Skin and Wound Issues in Patients with Parkinson’s Disease: An Overview of Common Disorders

Janice M. Beitz, PhD, RN, CS, CNOR, CWOCN, CRNP

Abstract

Parkinson’s Disease is a chronic neurodegenerative disorder that is expected to increase in coming decades as the American population continues to age. Although the motor dysfunction (bradykinesia, tremor, rigidity) of Parkinson’s Disease is well described in the literature, the nonmotor dysfunction related to autonomic system changes is not as commonly addressed. Ironically, nonmotor changes, such as seborrhea, sialorrhea, hyperhidrosis, and sensory denervation occur earlier in the disease process and exert a profound effect on patients’ quality of life. The depletion of dopamine, a critically important neurotransmitter, is the critical pathology of Parkinson’s disease. Therapies targeting this abnormality and the effect of insufficient dopamine itself can affect the integumentary system and potentially wound healing. The purpose of this review is to describe changes in the autonomic nervous system due to Parkinson’s Disease with a focused overview of common skin and wound care issues that may affect wound care clinician practice. Implications for nurses and other clinicians caring for Parkinson’s Disease patients include surveillance for melanoma and other skin cancers, skin protection against excessive moisture or the effects of insufficient moisture, monitoring of wound healing progress, and interventions to prevent or ameliorate complications of immobility.

Keywords: Parkinson’s Disease, autonomic dysfunction, melanoma, hyperhidrosis, sialorrhea

Background

Parkinson’s Disease is a progressive neurodegenerative disease that affects approximately 1% of people over the age of 60 years. In the United States, it is estimated that one million persons currently live with Parkinson’s Disease, with about 60,000 new cases diagnosed yearly.1,2 With the aging of the American population, the incidence and prevalence of Parkinson’s Disease are expected to rise.

Parkinson’s Disease hallmark is the loss of dopaminergic neurons of the substantia nigra. A pathological cellular hallmark is the presence of the Lewy Body, which is found without exception in Parkinson’s Disease. These intracellular inclusion bodies and the loss of dopamine characterize the critical pathophysiology of the disorder.3,4 Parkinson’s Disease is the most common neurodegenerative movement disorder but is now widely recognized as a complex nonmotor multifocal disease as well.5,6 In addition to the classic cardinal features of tremor at rest, postural instability, rigidity, and akinesia, and possibly some cognitive impairment,7 Parkinson’s Disease also is characterized by a number of autonomic dysfunction issues.5,8 What may not be as well recognized in the non-neurology community is the effect of Parkinson’s disease on the skin and its appendages (hair, nails).9

The purpose of this review is to describe changes in the autonomic nervous system due to Parkinson’s Disease with a focused overview of common skin and wound care issues that may affect wound care clinician practice.

Background

Nonmotor symptoms intrinsic to Parkinson’s Disease occur earlier than motor symptoms and can affect quality of life substantially. Autonomic physiology changes in Parkinson’s Disease underlie several nonmotor symptoms including orthostatic dizziness, gastrointestinal slowing, erectile dysfunction, and swallowing problems.10,11 As autonomic function deteriorates, patients can develop constipation, urinary urgency, frequency, nocturia, and urinary incontinence. It is often these nonmotor features that present the greatest management challenge.12,13 What is lacking in Parkinson’s Disease

Dr. Beitz is a Professor of Nursing, School of Nursing-Camden, Rutgers University, Camden, NJ. Please address correspondence to: Janice M. Beitz, PhD, RN, CS, CNOR, CWOCN, CRNP, 4 Coventry Court, Cherry Hill, NJ 08002; email: Janice.Beitz@camden.rutgers.edu.
is drug therapy that is disease-modifying, such as has occurred in rheumatoid arthritis therapy.3,14,15 As the disease progresses, both motor and nonmotor symptoms worsen. In addition, no good predictive tests or symptom screens are yet available in the pre-symptomatic interval of Parkinson’s Disease.15

The autonomic changes of Parkinson’s Disease are manifested in many signs/symptoms that can affect the skin and its appendages such as the hair. These include sialorrhea (excessive salivation), seborrhea, hyperhidrosis (excessive sweating), sensory dysfunction/denervation of the skin, and sympathetic changes.10,16-20 Although exact occurrence of these autonomic changes is difficult to obtain, multiple authors describe them as “common” or “frequent” in Parkinson’s Disease.19,21,22 A comparative descriptive design study10 analyzed 70 Parkinson’s patients and 22 control subjects; hyperhidrosis was noted in 36 Parkinson’s Disease patients, and 18.6% of Parkinson’s Disease patients had seborrhea.

