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Case Series

Seasonal Variation in Erectile Dysfunction: A Case Series of Three Patients and Review of Clinical Implications

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ABSTRACT

Erectile dysfunction (ED) is a prevalent condition affecting millions of men worldwide. Risk factors for ED are known to be more prevalent during some seasons, yet the potential seasonal variation in ED symptom severity remains underexplored. In this case series we present three men who reported marked seasonal fluctuation in erectile function. Each patient's pattern, associated factors, evaluation and response to targeted interventions are described. The cases illustrate multiple plausible mechanisms linking seasonality to erectile dysfunction (ED) — mood and circadian changes (seasonal affective disorder), environmental temperature and vascular reactivity, and seasonally-varying comorbidities/medication exposure (e.g., allergy medications, cardiovascular stress). We discuss a pragmatic diagnostic workup and management approach, and highlight the need for clinicians to ask about seasonal patterns when assessing ED.

Keywords*: Seasonal variation, (SAD), Erectile dysfunction (ED), Medication-related sexual dysfunction.*

INTRODUCTION

Erectile dysfunction (ED) is a multifactorial disorder with vascular, endocrine, psychological and iatrogenic etiologies.[1] Seasonal patterns in sexual function have been reported, including fluctuations in testosterone levels,[2] mood changes associated with seasonal affective disorder (SAD),[3] and environmental effects on vascular tone.[4] Cold exposure increases peripheral vasoconstriction and worsens vascular reactivity,[5] which may aggravate ED in individuals with cardiovascular disease. Seasonal illnesses such as allergic rhinitis also contribute to sexual dysfunction via sleep disturbance and medication effects.[6] Despite these associations, seasonal variation in ED remains underreported in psychiatric and sexual-medicine literature. We present three cases demonstrating clinically significant seasonal patterns.

Case 1 — Winter-predominant ED with mood features

A 45 yrs old male, educated up to 12th, self-employed, living in Hindu joint family of middle-socio-economic status of urban background of Delhi. Patient presented in Psychiatry OPD of tertiary care centre and reported near-normal erectile function from April–October but progressive loss of morning erections and decreased ability to achieve intercourse from November through February for the last three years. Sexual desire decreased in winter months. Along with erectile dysfunction patient also reported low energy, hypersomnia, carbohydrate craving and social withdrawal in winter months (suggestive of seasonal affective symptoms). No significant medical comorbidity. Non-smoker, occasional alcohol. No new medications. All the basic and regular blood investigation was normal. IIEF-5 score in winter: 8 (moderate ED). Moderate depressive symptoms during winter; daytime serum total testosterone borderline low (mid-normal in summer, low-normal in winter), TSH normal, fasting glucose and lipids within reference ranges. Seasonal pattern ED likely contributed by seasonal mood change (SAD spectrum) with associated libido reduction and relative fall in morning testosterone.

Patient was initiated with bright light therapy (30 minutes morning), sleep–wake regularization, short course of cognitive behavioural strategies targeted to seasonal mood, and on-demand PDE-5 inhibitor for intercourse. Over two winters of combined therapy he reported marked improvement in mood and partial restoration of erectile function; IIEF-5 improved to 17 in winter months.

Case 2 — Spring-onset ED temporally linked to allergic rhinitis and antihistamine use
A 30-year-old male, graduate, unmarried, bank employee, living in Muslim joint family of middle-socio-economic status of rural background of Delhi presented in Psychiatry OPD with history of two years of intermittent ED occurring each late spring (March–May), resolving by July. Along with this patient also reported severe seasonal allergic rhinitis during the symptomatic months with nasal obstruction, poor sleep and daytime fatigue. He began taking first-generation antihistamines (over-the-counter diphenhydramine) nightly to help sleep during the allergy season. IIEF-5 during symptomatic months: 10. No depressive symptoms on assessment. Morning testosterone normal. Sleep study not performed but partner reported snoring during allergies. Medication review showed nightly diphenhydramine use. All the basic regular blood investigation like CBC, TFT, HbA1c, LFT, KFT found to be normal.

