

## Serum Ischemia Modified Albumin Level and Erectile Functions in Individuals with Premature Greying of Hair

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### Abstract

Background: Premature Hair graying is a medical disease for which the causes and treatments are still up for debate. The etiopathogenesis of premature graying is believed to include many variables. Albumin that has had its N-terminal altered as a result of ischemia is known as ischemia-modified albumin. Some researchers believe that hormone imbalances and other biological factors are the root of erectile dysfunction. Further, the importance of oxidative stress in the development of erectile dysfunction was uncovered. The purpose of this paper is to examine the correlation between vasogenic erectile dysfunction and the amount of serum ischemia modified albumin in individuals who have premature hair thinning. Final thoughts: Possible involvement of serum IMA in the onset of premature hair graying. Its serum levels may also serve as independent indicators of vulnerability to, and severity of, premature hair graying. Those who took PGH and those who did not have statistically significant differences in erectile dysfunction. The vasculogenic kind of erectile dysfunction may be associated with IMA. Erectile dysfunction may be more common in those with polycystic ovary syndrome (PGH).

**Key words:** Topics covered include erectile dysfunction (ED), ischemia-modified albumin (IMA), and premature hair graying.

### 1. Introduction

Premature Prompt graying of the hair (PGH), also known as canities or achromotrichia, is when hair begins to gray before the age of 20 in Caucasians, before the age of 30 in African Americans, and before the age of 25 in Asians [1].

One definition of erectile dysfunction (ED) is the inability to get or keep an erection long enough to engage in sexual activity. It was formerly believed that ED was mostly a psychological issue, but now we know that it often has a physiological cause [2].

One indicator of oxidative stress is ischemia-modified albumin (IMA). In healthy individuals, IMA makes up around 1%-2% of total albumin concentration. However, in cases of ischemia and diseases that generate free radicals, including advanced cancer, liver cirrhosis, or acute infections, it rises to 6%-8%. In illnesses of the immunological system, gastrointestinal problems, or non-ischemic heart conditions, IMA does not seem to increase [3].

### 2. Materials and methods

**Data Sources:** The literature on PGH's etiology, pathophysiology, and the role of Ischemia modified albumin in PGH, as well as its potential relationship with the decrease of vasogenic erectile dysfunction up till 2024, was retrieved from the Medline databases (Pub Med and Medscape).

**Research Question Selection:** The inclusion of all research was determined by separate evaluations. Inclusion was contingent upon them meeting the following requirements: 1. Presented in an English-language format. 2. Featured in

scholarly publications that undergo a rigorous revision process. 3. Examine the origins and development of pelvic inflammatory disorder (PID). Talk about the part that ischaemia-modified albumin plays in this condition and how it may be linked to vasogenic erectile dysfunction.

**Information Retrieval:** Research was not considered for inclusion if it did not meet certain requirements. Considerations for judging the study's quality included whether or not it had received ethical clearance, the clarity of its eligibility requirements, the effectiveness of its controls, the quantity and quality of its data, and the clarity of its assessment tools. For our concerned research outcomes, data were independently extracted from all qualifying studies utilizing a data collecting form.

### Literature review:

Hair graying, often known as premature graying of the hair (PGH Canities), is a natural part of becoming older that affects people of all races and genders. Age of first gray hair is racially and ethnically specific. Premature graying of the hair is defined as occurrence before the ages of 20 for Whites, 25 for Asians, and 30 for Africans [4]. Due to the negative connotation associated with aging, premature graying of hair (PGH) may have a negative impact on a person's sense of self-worth [5].

The many hair fibers that develop over a person's life span include short, generally unpigmented vellus hair or finely pigmented intermediate hair, long, thick terminal hair shafts, and thin, unpigmented lanugo hair in the fetus or newborn. Aging also seems to alter the surface morphology of hair, namely causing the cuticular

scale size to decrease. Darkening of the scalp hair color is a common age-related phenomenon [6].

### **Caities' pathophysiology**

We still don't fully understand what causes graying. Many environmental influences are thought to interact with the underlying genetic component. The autosomal dominant disorder known as premature canities often manifests before the age of 20, however it can manifest in isolation without any underlying disease. In addition to atopic diathesis and progeria and pangeria, it may also occur with hyper- or hypothyroidism, pernicious anemia, and other organ-specific autoimmune illnesses [7].

Some dietary deficits, including as severe iron and copper shortages, chronic protein loss (from kwashiorkor, nephrosis, celiac disease, and other malabsorption-related conditions), and reversible hypopigmentation of hair have also been linked to these conditions [8].

Stress and the use of specific drugs such as chloroquine, mephenesin, phenylthiourea, triparanol, fluorobutyrophenone, dixyrazine, interferon-alpha, or medicinal oils, as well as topical agents such as dithranol, chrysarobin, resorcin, or prostaglandin F 2 alpha (PGF 2 alpha) analogs, are other potential causes [9].

