

Risk Assessment of Internal Malignancies in Patients with Vitiligo

Introduction

To date, studies examining the associations between cancer and vitiligo have had mixed results.

Hypothesis

Vitiligo patients will have decreased rates of internal malignancies because of the increased systemic immune surveillance.

Methods

- Abstracted data from the charts of 1484 vitiligo and 2988 age- and sex-matched patients
- Compared cancer prevalences, controlling for BMI (earliest recorded), smoking status, and alcohol use
- Used logistic regression models to analyze the associations

Key Takeaways:

- Vitiligo patients were more likely to have a history of a solid-tumor, non-skin cancer and were more likely to have a history of more than 1 cancer type.
- Also, vitiligo patients had a higher likelihood of facing breast, ovary/uterus/cervix, and prostate cancers.
- Significantly decreased risks were noted for lung cancer.
- Patients with unilateral vitiligo were significantly more likely than those with bilateral vitiligo to have a history of a solid-tumor, non-skin cancer, skin cancer, or female breast cancer.
- Our study extends prior registry-based observations by controlling for known cancer risk factors.
- Future directions: Implications for cancer screening, mechanisms involved, time course of the vitiligo and cancer events, and connections to the effects of cancer immunotherapy treatments.

Results

Vitiligo vs Control

Cancer	OR	95% CI	P-value
Solid non-skin	1.667	1.242-2.238	0.0007
Breast	1.877	1.092-3.227	0.0228
Ovary/Uterus/Cervix	2.693	1.036-6.998	0.0420
Prostate	4.356	2.139-8.869	<0.0001
2 or more cancers	4.568	2.063-10.114	0.0002

Unilateral vs Bilateral

Cancer	OR	95% CI	P-value
Solid non-skin	3.474	1.510-7.994	0.0034
Breast	3.341	1.014-11.008	0.0474
Skin	5.785	2.396-13.968	<0.0001

Discussion

Enhanced carcinogenesis may be seen in vitiligo patients because of chronic systemic inflammation and frequent use of immunosuppressants to control vitiligo.

Why vitiligo affects tissue-specific cancer rates differently may be explained by epitope spreading where immune cells involved in targeting melanocytes acquire specificity for some other tissue types, but also not for some tissue types.