The skin can be affected by Parkinson’s Disease therapy as well. Medication side effects, untoward effects, or drug delivery methods can cause iatrogenic lesions. In addition, the long-term motor deterioration of Parkinson’s Disease can alter the skin. As the occurrence of immobility and nonself-repositioning (related to possible “off” periods) rises, the risk for development of pressure ulcers also rises (see Table 1).23,24

Pathophysiology of Autonomic Nervous System Changes in Parkinson’s Disease

The autonomic nervous system can be divided into the central autonomic networks, sympathetic pathways, parasympathetic pathways, and the enteric nervous system. Parkinson’s Disease pathology affects these structures through a presynaptic protein called α-synuclein. When α-synuclein aggregates in the neuronal structures of the autonomic system, evidence supports that this aggregation is a precursor to nerve degeneration.11 The pathology of Parkinson’s Disease affects autonomic innervation to several organ systems, including the cardiovascular, urinary, and gastrointestinal systems, generating the previously described nonmotor symptoms.11

Skin alterations. What may be less recognized by wound clinicians and other non-neurological specialists are the alterations in skin regulatory mechanisms associated with Parkinson’s

| Table 1. Parkinson’s Disease terminology58, 61
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Akinesia</td>
<td>Delay in initiating movement, inability to move, “freezing”</td>
</tr>
<tr>
<td>Amantadine</td>
<td>A drug improving symptoms of Parkinson’s Disease by increasing dopamine; classified as an antiviral agent</td>
</tr>
<tr>
<td>Anticholinergic drugs</td>
<td>Group of drugs decreasing action of chemical acetylcholine; these drugs help reduce rigidity, tremors, and drooling in Parkinson’s Disease</td>
</tr>
<tr>
<td>Apomorphine</td>
<td>A form of morphine that can increase the amount of dopamine available in the brain; used as rescue therapy in cases of freezing or other worsening symptoms</td>
</tr>
<tr>
<td>Bradykinesia</td>
<td>Slowness of movement</td>
</tr>
<tr>
<td>Dopamine agonists</td>
<td>A class of drugs used to treat Parkinson’s Disease; these drugs copy the effect of the brain chemical dopamine and increase available dopamine</td>
</tr>
<tr>
<td>Dyskinesias</td>
<td>Involuntary nontremor movements</td>
</tr>
<tr>
<td>Levodopa</td>
<td>A drug containing a form of the brain chemical dopamine; used to treat Parkinson’s symptoms</td>
</tr>
<tr>
<td>Livedo reticularis</td>
<td>Cutaneous finding presenting as a mottled network of erythematous to violaceous discoloration; usually a benign condition affecting lower extremities</td>
</tr>
<tr>
<td>MAO-B inhibitors</td>
<td>Newer class of drugs used to treat Parkinson’s Disease; includes rasagiline and selegiline</td>
</tr>
<tr>
<td>“Off” periods</td>
<td>Relatively poor overall function and mobility corresponding to the PD medication not working (eg, slowed thinking, fatigue, irritability, drenching sweats)</td>
</tr>
<tr>
<td>Postural instability</td>
<td>The tendency to fall without explanation usually when pivoting</td>
</tr>
<tr>
<td>Rigidity</td>
<td>Stiffness of the limbs and trunk</td>
</tr>
</tbody>
</table>
Disease. Not only are eccrine sweat glands altered, but also the innervation to skin blood vessels and erector pilli muscles is decreased. Research has supported that sensory deficit in Parkinson’s Disease results from cutaneous denervation. Parkinson’s Disease patients have increased thresholds for cold, warm, cold and heat pain, mechanical pain, and touch. Two comparative descriptive studies examining Parkinson’s Disease patients versus controls support that this autonomic denervation occurs early in Parkinson’s Disease and is more prevalent than previously thought. Skin biopsy and measurement of sympathetic skin response, respectively, showed more absent skin responses and innervation loss in Parkinson’s Disease patients.

