعض الدراسات عن وجود علاقة بين التهاب الغدد العرقية المقيح وأمراض أخرى مثل متلازمة التمثيل الغذائي، والاضطراب متعدد الأوجه المرتبط بزيادة خطر الإصابة بتصلب الشرايين تحت الإكلينيكي وأمراض القلب والأوعية الدموية.

**الهدف من البحث:** قياس سماكة الطبقة الداخلية للشريان السباتي كدلاله لمخاطر القلب والأوعية الدموية لدى مرضى التهاب الغدد العرقية المقيح وعلاقته بواسم المصل الالتهابي وخاصة بروتين سي التفاعلي.

**المرضى وطريقه البحث:** تم إجراء دراسة على عشرين مريضاً مصابون بالتهاب الغدد العرقية المقيح ومقارنة بعشرين متطوعاً سليماً متطابقين في الجنس ومتوسط العمر ومؤشر كتلة الجسم وتم إجراء الفحوصات التالية لجميع المشاركين: بروتين سي التفاعلي وسرعه الترسيب وعدد كرات الدم البيضاء وبروتين دهني عالي الكثافة، والكوليسترول الكلي، والدهون الثلاثية، والجلوكوز. وتم قياس سماكة الطبقة الداخلية للشريان السباتي بالموجات فوق الصوتية في الوضع.

**الاستنتاج:** لم يكن قياس سماكة الطبقة الداخلية للشريان السباتي مختلفاً بشكل كبير بين المرضى والمجموعة الضابطة، ولكنه يختلف اختلافاً كبيراً وفقاً لشده المرض لذا، قد يزيد التهاب الغدد العرقية المقيح من خطر الإصابة بأمراض القلب والأوعية الدموية ويرتبط سمك الطبقة الداخلية للشريان السباتي مباشرة بالدلالات

الإتهابية وخاصة بروتين سي التفاعلي لذلك، يمكن إعتبار بروتين سي التفاعلي علامة من مؤشرات شدة المرض ويمكن إستخدامه لمتابعه الاستجابة للعلاج.

**الكلمات الدالة:** التهاب الغدد العرقية القيحي، تصلب الشرايين تحت الإكلينيكي، أمراض القلب والأوعية الدموية، سماكة الطبقة الداخلية للشريان السباتي، الموجات فوق الصوتية للشريان السباتي.