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Stanozolol in dermatology: Clinical applications and safety considerations

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ABSTRACT

Stanozolol, an anabolic steroid with a high anabolic/androgenic ratio, demonstrates therapeutic potential in various dermatological disorders. Here, we examine its clinical applications in hereditary angioedema (HAE), urticaria, Raynaud's phenomenon (RP), lipodermatosclerosis, cryofibrinogenemia, and pityriasis rubra pilaris. The drug's unique pharmacokinetic profile, including high oral bioavailability and hepatic metabolism, contributes to its efficacy, particularly in HAE management through enhanced C1 esterase inhibitor (C1-INH) production. Notable benefits include reduced frequency of HAE attacks, improved digital microcirculation in secondary RP, and accelerated ulcer healing in lipodermatosclerosis when combined with compression therapy. However, its use requires careful consideration of potential adverse effects, particularly hepatotoxicity, with close monitoring becoming essential. This review highlights both the therapeutic potential and limitations of stanozolol in dermatological practice, emphasizing its role as a reserve treatment option when conventional therapies fail.

Keywords: Hereditary angioedema, Lipodermatosclerosis, Raynaud's phenomenon, Stanozolol, Urticaria

INTRODUCTION

Stanozolol is an anabolic steroid (a synthetic derivative of testosterone), having one of the largest anabolic/androgenic ratios (30:1-100:1). It is an orally active agent, having in addition to the C-17 alkylation, a pyrazole ring attached to the steroid nucleus, which enhances its anabolic properties. Although approved for the treatment of hereditary angioedema (HAE), the utility of stanozolol in treating lipodermatosclerosis, Raynaud's phenomenon (RP), urticaria, cryofibrinogenemia, and pityriasis rubra pilaris (PRP) has also been described.

PHARMACOKINETICS

Stanozolol has a high oral bioavailability, due to the C17α alkyl group, and is resistant to gastrointestinal and hepatic metabolism. It exhibits a $t_{1/2}$ of 9 h and is subsequently metabolized into glucuronide and sulfate conjugates. Excretion of the drug follows the renal route.[1]

MECHANISM OF ACTION

Stanozolol exhibits different mechanisms for various dermatologic indications. For example, in HAE, it enhances production of C1 esterase inhibitor; in urticaria, it increases levels of protease inhibitors, and in lipodermatosclerosis, stanozolol demonstrates fibrinolytic properties.[2-4]

CLINICAL USES IN DERMATOLOGY

Hereditary angioedema (HAE)

HAE is an autosomal-dominant disorder characterized by the deficiency of C1 esterase inhibitor (C1-INH). Deficiency of C1-INH prompts excessive activity of the complement system, with subsequent release of kinin-like mediators.[5] Stanozolol, following hepatic metabolism, enhances the production of C1-INH. This effect, however, is not observed with parenteral anabolic steroids, thus propounding the necessity of hepatic metabolism for stanozolol to exhibit its therapeutic effects in HAE.[2] Although conventional doses of stanozolol (0.5-2 mg/day) clearly reduce the frequency and intensity of episodic attacks in HAE; correction of biochemical complement abnormalities is seldom achieved. Nevertheless, as attacks in HAE are controlled with conventional doses of stanozolol, attempting to completely correct the biochemical abnormality by escalating the dose becomes unnecessary.[6]

However, when used for short-term prophylaxis, higher doses of stanozolol have been employed in patients undergoing oral manipulation (e.g., endoscopy and dental procedures), due to the risk of attacks of laryngeal edema following oral trauma. In such cases, 6 mg/day of stanozolol can be given 7 days prior and 3 days following the procedure.[7]

For HAE, the suggested treatment algorithm with stanozolol is outlined in Figure 1.[7]