Another neurologic change in Parkinson’s Disease related to the skin is alteration in microRNAs (miRNAs). MiRNAs are small, endogenous, noncoding RNAs (ribonucleic acids) that regulate many protein-coding genes. MiRNAs play critical roles in important physiologic processes such as the cell cycle, cell differentiation, and cell development, growth, and apoptosis. MiRNAs also play some role, although not fully clarified, in angiogenesis. MiRNAs are involved in skin morphogenesis, hair follicle morphogenesis, skin carcinogenesis, and autoimmune and inflammatory disease affecting the skin such as in systemic lupus erythematosus. It is known that cutaneous wound healing is incomplete without functional miRNAs. Altered expression of miRNAs has been identified in Parkinson’s Disease. Although more research is needed to elucidate the impact of this alteration on skin health and wound healing in Parkinson’s Disease, impaired wound healing may theoretically occur as the disease progresses.

**Skin and Wound Challenges**

**Sialorrhea.** Hypersalivation is a common symptom of Parkinson’s Disease. The exact pathomechanism of this increased salivation is not clear. Parkinson’s Disease patients may have difficulty swallowing, but the excessive salivation occurs in addition to this change. In some instances, patients have good response to injection of botulinum toxin in the lingual, salivary, and parotid glands. From an integumentary system effects perspective, excessive salivation can irritate the skin around the mouth and upper chest (if drooling occurs). Skin protectants may be necessary, but they must be types that are safe around the face and oral cavity. In other words, oil- or petrolatum-based products would not be recommended because the potential exists for lipid pneumonia secondary to aspiration of lipids. Drug therapy usually is used to control the situation, including anticholinergic agents (to dry up secretions) or weak anticholinergic antidepressants, especially with concomitant depression. Belladonna compounds also can be used to decrease saliva production.

**Hyperhidrosis.** Hyperhidrosis, or excessive sweating, is a frequent nonmotor complaint of Parkinson’s Disease. The excessive sweating involves mainly the face, head, and trunk. Conversely, sweating decreases in the palms of the hands in some Parkinson’s Disease patients. One study compared 13 Parkinson’s Disease patients with excessive sweating versus 37 Parkinson’s Disease patients with no hyperhidrosis and identified the difference in axial and palmar sudomotor skin response. The investigators suggest this axial hyperhidrosis could be a compensatory phenomenon for reduced sympathetic function in the extremities, but this is only a theory. Hyperhidrosis can be caused by too-high or too-low doses of dopamine or dopamine promoting drugs. Off-period hyperhidrosis can be treated with dopamine agonist (eg, L-Dopa) dosage increases. Beta-blockers (eg, propanolol) and injections with botulinum toxin also have been used. Drenching sweats, especially at night, can be notably troublesome.

**Seborrhea.** Chronic seborrhea is a common nonmotor symptom of Parkinson’s Disease. The outcome is greasy skin on the face that can be associated with erythema and scaly patches in skin creases. The hair also can be very oily with dandruff. The oiliness seems to be associated with periods when the disease is most active. When seborrheic dermatitis occurs around the eyes, flaky skin may involve the eyelashes and get into the eyes. Irritation of the face and eyes can be very troublesome to the patient.

The condition has been acknowledged for decades. In 1977, Flint described the skin of the Parkinson’s Disease patient as “coarse and thickened” and the epidermis as shiny and glistening. She noted the oily limp appearance of the hair. The mechanism by which the seborrhea develops is not well understood but is likely the impact of altered dopamine state on glandular function. Whether seborrhea contributes to any impaired wound healing is unknown.

**Malignant skin lesions.** Convincing epidemiologic evidence suggests a negative association between Parkinson’s Disease and many cancers. Conversely, others support that melanoma and other nonskin cancers occur more often in Parkinson’s Disease. Inzelberg et al conducted a cross-sectional study in which 490 Jewish patients in a Parkinson’s Disease clinic were genotyped and then assessed for oncologic history. Patients with Parkinson’s Disease with a specific genetic mutation had higher likelihood of nonskin cancers such as breast, prostate, stomach, and colon cancer.

