Case Report

A case of Psoriasis with an increase in HDL cholesterol

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Abstract:
Psoriasis is an immune mediated chronic skin infection which is often associated with an abnormal lipid metabolism. Studies have proved that the abnormality in lipid profile is manifested by an increase in total cholesterol, LDL and VLDL cholesterol and a decrease in HDL cholesterol. The present case of psoriasis presents with an increased HDL cholesterol. Various studies have shown the alteration in size and number of HDL lipoproteins in psoriasis which hampers the normal efflux function of HDL lipoproteins. These HDL also have a significant alteration in protein and lipid content. These small and altered HDLs are more atherogenic and thereby increase the risk of cardiovascular diseases in such psoriatic patients.

Keywords: psoriasis; HDL; small HDL; lipid metabolism

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Introduction:

Psoriasis is a chronic inflammatory, immune-mediated skin disease, which affects 2-3% of the population worldwide. Since the beginning of 20th century studies on lipid metabolism in psoriasis were conducted, and abnormal fat metabolism is considered to be an important factor in the etiopathogenesis of psoriasis[1]. Philadelphia - Collaborative research from Perelman School of Medicine at the University of Pennsylvania has shown that psoriasis patients have an increased risk of heart attack, stroke and cardiovascular death, especially if the psoriasis is moderate to severe. Also greater the area involved in psoriasis stronger is the association with abnormal lipid metabolism, manifested by an abnormal lipid profile[2].

Case report:

A 50 year old male patient was admitted in the Dermatology ward of Ruxmaniben Deepchand Gardi Medical College with complaints of multiple reddish raised lesions over back, both upper & lower limbs and scalp, shedding of white scales from the lesions and itching over the lesions since 6(six) months. There was also yellowish discolouration of nails since 1(one) month.

As detailed by the patient, he was apparently alright 6(six) months back. He then started developing multiple reddish raised lesions which initially appeared over the back, then lower limbs, scalp and upper limbs in the same order. The onset was insidious and lesions were gradually increasing in size and number. They were covered with whitish scales which shed from the lesions on their own. These were associated with mild itching over the lesions which was present throughout day and night and which relieved only on medications. The above complaints aggravated in the winter season. Since last 1(one) month, he also complained of yellowish discoloration of nails of fingers of left hand along with thickening of nail plate and deposition of debris within it. There was no history of fever, joint pain and pain abdomen.

He had no past history of tuberculosis, diabetes mellitus, hypertension, cardiovascular disease, psychiatric disease, asthma, leprosy, allergy etc. He had no history of similar illness in the family members. His bowel and bladder habits were normal, sleep was adequate, appetite was normal. He had no history of alcohol and tobacco addiction.

In General examination the patient was conscious and oriented, blood pressure was 110/80 mm of Hg, pulse was 82/min and respiratory rate was15/min. The patient was afebrile, and pallor, edema, icterus, clubbing and cyanosis were absent. All the system examinations were within normal limits.

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In local examination, lesions like plaque and scales were distributed in scalp, both upper & lower limbs and back (constituting about 38-40% of body surface area). Lesions constituted multiple erythematous well defined plaques with rough surface, irregular margins, no induration, no tenderness, covered with loosely adherent silvery white scales with positive Auspitz sign & positive Grattage test. Nails showed yellowish discolouration, thickening of nail plates, subungual hyperkeratosis, pitting positive. Hairs & mucus membrane were normal.

In blood biochemistry, fasting sugar was 86 mg/dl, post prandial sugar was 126 mg/dl, total cholesterol was 199 mg/dl, triglyceride was 57 mg/dl, HDL cholesterol was 156 mg/dl (after dilution), LDL cholesterol was 64 mg/dl (calculated), VLDL cholesterol was 11 mg/dl (calculated), creatinine was 0.5 mg/dl, SGOT was 67 IU/L, SGPT was 31 IU/L, ALP was 91 IU/L, sodium was 145 mmol/L and potassium was 4.5 mmol/L. All the blood biochemistry investigations were done by Vitros 250 dry chemistry autoanalyzer, based on reflectance photometry and manufactured by Ortho Clinical Diagnostics, a division of Johnson and Johnson, USA.

In routine blood examination, done by cell counter manufactured by Siemens, USA, WBC was 6.79 ? 10^3 U/L, Neutrophil was 55%, lymphocytes was 32%, monocytes was 7.2%, eosinophil was 4.7%, basophil was 1%, platelets was 3.08 ? 10^5 U/L and Hemoglobin was 12.1 gm/dl.

The patient was diagnosed to be suffering from psoriasis with taenia unguium.