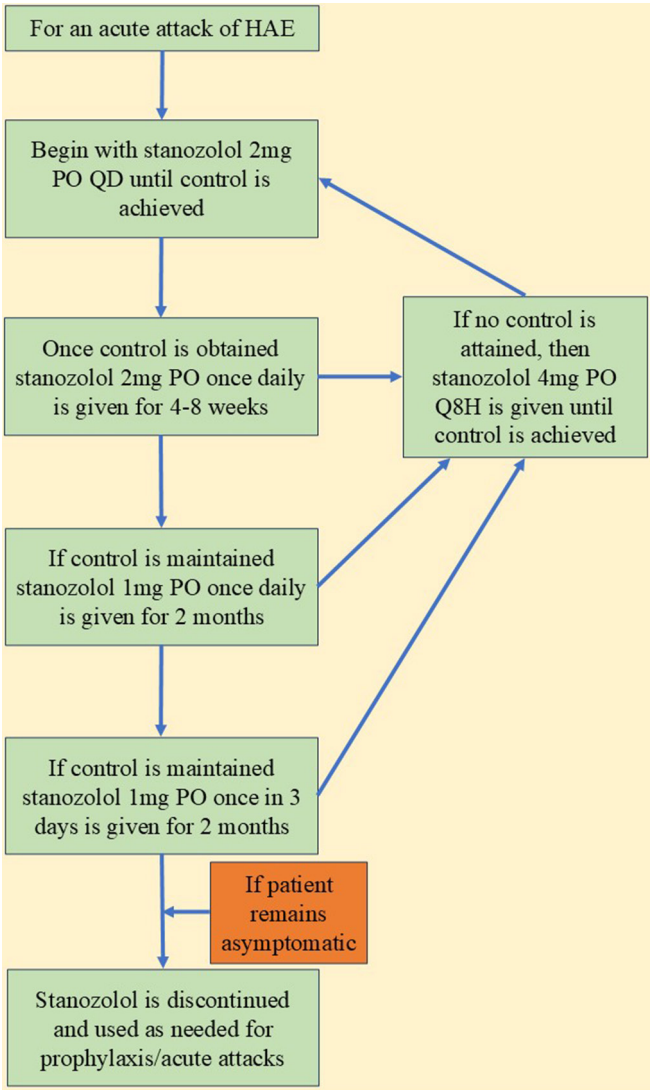

Figure 1: Treatment algorithm for stanozolol in hereditary angioedema. HAE: Hereditary angioedema, PO: *Per os*. (by mouth), QD: *Quaque die* (once a day), Q8H: *Quaque octava hora* (every 8 hours)

Urticaria

Rare cases of chronic urticaria and familial cold urticaria have demonstrated benefit with stanozolol. It is theorized that these conditions are caused by deficiencies of protease inhibitors (as seen in HAE), thus outlining the rationale for stanozolol use.[7,3] Nonetheless, as stanozolol is associated with potential side effects, it should be employed only following the failure of conventional therapy.

Raynaud's phenomenon (RP)

RP presents with episodic attacks of digital ischemia involving few digits of the hands and feet. Interestingly, stanozolol's utility is observed only in secondary RP.

In secondary RP, there is hyper-viscosity, hyper-fibrinogenemia, and reduced fibrinolytic activity; factors against which stanozolol acts, thus demonstrating benefit.[8-11]

In a double-blind cross-over study of the effects of stanozolol (5 mg Q12H) on both primary and secondary RP, patients with primary RP did not delineate any improvement. In contrast, in patients with secondary RP, digital microcirculation substantially improved, despite the absence of subjective improvement.[12]

Nonetheless, stanozolol needs to be kept as a reserve drug for unresponsive cases and should be used only if necessary.

Lipodermatosclerosis

The existence of a faulty fibrinolytic system is evident in patients with lipodermatosclerosis; and based on its fibrinolytic property, stanozolol is employed in treating this condition. A schematic representation of the mechanism of stanozolol in lipodermatosclerosis is outlined in Figure 2.[4,13,14]

However, although lipodermatosclerosis is reduced following treatment with stanozolol, an increased rate of ulcer healing of the involved skin is not elucidated. Besides, stanozolol

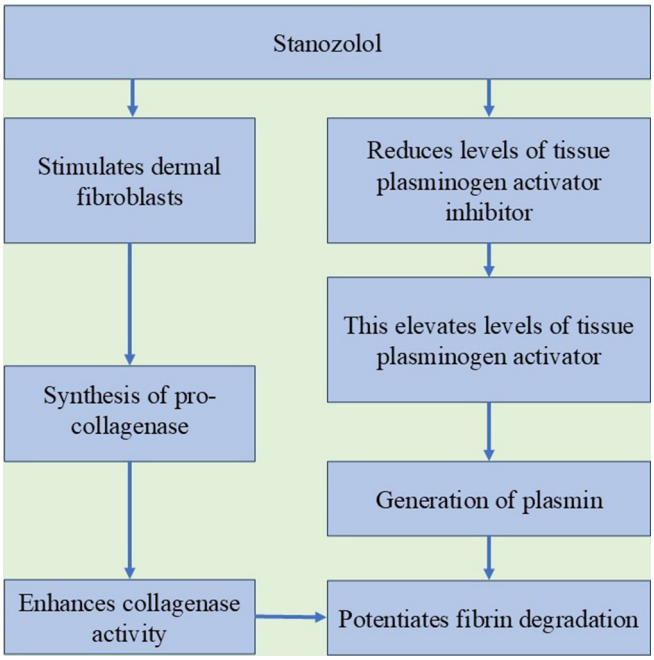

Figure 2: Mechanism of action of stanozolol in lipodermatosclerosis.

does not have any protective effect on ulcer recurrence. The exact reason for this variability though remains elusive.[15-17]

Moreover, it is observed that dramatic responses with low-dose stanozolol therapy are more common in the acute phase of lipodermatosclerosis; wherein there is a remarkable reduction of pain within few weeks, and by 2-3 months, significant reduction of skin induration is observed.[18]

In a double-blind cross-over trial, stanozolol plus compression stockings in lipodermatosclerosis was compared with placebo plus compression stockings. Although both treatments significantly reduced the area affected by lipodermatosclerosis, the mean rate of improvement in the stanozolol group was twice as great in comparison to the placebo arm. Further, in patients on stanozolol, significant reduction in blood fibrinolysis, mean plasma fibrinogen levels, and extravascular fibrin deposits were noted.[15]

In another study, stanozolol along with elastic stockings, highlighted an increase of ~100% in the healing rate of affected skin in patients with lipodermatosclerosis.[16]

The use of stanozolol in lipodermatosclerosis is best applied as an adjuvant and with appropriate monitoring, best results can be obtained, without encountering side effects associated with its use.