Melanoma, a cancerous tumor of melanin-producing cells in the skin, occurs more frequently than should be expected in Parkinson’s Disease patients. No identified biologic mechanism is available; some but not all sources suggest levodopa may increase the risk of melanoma.

The nature of the Parkinson’s Disease-skin cancer (melanoma) relationship is complex. Some reviews suggest skin cancer rates are higher in Parkinson’s Disease patients due to the disease process itself, while others suggest that dopamine therapy in its many forms is the critical factor. This focus is not a new area of study. The possible association between levodopa use and the development of malignant melanoma has been described since the 1970s. It is hypothesized that a genetic profile contributes to both melanoma development
among Parkinson’s Disease patients. Larger population studies are needed.

Schwid et al. studied skin cancer incidence in Parkinson’s Disease patients treated with a new agent (CEP-1347) designed to slow cell death of neuronal cells. Their multicenter study of 806 patients with early Parkinson’s Disease found that non-melanoma skin cancers were the most common type, but they did not occur at an excessive rate due to the drug. The incidence of melanomas was 20.9 times that predicted in the general population. This increased rate was seen in patients who had never received dopaminergic therapy. The researchers proposed that Parkinson’s Disease itself increases melanoma risk unrelated to dopaminergic therapy.

### Drug Therapy and Parkinson’s Disease: Latrogenesis

**Levodopa and malignant melanoma.** Levodopa, the major drug used in Parkinson’s Disease therapy, has been implicated in the development of malignant melanoma (see Table 2). Despite the previously described research findings, common drug guides for clinicians continue to warn against using levodopa in patients with suspicious skin lesions or a history of melanoma. The concept developed because of “shared biochemical pathways between synthesis of both dopamine and melanin.” Levodopa is an important substance in the formation of melanin.

In 2003, Fiala et al. performed a systematic review of the literature and reviewed case studies from their home institution on the association between levodopa use in Parkinson’s Disease and melanoma. They found that in a total of 54 patient cases, the occurrence of both Parkinson’s Disease and melanoma was coincidental rather than causal. Vigilance about the issue is warranted, because the controversy exists and the warning about melanoma and dopaminergic therapy persists in drug references. For example, Lehne states levodopa can “activate malignant melanoma and consequently should be avoided in patients with undiagnosed skin lesions.”

**Apomorphine and skin necrosis.** Apomorphine is a dopamine agonist that is prescribed for treatment of acute, intermittent off episodes associated with Parkinson’s Disease. Considered “rescue” therapy for off periods, apomorphine is given by injection or infusion.

Although generally benign, apomorphine can be associated with skin damage. Ganesalingam and Bain described a case study of a 57-year-old man receiving apomorphine infusion subcutaneously who developed necrotic skin ulcers despite the fact that he had received the drug for 4 years without problem. His necrotic ulcers resolved 1 month after stopping apomorphine.

**Transdermal drug delivery skin reactions.** Some anti-Parkinson drugs are available in transdermal patches. The benefit of this delivery system is substantial in that it can lower the pill burden of Parkinson’s Disease and help with compliance. In addition, transdermal delivery systems can

---

**Table 2. Parkinson’s Disease medication therapy: commonly used agents**

<table>
<thead>
<tr>
<th>Dopamine</th>
<th>Dopamine agonists</th>
</tr>
</thead>
<tbody>
<tr>
<td>Levodopa</td>
<td>Pramipexole (Mirapex)</td>
</tr>
<tr>
<td>o Carbidopa-levodopa (Parcopa)</td>
<td>Rotigotine (Neupro)</td>
</tr>
<tr>
<td></td>
<td>Apomorphine (Apokyn)</td>
</tr>
<tr>
<td></td>
<td>Bromocriptine (Parodel)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Catechol O-methyltransferase (COMT) inhibitors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tolcapone (Tasmar)</td>
</tr>
<tr>
<td>Entacapone (Comtan)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>MAO-B inhibitors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rasagiline (Azilect)</td>
</tr>
<tr>
<td>Selegiline (Eldepryl)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>N-methyl-D-aspartate receptor inhibitor</td>
</tr>
<tr>
<td>o Amantadine</td>
</tr>
<tr>
<td>o Anticholinergic Agents</td>
</tr>
<tr>
<td>o Benztrapine (Cogentin)</td>
</tr>
</tbody>
</table>

