Cutaneous manifestations of endocrine-metabolic disease
and nutritional deficiency in the elderly

Joseph Brant Schneider, MSIV, Robert A. Norman, DO, MPH, FAAD*

7902 West Waters Avenue, Tampa, FL 33615, USA

This article explores cutaneous manifestations of endocrine-metabolic disease and nutritional deficiency in the elderly. Topics covered include the following: diabetes mellitus (DM), thyroid disorders, adrenal dysfunction, pituitary disorders, parathyroid disease, nutritional deficiencies, menopause, and HIV.

Diabetes mellitus

Diabetes mellitus is the most common endocrine disease in America. It is reaching epidemic proportions in the United States affecting over 16 million people. DM is not always thought of as a disease of the elderly, but estimates have placed the proportion of adults aged 65 to 74 with diagnosed diabetes at nearly 25% in some ethnic groups [1]. It is estimated that an additional 11% of adults ages 60 or older have undiagnosed DM [2]. DM type 1 is an autoimmune disease resulting in destruction of the pancreatic β-islet cells. This results in no endogenous insulin production, and patients rely on multiple insulin injections daily to survive. DM type 2 is caused by target organ insulin resistance and a progressive inability of the pancreas to keep up with the insulin demand of the body. DM type 2 accounts for 90% of the cases of diabetes. Most cases of DM type 2 have a genetic component, and are related closely with obesity. High serum glucose levels characterize both diseases [2]. DM manifests itself as a vascular and neuropathic disease affecting nearly every organ of the body, most notably the kidneys, eyes, and cardio-vascular system. Older persons with DM have higher rates of premature death, functional disability, depression, cognitive impairment, urinary incontinence, injurious falls, persistent pain, and coexisting disease [3]. Often affected, but frequently overlooked, is the resulting dermatologic dysfunction. This is of paramount importance for practitioners involved in the care of the integumentary system. The effects on the skin are not only sequelae of the disease, but can also be clues to aid in the diagnosis of DM, because DM type 2 often goes undiagnosed for many years [4].

Diabetic dermopathy

Diabetic dermopathy is also known as shin spots, and occurs in up to 40% of diabetics. Shin spots are the most common cutaneous manifestation of DM [5]. Diabetic dermopathy may serve as a clinical sign of an increased likelihood of internal complications in diabetic patients, such as retinopathy, nephropathy, and neuropathy [5]. Round to oval circumscribed, atrophic, slightly depressed, hyperpigmented lesions are seen on the anterior aspect of the lower legs, and are generally bilateral and asymmetric. The lesions are generally asymptomatic. The exact cause of this disorder is unknown [6]. The lesions recur in crops and spontaneously resolve eventually healing with scar formation. There is currently no standard of treatment for diabetic dermopathy [7].

Diabetic ulcers

Diabetes is responsible for the largest number of nontraumatic amputations in the United States. For many of these patients, amputation is the eventual result of a nonhealing ulcer [8]. Ulcers arise in dia-
abetes by two mechanisms. The first is diabetic neuropathy, which severely decreases sensation in the periphery. With decreased sensation, patients tend to injure themselves without knowing. DM is the most common cause of diabetic polyneuropathy, which includes motor impairment and sensory impairment including proprioception loss, and puts the patient at an even higher risk of injury [9]. The second pathway of ulcer formation occurs as a consequence of vascular insufficiency. These patients, in contrast to the neuropathic patients, have a higher sensation of pain. The pain is generally disproportionate to their injury and often excruciating. The best management of ulcers in diabetics is prevention. Diabetic patients, especially the elderly, should check their own feet daily and have their feet checked at every physician visit. The nylon monofilament test is a good tool to assess early sensation loss and identify patients at risk [10]. Treatment options include compression, debridement, dressings, strict infection control, growth factors, skin grafts, skin substitutes, and restoration of blood supply [5].

Perforating dermatosis

Acquired perforating dermatosis is a disorder generally seen in diabetics who have progressed to chronic renal failure and dialysis. The lesions are pruritic, hyperkeratotic, and often umbilicated 2- to 10-mm papules or nodules. They are usually located on the extensor surfaces of the legs, but can be seen on the trunk or the face. Often they coalesce into larger plaques, but can exist in rows [5]. The main feature of this process is a transepidermal elimination of collagen and elastin. Lesions can be reproduced by trauma to the area, such as scratching. This condition is difficult to treat, but some success has been seen with the use of topical keratolytics, retinoids, PUVA, UVB, steroids, oral antihistamines, and cryotherapy [10].