Cryofibrinogenemia

Ulceration resulting from cryofibrinogenemia is associated with severe pain and is usually unresponsive to most treatments. Dermal capillaries of these patients elucidate intravascular fibrin thrombi.

Kirsner *et al.*,[19] in a series of eight patients (seven with essential cryofibrinogenemia and one with cryofibrinogenemia resulting from lymphoma), outlined stanozolol to be effective in bringing about rapid pain resolution of cryoglobulinemic ulcers in all eight patients, with complete healing of ulcers in seven patients. Besides, there was an improvement in purpura and livedo reticularis as well. Interestingly, in four patients, cryofibrinogen was not detected in the plasma, and intravascular fibrin thrombi were not identified in biopsies of healed ulcers, thereby pointing to the beneficial effects of stanozolol in cryofibrinogenemia.

Pityriasis rubra pilaris (PRP)

Stanozolol has shown to be of value in the management of PRP, though limited to very few reports.[20-22] Stanozolol, in PRP, elevates levels of retinol-binding protein, thereby optimizing the transport of vitamin A to the skin and in this way helps in normalizing the disrupted keratinization.[20]

In PRP, stanozolol is generally given as 2 mg Q8H, for a period of around 6 weeks. Body lesions are first to clear, with palmar/plantar lesions taking a longer time to heal, which usually occurs by 1 month.[20,21]

However, in the report by Brice and Spencer,[22] conflicting results were obtained. Out of five patients, two did not delineate any improvement following stanozolol therapy, making the authors conclude the suboptimal potential of stanozolol in PRP.

As there exist very few publications with regard to the utility of stanozolol in PRP (level of evidence "E"), and that too of historical significance, it is difficult to obtain a concrete conclusion regarding the same.

ADVERSE EFFECTS

Table 1 outlines the side effects of stanozolol.[23] Of the adverse effects, the development of new or worsening acne, sleep disturbances, headache, and changes in sexual desire are commonly encountered. Rarer side effects, though important and warranting caution, involve the hepatic, renal, and cardiovascular systems.[23]

USE IN SPECIAL POPULATIONS

Stanozolol is an absolute contraindication in pregnancy and lactation.[23] In children and elderly, stanozolol needs to be used with caution owing to its propensity for hepatotoxicity.[23]

DRUG INTERACTIONS

- Stanozolol when prescribed with oral hypoglycemic drugs can increase their hypoglycemic effects[23]

Table 1: Side effects of danazol.

S No.	System affected	Side effects
1	Hepatic	Elevation of hepatic transaminases
		Peliosis hepatitis
		Hepatocellular adenomas and carcinomas
		Cholestatic jaundice
2	Cardiac	Sodium retention
		Worsening of hypertension
		Congestive cardiac failure
3	Skeletal	Premature closure of epiphysis
4	Psychogenic	Euphoria
		Anger
		Increased libido
5	Dermatologic	Exacerbation of acne vulgaris
		Rosacea
		Seborrheic dermatitis
		Folliculitis
		Hirsutism
6	Androgenic	Females
		Deepening of voice
		Menstrual irregularities
		Clitoral hypertrophy
		Males
		Impairment of spermatogenesis
		Exacerbation of prostatic hypertrophy
7	Lipid	Increase in low-density lipoproteins
		Decrease in high-density lipoproteins

- Stanozolol when used with anticoagulants can potentiate their effects and need careful monitoring[23]
- When used with other steroid molecules, stanozolol can increase the fluid-retaining properties of these drugs.[23]

MONITORING GUIDELINES

Owing to the propensity for hepatotoxicity with stanozolol, it becomes mandatory to evaluate the hepatic profile at baseline. Further, as stanozolol has been linked with the development of hepatic adenomas (in some cases), a baseline ultrasonography would be helpful. Furthermore, the lipid profile, complete hemogram, and prostate-specific antigen at baseline are suggested.[23] Ongoing evaluation, every 3 months for hepatic transaminases would assist in assessing the hepatic profile of patients receiving stanozolol. However, there are no strict monitoring guidelines with regard to stanozolol currently.[24]

CONCLUSION

Stanozolol, though effective in a number of dermatologic conditions, is never the first-line drug. Further, owing to the associated side effects, it needs to be used with caution, along with careful monitoring.

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