Melanocytes are subjected to a higher oxidative stress load due to the incredible melanogenic activity of pigmented bulbar melanocytes in the anagen hair follicle, which can last for as long as ten years in certain hair follicles. Melanocytes produce a great deal of reactive oxygen species (ROS) through the hydroxylation of tyrosine and the oxidation of DOPA to melanin. Accumulation of reactive oxygen species (ROS) and oxidative stress, which harm the melanocyte, likely result from an aging antioxidant system [10].

The antioxidant capacity of the melanocytes in the hair follicle is overwhelmed by exogenous oxidative stress, which can be caused by pollution, ultraviolet light, psychological, emotional, or inflammatory stress, among other things. As a result, the terminal damage in the aging hair follicle is worsened [11].

Damage occurs as a result of reactive oxygen species (ROS) produced by oxidative stress, which include superoxide ( $O_2^-$ ), hydrogen peroxide ( $H_2O_2$ ), and hydroxyl free radicals. The strong reactivity of these substances makes them very unstable, and they may damage cell membranes (lipid peroxidation of membranes), DNA (single-stranded breaks in DNA), and proteins (protein fragmentation and destruction of key enzymes) [12]. Lipid peroxidation, single-strand DNA breakage, mutations, and denaturation of proteins/enzymes are some of the macromolecular and cellular damage caused by excessive ROS formation [13].

Active hair development creates less ideal circumstances for melanocyte survival in the hair follicle, which some writers have proposed as the main cause of hair graying [14]. Other possible factors include hair growth pace, anagen prolongation, and hair growth patterns.

There are a number of potential contributors to aging beyond oxidative stress. A potential reason for hair graying might be an inadequate neuroendocrine stimulation of hair follicle melanogenesis by locally generated substances, such as adrenocorticotrophic hormone,  $\alpha$ -MSH, and  $\beta$ -endorphin [15].

In addition, two patients with long-term cervical and lumbar sympathectomy reported a delay in the typical thinning of their scalp and pubic hair, respectively. This finding lends credence to the idea that sympathetic denervation may impede or at least delay the aging process of hair. A single case study hypothesized that stem cell regeneration capacity degradation due to drug misuse caused hair graying, and a substantial correlation between smoking and hair graying was also documented [16].

### **Features of a graying hairstyle**

Many people think that gray hair is harder to style and maintain than dyed hair because it is rougher and stiffer [16]. Some studies have shown that dark brown hair is less susceptible to UV damage than gray hair, which means that gray hair requires more UV protection [17]. Due to changes in the fundamental substructure of the hair strand, gray hair is more resistant to adopting artificial color and often fails to keep a temporary or permanent set [18]. Hair that is not melanized is much thicker, grows faster, and has a longer hair shaft than hair that is melanized [14]. Research has shown that white beard hair may grow up to four times longer and has stronger shafts compared to beard hair with pigmentation [16].

Points for hair thinning before its time

One numerical, objective, and repeatable way to evaluate the severity of premature canities is the Graying Severity Score (GSS). There are five distinct areas on the scalp: the front, the vertex, the temporal lobes (both sides), and the occipital. Visual inspection reveals regions of highest graying in each of these zones. A square of 1 cm<sup>2</sup> was delineated on the skin, and the hair inside that region was shaved to a height of around 1 mm above the scalp. In order to determine the percentage of white and black hair, these five squares are photographed and shown on a computer screen. Each area receives a score based on the hair count, with the proportion of gray hair in each square determining the score. The results are then evaluated as follows: 1 for less than 10% gray hair/cm<sup>2</sup>, 2 for 10% to 30% gray hair/cm<sup>2</sup>, and 3 for 30% or more gray hair/cm<sup>2</sup>. Finally, each patient's GSS is determined by adding together the

scores at the five typical locations. So, a patient may get a maximum score of 15 ( $3 \times 5$ ). We further categorize the objective scores as Mild (a score between 0 and 5), Moderate (a score between 6 and 10), and Severe (a score between 11 and 15). [19].

### Dysfunction of the erection

A large percentage of men have erectile dysfunction on occasion, which is defined as the inability to get or maintain an erection strong enough for good sexual performance [20].

While most erectile dysfunctions were formerly thought to have a psychological cause, new research indicates that an organic etiology is present in over 80% of instances [21].

Crucially, erectile dysfunction is now seen as a sign of systemic endothelial dysfunction and is no longer limited to sexual activities alone. Clinically, erectile dysfunction is a useful indicator of men at high risk of developing serious cardiovascular disease since it often occurs before cardiovascular events [22].