Eruptive xanthomas

Eruptive xanthomas are discrete inflammatory papules of lipid deposits formed mainly from cholesterol esters. They are red to yellow in color with an erythematous base and can become confluent in areas representing tuberoeruptive lesions. They appear suddenly in crops, usually on the buttocks and elbows, but can be seen anywhere on the body [11]. These lesions are caused by a hyperlipidemic and hyperglycemic state most commonly secondary to a familial hypertriglyceridermia or insulin deficiency with uncontrolled insulin-dependent DM [12]. Control of the underlying hyperlipidemia or hyperglycemic state results in complete resolution of the lesions.

Necrobiosis lipoidica diabeticorum

Necrobiosis lipoidica is a disorder often associated with diabetes. Although only 0.3% to 1.6% of diabetics acquire this condition, approximately 90% of patients with this disorder develop diabetes, have an abnormal glucose tolerance, or have a strong family history of diabetes [13]. The lesions begin as brownish red or skin-colored papules and evolve into distinctive, sharply circumscribed, multicolored plaques with an elevated border. With time, the lesions become brownish yellow, telangiectatic, porcelain-like, and depressed. Classically, the lesions are seen bilaterally on the anterior and lateral surfaces of the lower legs [8]. The lesions are painless, but they may ulcerate, resulting in pain, especially if they become infected. The etiology is not completely understood, but there is degeneration of collagen in the dermis and subcutaneous fat with an atrophic epidermis and granulomatous dermis [14]. There is no standard of treatment, and strict control of insulin levels seems not to have an effect on the course of this condition [9].

Acanthosis nigricans

Acanthosis nigricans is a disorder in which a diffuse, velvety thickening, and hyperpigmentation of the skin is seen particularly on the neck and axillae, and occasionally the groin, umbilicus, hands, areolae, and submammary areas. Acanthosis nigricans is not exclusive to diabetics, and can be seen in patients with obesity; drug administration (nicotinic acid, corticosteroids); and stomach carcinoma. The physician should rule out these other possible etiologies if seen in a diabetic. The cause is unclear and the only adverse affects seem to be cosmetic. Treatment is generally by correcting the underlying cause, but retinoic acid and salicylic acid may be of benefit [15].

Other conditions secondary to diabetes

A few other dermatologic conditions have been associated with diabetes and should be mentioned. Diabetics have been shown to have thicker skin than their age-controlled counterparts, apparently caused by a change in the dermal collagen [16]. The term “scleredema diabeticorum” is a rare disorder with very tight thickening of the neck and posterior back, occasionally extending to the arms and hands. The pathogenesis has not been clearly defined, and the clinical significance seems to be minimal unless
The thickness compromises joint mobility. There is no treatment for diabetic thick skin, but strict glycemic control may be of some benefit [17]. Poor glycemic control can increase glycosylation of dermal collagen and proteins, resulting in yellow skin resembling carotenemia. The clinical significance is unknown and there is no treatment for this phenomenon [18]. Poorly controlled, long-standing diabetics with peripheral neuropathy are predisposed to blister formation (diabetic bullae). They are generally nonscarring with resolution in a couple of weeks; however, an unusual subtype does heal with atrophy and scarring [19]. Therapy is supportive with a focus on infection prevention. There seems to be a higher incidence of vitiligo in type 1 diabetics. This should be of no surprise because type 1 diabetes is an autoimmune process, and patients tend to have family history of other autoimmune diseases, such as thyroiditis and Graves’ disease [12].

The treatment of diabetes is not without its own consequences. The use of first-generation sulfonylureas has been associated with a maculopapular rash, generalized erythema, and urticarial eruptions. These generally appear during the first month of therapy, and resolve spontaneously with continual therapy. Second-generation sulfonylureas are much more widely used, and seem to cause fewer side effects [20]. Insulin allergy has been reported, and may be from impurities in the insulin preparation, additives, preservatives, or to the insulin molecule itself [21]. A local effect of lipohypertrophy has been reported, and may be caused by the lipogenic action of insulin [22].

