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Serum Omentin-1 in Psoriasis

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Sir,

Psoriasis is a chronic inflammatory skin disease that affects 2 to 3% of the population worldwide and causes significant morbidity.[1]

Although the exact cause remains unknown, the evolving evidences suggest that psoriasis is a complex disorder caused by the interaction of multiple genes, the immune system, and the environment.[2]

Adipokines are pharmacologically active, low molecular weight proteins that exert pleiotropic functions through several metabolic pathways.[3]

In psoriasis, adipokines may be implicated in disease onset, progression, severity as well as in the pathogenesis of comorbidities.[4]

Omentin-1 is a fat depot-specific adipokine that is synthesised by visceral stromal vascular cells.[5]

The aim of the present work is to investigate serum levels of omentin-1 in nonobese patients with psoriasis compared with healthy controls.

This study included 45 nonobese cases with chronic plaque psoriasis and 45 age, sex and body mass index-matched healthy controls. Clinical characteristics of selected cases are summarized in Table 1. A written informed consent was taken from every participant and the study was approved by the local ethical research committee. All cases and control participants with obesity, smoking, diabetes, acute and chronic infections, malignancy, autoimmune disorders, cardiac, hepatic and renal diseases, polycystic ovarian syndrome, and dermatological disease other than psoriasis were excluded from this study. Extent and severity of psoriasis were assessed according to Psoriasis Area and Severity Index (PASI) score.[6] Serum omentin-1 was measured by enzyme-linked immunosorbent assay.

Serum level of omentin-1 was significantly lower in patients than control group and was negatively correlated with PASI score [Figure 1]. No significant association was detected between serum omentin-1 level and other clinical data of selected cases.

Low serum omentin-1 in psoriatic cases compared with healthy controls was in agreement with Ismail and Mohamed and Turan et al.[7,8]

Patients with psoriasis display impaired endothelial-dependent relaxation which may correlate with the future development of atherosclerosis and cardiovascular events[9] with increased rate of myocardial infarction and stroke.[10] Increased carotid intima-media thickness, a measure of subclinical atherosclerosis, has been demonstrated repeatedly in patients with psoriasis.[11]

Some data suggest that the likelihood of endothelial dysfunction is correlated with disease severity and disease duration.[12]

Omentin-1 directly induces an endothelium-dependent relaxation that is caused by nitric oxide produced by the

| Table 1: Clinical data of selected cases |
|----------------------------------------|
| Variable                              | Patients (n=45) |
| Age (year) (mean±SD)                  | 35.5±9.14      |
| Age of onset (year)(mean±SD)          | 29.7±6.4       |
| Disease duration (months) (mean±SD)   | 5.8±3.9        |
| PASI (mean±SD)                        | 12.38±9.1      |
| Gender, n (%)                         |                |
| Male                                  | 27 (60)        |
| Female                                | 18 (40)        |
| Family history, n (%)                 |                |
| Positive                              | 32 (71.1)      |
| Negative                              | 13 (28.8)      |
| Site of affection, n (%)              |                |
| Axial                                 | 18 (40)        |
| Extremities                           | 24 (53.33)     |
| Axial and extremities                 | 3 (6.67)       |
| Scalp affection, n (%)                |                |
| Present                               | 20 (44.4)      |
| Absent                                | 25 (55.6)      |
| Palm and sole affection, n (%)        |                |
| Present                               | 15 (33.3)      |
| Absent                                | 30 (66.7)      |
| Nail involvement, n (%)               |                |
| Present                               | 16 (35.6)      |
| Absent                                | 29 (64.4)      |
| Joint involvement, n (%)              |                |
| Present                               | 8 (17.8)       |
| Absent                                | 37 (82.2)      |
| Itching, n (%)                        |                |
| Positive                              | 34 (75.6)      |
| Negative                              | 11 (24.4)      |
| Koebnerisation, n (%)                 |                |
| Positive                              | 28 (62.2)      |
| Negative                              | 17 (37.8)      |
| Course of disease, n (%)              |                |
| Progressive                           | 33 (73.3)      |
| Stationary                            | 8 (17.7)       |
| Remission and relapse                 | 4 (8.8)        |

PASI: Psoriasis Area and Severity Index, SD: Standard deviation
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Omentin-1 exhibits powerful anti-inflammatory properties by inhibiting the inflammatory cytokine network and downregulating tumour necrosis factor (TNF)-α-induced expression of endothelial adhesion molecules and TNF-α-induced cyclooxygenase-2 expression.

The primary effect of TNF-α in pathogenesis of psoriasis is the regulation of interaction between dendritic cells and antigen-specific T-cells that drives the stimulation of T-cell responses. Also TNF-α, by the induction of IL-23 production from dendritic cells, results in enhanced Th17 responses which has a key role in cytokine network of psoriasis.

Therefore, taking all aforementioned data together, low omentin-1 may enhance psoriasis development through increasing TNF-α. Cardiovascular comorbidity in psoriasis may be explained, at least in part, by low serum omentin-1.

Nonobese psoriatic cases are also at risk of developing cardiovascular morbidity. Follow-up of psoriatic cases is mandatory to guard against cardiovascular accidents even if these cases are nonobese.

The negative correlation between omentin-1 and PASI score went with Takahashi et al. and may indicate that omentin-1 could be a useful biomarker for disease severity or monitoring the response to therapy.

And now, a question arises; what is the therapeutic value of omentin-1 in psoriasis management? Could it induce disease control? Could it prevent the associated cardiovascular morbidity? Clinical trials are needed to get the answer.

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**Conflicts of interest**
There are no conflicts of interest.

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Sir,

Nail plate dermatoscopy is a simple, quick and quite helpful technique in the diagnosis and follow-up of onychomycosis. Dermatoscopy has also been used to identify the best location in the nail plate to obtain samples for mycological examination. The dermatoscopic findings that are considered specific for onychomycosis include jagged proximal edge of onycholysis with sharp structures called spikes directed to the proximal nail fold, white-to-yellow longitudinal striae, and parallel bands of different colours called the “Aurora Borealis” pattern. Jagged proximal edge is due to progression of dermatophytes proximally along longitudinal ridges of the nail bed. However, a thick nail plate [Figure 1] can obscure deeper dermatoscopic findings of longitudinal striae and jagged proximal edge when examined with a routine white light dermatoscope [Figure 2].

We used a multispectral dermatoscope which gives a 10x magnification (DermLite DL II Multispectral, 3Gen Inc., USA) which emits light at three wavelengths, namely, 470 nm, 580 nm and 660 nm corresponding to blue, yellow and red colours, respectively, to examine this case of onychomycosis confirmed with KOH microscopic examination. Images were captured using Nikon1 AW1 14.1 MP mirrorless camera (Nikon Corp., Tokyo, Japan) and ultrasound gel was used as interface fluid. Light penetrates deeper into the tissues as the wavelength increases. Depending on the tissue, light penetrates <1 mm at 400 nm, up to 2 mm at 514 nm, and up to 6 mm at 630 nm.

In our case, though spikes and white longitudinal striae are seen with white light, the delineation was better with yellow light (580 nm) as the light penetrates deeper to highlight the nail bed features excluding the superficial distractors seen with white light [Figure 3]. The same

Figure 2:
White light dermatoscopy showing spikes (short arrow) and longitudinal striae (black arrow) (×10)

Figure 1:
Onychomycotic thick nail plate viewed from front with white light dermatoscopy (×10)