### erectile dysfunction pathogenesis

Contracting the smooth muscle causes the penis to stay in its flaccid condition. Several mechanisms work together to modulate the smooth muscle contraction: the adrenergic system (noradrenaline), the intrinsic myogenic system, and the endothelium-derived contracting factors (endothelins and prostaglandins). The release of nitric oxide (NO) and acetylcholine by parasympathetic cholinergic nerve fibers and nonadrenergic noncholinergic (NANC) nerve fibers, respectively, causes a rise in cyclic GMP (cGMP) concentrations, a fall in intracellular  $Ca^{2+}$  levels, and the relaxation of smooth muscle cells, all of which contribute to the erection that happens in response to sexual stimulation [20].

When smooth muscles relax, blood may flow into the corpora cavernosa's lacunar spaces, compressing the subtunical venules and blocking their outflow (a condition known as veno-occlusion). Phosphodiesterase type 5 (PDE5) hydrolyzes cGMP, inverting the process. Any disruption to any of these processes may lead to erectile dysfunction [23].

### Causes of organic ED

Neurogenic: A malfunction in nerve signaling to the corpora cavernosa is the root cause of neurogenic erectile dysfunction. An injury of this kind alters the smooth muscle's functional capacity by decreasing the amount of NO load it can draw upon [24]. Apoptosis of blood vessel endothelial cells and smooth muscle, together with increased production of fibrogenetic cytokines that cause smooth muscle to collagenize, are at the heart of the structural alterations. A venous leak, or veno-

occlusive dysfunction, is the end consequence of these alterations [25].

Vasculogenic Diseases: Erectile dysfunction may be caused by a decrease in blood flow, insufficiency or narrowing of the arteries, or endothelial dysfunction as a result of vascular disease. One of the most prevalent causes of organic erectile dysfunction is vascular erectile dysfunction. Indeed, a vascular problem may present as erectile dysfunction. Men with hypertension, diabetes, or dyslipidemia are more likely to have vasculogenic erectile dysfunction. The odds ratio (OR) for men using anti-hypertensive medication is 3.04, whereas the odds ratio (OR) for men not taking medication is 1.35. Corporal veno-occlusive dysfunction develops when the cavernosa's capacity to compress the subtunical veins declines with increasing collagen content and decreasing smooth muscle to collagen ratio.[27]

Radical pelvic surgery is the leading iatrogenic cause of erectile dysfunction. However, auxiliary pudendal artery injury may also play a role, although neurogenic damage is more common during these operations (cavernous nerve injury)[28].

Hormonal factors Discrepancies in the results of clinical trials and the fact that both hypogonadism and erectile dysfunction are common in ageing make the role of testosterone replacement therapy in erectile dysfunction controversial, despite androgens being considered the major hormonal regulator of penile development and physiology. Although there is mounting evidence linking erectile dysfunction with age-related drops in testosterone levels, this does not prove causation[29].

### Albumin Formulated for Ischemia

Ischemia modified albumin (IMA) is a systemic measure of oxidative stress and a sensitive diagnostic of myocardial ischemia during percutaneous coronary intervention [30]. Atherosclerotic lesions narrow blood arteries, which leads to oxygen deprivation and oxidative stress, which in turn converts free albumin to IMA in the blood.

A structural shift occurs at the N-terminal end of albumin in the presence of ischemia and oxidative stress. A process of oxidation and the cleavage of the first two residues occur when free radicals and free iron and copper ions are present. This specific kind of albumin is known as IMA[32].

Liver cirrhosis, infections, neoplasms, and elevated IMA levels have all been linked together [33]. While conditions like sepsis and renal failure are associated with high lactate levels, researchers have shown that IMA levels are low in these conditions [34].

Utilizing IMA for hair disorders

Some have speculated that oxidative stress has a role in the pathophysiology of androgenetic alopecia [36], and it has already been shown to have a function in alopecia areata [35].

Damage to the dermal papilla cells and eventual cell dysfunction may occur when reactive oxygen species (ROS) build up in the hair follicles and the body's antioxidant defenses are unable to neutralize them. This happens in hair illnesses [37].

In alopecia areata patients, there was a positive link between illness duration and severity. Another lipid peroxidation measure that was shown to have a favorable connection with IMA was malondialdehyde [38].

Androgenetic alopecia patients had IMA levels that were correlated with the severity and duration of their condition. The authors propose a connection between androgenetic alopecia and metabolic diseases [39] based on the fact that levels were greater in obese individuals than non-obese patients.

Researchers looked examined serum IMA levels in telogen effluvium patients and found that oxidative stress may have a role in the development of the disease [40].

Impotence and intramuscular anaesthesia

The results showed that the diabetes group had considerably greater IMA than the control group. This suggests that diabetic erectile dysfunction patients are more likely to be at risk for atherosclerosis risk factors and oxidative stress compared to the nondiabetic group [41].

A case-control study found that elevated IMA levels in type 2 DM patients may suggest an underlying subclinical vascular disease [42], suggesting that IMA is employed as a marker of ischemia alterations in this patient population.

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