---

and the substantia nigra pathology of Parkinson’s Disease. A literature review suggests the increased incidence of melanomas in patients with Parkinson’s Disease, both before and after diagnosis, supports the pathophysiological rather than the pharmacological contribution. However, both Parkinson’s Disease and malignant melanoma incidence increase with age. In the 2 x 2 factorial, randomized, double-blind, placebo-controlled multicenter clinical DATATOP trial, Constantinescu et al. followed 800 Parkinson’s Disease patients for 6 years. They found a higher-than-expected incidence (1.4%) of malignant melanoma compared to a healthy population. No causal relationship between L-Dopa therapy and melanoma was noted. Age was an important factor for both Parkinson’s Disease and melanoma. Consequently, it is expected that over 100 Americans every year will be coincidentally affected by both Parkinson’s Disease and malignant melanoma without a causal relationship between the two diseases.

Ferreira et al. analyzed the frequency of neoplastic and precancerous skin lesions in Parkinson’s Disease patients. Using a Parkinson’s Disease group and a matched control group (150 and 146 patients, respectively), they found Parkinson’s Disease patients had an increased risk of nonmelanoma skin cancer. They supported screening of the skin more frequently in Parkinson’s Disease patients, because a higher prevalence of preneoplastic and neoplastic skin lesions was observed in Parkinson’s Disease patients versus age-matched controls. The researchers hypothesized that nonmelanoma skin cancer’s higher risk in Parkinson’s Disease patients may be due to increased sensitivity to the sun’s role in skin cancer genesis.
be used safely in elderly patients and avoid the well-known “first pass” phenomenon of oral agents.41 Schnitzler et al assessed compliance rates of transdermal once-daily rotigotine patch use in Germany. The majority of the 943 patients studied were compliant with patch use regardless of their clinical state. The downside of use was skin reactions. Adverse skin reactions at the application site (3.7% frequency) led to early withdrawal in 34 patients. The skin reactions ranged from mild to moderate but were severe in one patient.

Skin sensitivity, especially in elders with sensitive and fragile skin, is the most frequent adverse side effect of transdermal delivery systems. A review suggests one approach to minimizing skin sensitivity reaction is varying the site of patch application. Both rotigotine and selegiline were available in transdermal formulations. Watts et al conducted a multicenter, randomized, double-blind study of early Parkinson’s Disease patients (N = 277) comparing rotigotine patch (n = 181) to placebo (n = 96). The results showed that mild to moderate application site reactions occurred in 44% of rotigotine transdermal patch versus 12% of placebo recipients. Skin reactions were so bothersome, the rotigotine patch was removed from the market.42 A recent drug safety evaluation of rotigotine patch was positive, and the transdermal form is still available in Europe.43 Transdermal selegiline is still available but is used to treat major depressive disorder, and is not labeled for Parkinson’s Disease.28

Drug side effects: amantadine. Certain oral anti-parkinsonian drugs are associated with dermatologic conditions. Amantadine, an antiviral drug that releases endogenous dopamine, is used more often in advanced Parkinson’s Disease to mitigate problematic dyskinesias. However, it also can cause skin changes.

Drug rashes can occur from amantadine. The lower extremities may acquire a red and mottled edematous appearance called livedo reticularis (see Figure 1). Livedo reticularis is a reticulated (network) vascular pattern that appears as a lace-like purplish discoloration.45 The exact mechanism of amantadine-induced livedo reticularis is unclear. It is usually asymptomatic and can occur 1 month to 4 years after drug initiation. Most often, the lower extremities are affected, but the trunk and upper extremities can be altered as well. The change usually resolves when the drug is discontinued.16