**Thyroid disorders**

**Hyperthyroidism**

Graves’ disease is by far the most common cause of endogenous hyperthyroidism [23]. Sawin et al [24] reported a prevalence of 5.9% in a population of patients 60 and older. Graves’ disease is an autoimmune disorder secondary to autoantibodies that bind to the thyroid-stimulating hormone (TSH) receptor in the thyroid cell membranes causing abnormal hyperactivity of the gland and subsequent thyrotoxicosis. Other causes are thyroid replacement therapy; subacute thyroiditis secondary to a viral infection; iodine-induced hyperthyroidism (Jodbasedow disease); struma ovarii; pituitary tumor; and Plummer’s disease (which reportedly does not cause thyroid dermopathy) [25]. Women are affected up to seven times as often as men, occurring in 1.5% to 2% in women in the United States [2]. The clinical manifestations of thyroid disease are frequently subtle and their sign and symptoms are often attributed to normal aging by both the patient and the physician [17]. The most common dermatologic manifestations of hyperthyroidism include warm, moist skin; palmer erythema; fine and friable scalp hair with alopecia in severe cases; and onycholysis [26]. Twenty percent to 40% of patients develop ophthalmopathy characterized by chemosis, periorbital edema, lid lag, and exophthalmos or proptosis. Approximately 33% develop atopic dermatitis, and 7% develop vitiligo. Thyroid dermopathy is known as pretibial myxedema; however, this condition occurs in only 3% of patients. Pretibial myxedema is classified into early and late lesions. Early lesions are bilateral, asymmetric, firm, non-pitting nodules and plaques that are pink, skin-colored, or purple [12]. Late lesions are a confluence of early lesions that symmetrically involve the pretibial regions. Occasionally, these lesions can produce gross disfigurement of the legs appearing as smooth surfaces with orange peel quality [18]. The pathogenesis of pretibial myxedema remains to be defined [27]. Nondermatologic manifestations of hyperthyroidism include weight loss despite increased appetite, heat intolerance, sinus tachycardia and arrhythmias, wide pulse pressure, diarrhea, emotional lability, muscle weakness and fatigue, and fine tremor [2]. Diagnosis is by radioiodine uptake, TSH, total T4, and free T4. Treatment is with antithyroid drugs, radioiodine, and β-blockers. Treatment with triamcinolone has been shown to have a good effect on pretibial myxedema [25]. Prognosis of Cushing’s disease is 90% at 10 years with successful adrenalectomy.

**Hypothyroidism**

Hypothyroidism is caused most commonly by the treatment of Graves’ disease or by chronic autoimmune thyroiditis (known as Hashimoto’s disease if goiter is present). Other causes include alterations in the hypothalamic-pituitary axis resulting in hypopituitarism and congenital chemical defects resulting in decreased thyroid hormone secretion. Iodine deficiency is rare in developed countries, but is common in some areas of the world. Thyroid hormone controls metabolism and a deficiency can affect virtually all body functions. The sine qua non of hypothyroidism is myxedema. This is caused by an accumulation of water-binding mucopolysaccharides in the dermis. The manifestations range from unnoticeably mild to markedly severe. Patients present with dry, rough skin and in severe cases this can simulate ichthyosis vulgaris; occasionally hyperkeratosis develops on the palms and soles (palmoplantar keratoderma). The skin
may appear cool, waxy, coarse, pale, and swollen especially periorbitally because of the increased water content and vasoconstriction. A yellowing of the skin may occur, and is caused by carotenemia [28]. Coarse, brittle hair or diffuse alopecia may be present, with particular thinning of the lateral eyebrows. The facial skin has puffy features with increased skin creases and patients may have a flat, expressionless facies. The tongue is large, red, smooth, and clumsy [12]. Finger-nails become coarse and easily break. Nondermatologic signs and symptoms include weakness, fatigue, cold intolerance, constipation, weight gain, decreased mental function, depression, hypothermia, bradycardia, slow return of deep tendon reflexes, menorrhagia, and hoarseness. Diagnosis is with TSH, T4, and free T4. Patients can live completely normal lives with replacement therapy [2].