Seasonal ED likely mediated by combination of sedating first-generation antihistamine (anticholinergic) effects lowering libido/erectile responsiveness and nasal obstruction degrading sleep and increasing fatigue. Patient was advised to stop first-generation antihistamines; switched to non-sedating second-generation antihistamine and intranasal corticosteroid. Emphasized nasal hygiene, nasal dilators for sleep. Sexual function improved within weeks; IIEF-5 returned to baseline (≥22) by late spring after medication change and better nasal control.

Case 3 — Cold-season worsening of ED in a patient with vascular disease
A 55-years-old man, graduate, married, government employee, living in Hindu nuclear family of upper-middle-socio-economic status of Urban background of Haryana presented in Psychiatry OPD with complaint of adequate erections from May–September but a progressive reduction in erectile rigidity and frequency from October–March for five years. Symptoms peaked in the coldest months. Patient also complained of increased exertional angina episodes and reduced physical activity in winter, weight gain in the colder months, and difficulty with outdoor activity due to cold. As patient was known case of ischemic heart disease and hypertension. He was on beta-blocker and a thiazide diuretic.

Investigations: IIEF-5 in winter: 7. Cardiovascular evaluation showed stable coronary disease; BP control reasonable but slightly higher in winter. Morning testosterone normal. Vascular risk factors: dyslipidaemia, controlled diabetes. Nocturnal penile tumescence testing suggested reduced nocturnal tumescence in winter months. Seasonal worsening of ED likely due to increased peripheral vasoconstriction and vascular load in cold weather (cold-induced vasoconstriction), reduced physical activity, and possible medication contribution (beta-blocker). Cardiovascular disease was an important baseline contributor.

Multidisciplinary approach was initiated. Optimize cardiovascular risk (exercise program adapted for cold weather, smoking cessation counselling was done), review antihypertensive regimen with cardiology (switched from a non-selective beta-blocker to a vasodilatory antihypertensive where appropriate), trial of daily PDE-5 inhibitor in winter months under cardiovascular supervision. Patient reported partial improvement in winter symptoms over two seasons with improved exercise tolerance and medication adjustment.

DISCUSSION

These three cases illustrate different, clinically plausible mechanisms for seasonal variation in erectile function:
In first case winter-predominant ED with seasonal affective features:-Decreased libido and erectile function during winter months have been associated with SAD-related reductions in dopaminergic activity and circadian dysregulation.[7] Seasonal variation in testosterone has also been reported, with lower levels during winter.[2]

In second case spring-related ED associated with allergic rhinitis and antihistamine use: First-generation antihistamines have recognized adverse sexual effects due to anticholinergic sedation and decreased libido.[8] Allergic rhinitis significantly affects sleep quality, fatigue and sexual satisfaction.[9]

In third case cold-season ED in a patient with vascular disease: Cold temperatures lead to increased sympathetic vasoconstriction and hemodynamic stress,[5] which can negatively influence erectile hemodynamic. ED and coronary artery disease share endothelial-dysfunction pathways.[10]

Clinical implications: When history reveals seasonal fluctuation in ED clinicians should:

- Take a targeted seasonal history (timing, duration, associated mood/sleep/allergy/medication changes).
- Screen for mood disorders, sleep disturbance, and nocturnal erections.
- Review medications for seasonal agents (e.g., antihistamines, sedatives).
- Obtain baseline metabolic and endocrine tests (fasting glucose, lipids, TSH, morning testosterone) as clinically indicated.

- Consider cardiology/urology/endocrinology referrals when vascular or endocrine disease suspected.
- Offer targeted treatments timed to the seasonal trigger (light therapy for SAD, switch antihistamines, optimize cardiovascular meds and activity, PDE-5 inhibitors, testosterone therapy only when deficiency confirmed).

Research implications: The prevalence and mechanisms of seasonal ED remain underexplored. Prospective monitoring (e.g., repeated IIEF-5, hormone sampling across seasons, actigraphy) would clarify effect sizes and modifiable contributors.

CONCLUSION

Seasonal variation in psychological wellbeing, testosterone secretion, sleep quality and vascular function can influence erectile capacity. Light therapy is effective for seasonal major depression and associated sexual impairment,[11] and PDE-5 inhibitors remain a first-line treatment for ED with demonstrated benefit across etiologies.[12]

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