Skin/Wound Risks in Parkinson’s Disease

As Parkinson’s Disease progresses, immobility and off periods may increase in severity. Difficulty in turning and moving in bed can exacerbate the challenges. The effects of pressure, shear, moisture, and friction on skin microcirculation have been known for decades. In 1994, Schubert and Heraud studied 30 elderly patients and the impact of pressure and shear on skin microcirculation in the sacral area using a laser Doppler. Immobility, especially in the supine position, increased the risk for ischemic skin damage. Parkinson’s Disease immobility can play a great role in increasing risk for pressure ulcers. In addition, urinary/fecal incontinence challenges associated with more long-term Parkinson’s Disease can combine with mobility alterations to increase risk for pressure ulcerations. The role of moisture-associated skin damage (MASD) and incontinence-associated dermatitis (IAD) in augmenting wound risk has been well documented.48-55 MASD in the form of urinary/fecal incontinence affects the normal protective strength of the skin. Once the incontinence becomes IAD, substantive skin breakdown can occur in as little as 2 days.49 Although pressure ulcers need to be differentiated from IAD or MASD because treatments differ,49-51 both MASD/IAD increase the risk for pressure ulceration.

Sialorrhea that persists as drooling can generate MASD. In addition to drug therapy, topical protection such as nonalcohol skin sealants may help prevent breakdown and maceration. Oil-based ointments would not be recommended, because they could potentially be inhaled or aspirated into nearby airways. Parkinson’s Disease patients’ excessive sweating also can generate skin breakdown risk. A potential exists for intertriginous dermatitis, another form of MASD, particularly if hyperhidrosis occurs near or within skin folds. Drying agents such as powder or materials that wick moisture away from the skin fold (eg, InterDry, Coloplast Corp, Minneapolis, MN) may help with management.
Risk for fungal skin infection of the perineal and perirectal area increases when the Parkinson’s Disease patient is incontinent. Commonly, fungal infections of the perigenital area present as a moist, red, itchy rash with classic satellite lesions as the fungal infection spreads across the skin. Antifungal ointment topical therapy (eg, mycostatin ointment) may be needed to control the infection. In addition, drying materials with topical silver can act to wick moisture away plus decrease skin microbial count (InterDry Ag; Coloplast Corp, Minneapolis, MN).

Periwound moisture also may affect the Parkinson’s Disease patient’s skin integrity. As stated previously, Parkinson’s Disease patients have altered autonomic nerve supply, especially as the disease progresses. Chronic wounds such as exudative venous ulcers also could pose a special challenge due to MASD.

Hospitalization and/or critical illness can increase skin and wound risk for the Parkinson’s Disease patient. In a recent retrospective correlational study of predictors of pressure ulcers in critical care, Cox examined 347 critical care patient risk factors for predicting pressure ulcer development. She found altered mobility, older age, length of stay, and cardiovascular disease were the most significant predictors. All may affect the Parkinson’s Disease patient.

Implications for Clinical Practice and Education

Nurses have great opportunity to influence the quality of Parkinson’s Disease patient care by being cognizant of and vigilant for potential skin problems affecting this patient population. The physiologic, pathophysiologic, and disease therapy mechanisms that may affect skin integrity affect nursing care.

Results of a review by Pan et al. support that the skin changes associated with Parkinson’s Disease, especially the risk of malignant melanoma and other nonmelanoma skin cancers, suggest that periodic dermatological surveillance is critical to good care whether in primary, specialist, or long-term care settings. Nurses and other clinicians should carefully inspect the skin of the entire body (including the feet because sun exposure may not be a critical factor in skin cancer pathogenesis). For melanoma, clinicians should look at identified lesions with the melanoma mnemonic ABCDE (asymmetry, irregular border, color variability, diameter >6 mm, enlarging and elevated). The appearance of both basal cell and squamous cell cancers should be noted as well. Suspicious skin lesions should be referred to dermatology.

Sialorrhea, seborrhea, and hyperhidrosis mechanisms should be understood and addressed. Skin protection around the mouth and upper chest may be needed. Commercial products are available for protection, and nurses should contact skin and wound specialists such as wound, ostomy, continence nurses for advice.

To help seborrheic skin and hair, both should be frequently washed. If dermatitis occurs, topical steroids or dandruff shampoos may be necessary. If dermatitis with flakiness develops around the eyes, the use of a dandruff shampoo pyrithione zinc (Head and Shoulders, Proctor & Gamble, Cincinnati, OH) or selenium sulfate (Selsun Blue, Sanofi-Aventis, Bridgewater, NJ), allowed to run over the face (with the eyes closed) may help. Interestingly, both seborrhea and seborrheic dermatitis improve with treatment of levodopa in Parkinson’s Disease.