Discussion:

Psoriasis is considered to be an autoimmune disorder, probably initiated by the overactive skin innate immune system, and maintained by immigrating activated type 1 T cells and abnormally proliferating and differentiating keratinocytes. A complex network of cytokines and chemokines mediates the pathological reaction, whereas the abnormal function of psoriatic regulatory T cells is likely responsible for the chronic nature of psoriasis[3].

Dyslipidemia in psoriatic patients is thought to be because of unhealthy lifestyle, activation of type 1 helper T cells, autoantibodies recognizing oxidized LDL and some medications used to treat psoriasis such as oral retinoids and cyclosporine[4]. In most of the studies on psoriatic patients, a statistically significant elevated level of total cholesterol, LDL cholesterol and/or triglycerides and decrease of HDL cholesterol was demonstrated[1,5,6].

HDL is a very important factor in reverse cholesterol transport. It takes part in the transport of cholesterol produced or accumulated in the peripheral tissues to the liver or other steroidogenic tissues and exerts antioxidant, antiinflammatory, antithrombotic and fibrinolytic action[1]. The two main proteins that make up HDL are called ApoA-I (75%) and ApoA-II (25%). ApoA-I is beneficial as it helps in maintaining the integrity of structure of HDL and is vital for HDL's ability to clear damaged LDL from the circulation and the walls of the arteries. New discoveries are showing that ApoA-I is also vital for HDL's enzyme functions like anti-inflammatory and antioxidant activity. The role of ApoA-II is much less understood. It is implicated as part of problems with fat metabolism and too much of it causes poor HDL function [7].

Until recently, high HDL levels were considered universally beneficial: The higher, the better. Now physicians understand that an HDL above 65, called hyperalphalipoproteinemia or HA, is too much of a good thing. Especially when HDL is greater than 90 mg/dL, the risk of arteriosclerosis rises. This is because at a very high level, HDL reverses its job and starts moving fat from the liver to body tissues. The range for norms depends upon the lab reference ranges which can go from 40 to 90. More specific testing uses the measurements for particle size/number and particle density[8].

The present case presents with an abnormal lipid profile as is expected in a case of psoriasis. The case is interesting because the patient presents with a high HDL level. It has been proved that HDL in psoriatic patients has an altered protein cargo and lipid composition. A study by Michael et. al. in Austria has shown that there is significant alteration in some 55(fifty-five) HDL associated proteins in psoriatic patients. There is also a significant decrease in the content of total cholesterol, phosphatidylcholine and sphingomyelin in HDL from psoriatic patients. Consequently, HDL from psoriatic patients are significantly less efficient in promoting cholesterol efflux. More importantly HDL cholesterol efflux capability negatively correlates with psoriasis area and severity index. Thus the beneficial role of reverse cholesterol transport performed by normal HDL cannot be performed by such altered HDL in psoriasis. Thus there is a striking alteration in the lipid metabolism of a psoriatic patient predisposing them for atherosclerosis. There is also an increased level of autoantibodies against psoriatic HDL therefore an immune complex formation may cause an early clearance of HDL and

thereby rendering HDL dysfunctional[9]. Psoriatic HDL also has an increased platelet basic protein thereby contributing to an increase in thrombotic events[10]. Again studies have shown that chronic inflammatory states like psoriasis are associated with an altered lipoprotein number and size. Very recently, it has also been shown that psoriasis is associated with an increase in small HDL particles which are associated with an increase incidence aortic inflammation[9,11]. These small HDL are defective and pro-inflammatory and consequently increases the risk of atherosclerosis. The European Prospective Investigation into Cancer and Nutrition (EPIC) - Norfolk study has shown that an increase in larger HDL and not small HDL is associated with a lower risk of first coronary events[12]. So an increase in small HDL in psoriasis may increase the cardiovascular risk in such patients and this may provide potential mechanistic insight linking psoriasis with cardiovascular diseases[2,11].Thus the increase in HDL in the present case may be due to an increase in small HDL and which definitely is not beneficial for the patients. As our clinical laboratory does not have the facility to separate the HDL fractions, so presence of small HDL in this particular case remains an assumption. But the above discussion certainly goes in favor of our assumption. Even we feel that clinicians and laboratory personals should investigate for the size of HDL whenever a patient presents with an abnormally high HDL, and manage accordingly. This can be done by completing an "expanded lipid profile" lab test with the help of Nuclear Magnetic Resonance(NMR) spectroscopy, ultracentrifugation, size exclusion or affinity chromatography, electrophoresis, and selective precipitation [2,7,12,13].

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