In addition to pharmacological (dopamine) therapy for hyperhidrosis, other approaches may help. The patient with hyperhidrosis can be taught to take lukewarm showers, wear lighter weight clothing in warm weather, drink extra fluids, use lightweight cotton sheets for bedding, and avoid saunas. Dry skin on the extremities and palms can benefit from moisturizing lotions.

Skin changes related to drug therapy should be in the mind of the clinicians caring for the Parkinson’s Disease patient. Attention to the development of skin rashes, lesion patterns, and areas of irritation is necessary especially if apomorphine or amantadine are being used.

www.o-wm.com
Another noteworthy issue is the change in the protective nature of the integumentary system due to the disease. Research suggests that Parkinson's patients' skin has less sensitivity to heat, cold, and mechanical pain. Other sources suggest altered sensitivity to sunlight. Both require the nurse to educate patients about these risks and good preventative self-care related to burns and cold exposure.

Jaul's review notes that pressure ulcer prevention and treatment involves optimal treatment of irreversible medical conditions. Specifically, rigidity and immobility must be treated promptly with dopaminergic therapy as necessary. The implications for prompt delivery of Parkinson's medications are evident in that they may prevent or ameliorate the motor issues and off periods. Nursing staff and caregivers must understand this relationship and make sure patients experiencing poor symptom control are promptly medicated.

Parkinson's Disease also is associated with urinary tract dysfunction, especially nocturia and urgency. Skin integrity can be at risk with urinary incontinence. Frequent assessment and use of continence aids may be necessary. Barrier ointments in the perineal area can protect against MAD and IAD.

Wound healing processes in long-term Parkinson's Disease patients are an area for future research and nursing vigilance. Wounded Parkinson's patients should be monitored closely for healing progress. A slow-to-heal wound may have other inhibitory processes occurring because of the disease. Attention to known wound healing factors such as nutrition and topical therapy is warranted.

The effects of Parkinson's Disease on the skin have been described in this article. But disease impact is also more profound. Leibson et al.'s epidemiologic study of 197 Parkinson's Disease patients in Minnesota for 5 years before and 15 years after diagnosis supports that Parkinson's Disease patients have greater mortality and morbidity than matched non-Parkinson's Disease patients over time. These disorders include other diseases of the circulatory system, veins, lymphatics, and digestive system. Nurses and other providers need to be attentive for comorbidity development.

Conclusion

Parkinson's Disease affects the skin in some manner across the trajectory of the disease. The nonmotor autonomic changes include ataxia, hyperhidrosis, and seborrhea associated with Parkinson's Disease, malignant skin lesions associated with the disease pathology and/or therapy, and skin and wound care issues with nursing implications. Nurses and other health clinicians can promote optimal care by increasing their understanding of disease impact and the methods by which potential complications can be anticipated and prevented.

Acknowledgment

This article was generated as a result of participation in the Visiting Nurse Faculty Program of the Edmond J. Safra Foundation. The author offers special thanks to all who devised and implemented this wonderful activity for nurse educators.
Dermagraft®

Human Fibroblast-Derived Dermal Substitute Essential Prescribing Information

Numbers in parentheses ( ) refer to sections in the Directions for Use of the product labeling.

Device Description: Dermagraft® is a cryopreserved human fibroblast-dervied dermal substitute. (1)

Intended Use/Indications: Dermagraft® is indicated for use in the treatment of full-thickness diabetic foot ulcers greater than six weeks duration which extend through the dermis, but without tendon, muscle or bone exposure. Dermagraft should be used in conjunction with standard wound care regimens and in patients that have adequate blood supply to the involved foot. (2)

Contraindications:
1. Dermagraft is contraindicated for use in patients that have signs of clinical infection or in ulcers with sinus tracts.
2. Dermagraft is contraindicated in patients with known hypersensitivity to bovine products, as it may contain trace amounts of bovine proteins from the manufacturing medium and storage solution. (3)

Warnings: None (4)

Precautions:
1. Caution: The product must remain frozen at −50°C ± 10°C continually until ready for use.
2. Caution: Do not use any topical agents, cytotoxic cleansing solutions, or medications (e.g., lotions, ointments, creams, or gels) on an ulcer being treated with Dermagraft® as such preparations may cause reduced viability of Dermagraft®.