Adrenal dysfunction

Cushing’s syndrome

Cushing’s syndrome refers to the manifestations of increased cortisol secretion by the adrenal glands for any reason. Cushing’s disease, which accounts for 43% of Cushing’s syndrome, is hypercortisolism caused by adrenocorticotropic hormone (ACTH) hypersecretion by the pituitary. Neoplasms in the pituitary, adrenals, and small cell lung cancer account for 85% of these cases. Hypertension and weight gain are early manifestations. The classic cutaneous manifestations of Cushing’s syndrome include a redistribution of body fat with central obesity, “buffalo hump,” moon facies, and thin arms [11]. These findings occur slowly and may be quite subtle at first. Purple stria is particularly common on the torso and is believed to be caused by an increase in melanocyte-stimulating hormone [29]. Patients have atrophic skin, with easy bruisibility, and telangiectasia secondary to the catabolic effect of glucocorticoids on the muscles and perivascular supporting tissue [6]. These effects may be dismissed initially as being secondary to normal aging of the skin, or steroid medications commonly used for a variety of disorders, such as chronic obstructive pulmonary disease. Pigmented facial hypertrichosis, general increase in lanugo, and androgenic alopecia may be seen. Patients may have poor wound healing and an increase in superficial skin infections secondary to the immune suppression of corticosteroids. Some nondermatologic manifestations include psychologic changes, osteoporosis, proximal muscle weakness, hypertension, hyperglycemia, glycosuria, leukocytosis, lymphocytopenia, and hypokalemia. Diagnosis can be made by plasma cortisol levels (morning and evening); 24-hour cortisol levels; and dexamethasone suppression test [2]. Prognosis varies widely depending on cause, and treatment is generally surgical.

Addison’s disease

Addison’s disease is chronic adrenocortical insufficiency. Autoimmune destruction of the adrenals accounts for over 80% of the cases in the United States. Adult onset, or type 2 polyglandular autoimmune syndrome, is associated with autoimmune thyroid disease, vitiligo, type 1 diabetes mellitus, alopecia areata, and celiac sprue. Tuberculosis is no longer a major cause of Addison’s disease in the United States, but in countries where tuberculosis is more prevalent, this is still a concern. Metastatic neoplasms are another potential cause of adrenal insufficiency. The most striking dermatologic sign is increased skin pigmentation especially in the creases, pressure areas, new scars, and nipples [30]. Patients may present simply with a complaint of “getting darker.” ACTH stimulates melanocytes and is secreted by feedback mechanisms in the hypothalamus. Without the negative feedback from the adrenals, ACTH is secreted in relatively large amounts causing hyperpigmentation. Hyperpigmentation is not seen in primary pituitary or hypothalamus disease. Although these skin signs alone are not specific for Addison’s disease, the combination of skin and mucosal pigmentation with gut disturbances and weight loss has been found to have a high predictive value in the diagnosis of Addison’s disease [31]. The most common nondermatologic signs and symptoms include slow-onset weakness, easy fatigability, anorexia and weight loss, nausea and vomiting, abdominal pain, and amenorrhea. Acute stressors, such as infections, surgery, or trauma, may precipitate an acute adrenal crisis, which is potentially life threatening; skin infections should be treated rapidly and vigorously. Low plasma cortisol levels (< 5 mg/dL) are diagnostic. Prognosis is good with replacement therapy.

Pituitary disorders

Hypopituitarism

Pituitary insufficiency is most commonly secondary to postpartum hemorrhage, known as Sheehan’s syndrome, or to ablation of the gland by surgery or
cancer radiation. Secondary hypopituitarism is caused by disorders of the hypothalamus. Idiopathic hypopituitarism is the least common cause. Hypofunction of the anterior pituitary does not occur until 75% of the parenchyma is lost or absent [32]. The pituitary gland’s vital hormones are ACTH, TSH, follicle-stimulating hormone, growth hormone (GH), and prolactin.

The most common cutaneous manifestations of hypopituitarism are pallor and decreased ability to tan secondary to decrease in melanin-stimulating factor from the pituitary. The texture of the skin is smooth and course with dryness and scaling. Fine wrinkling of the face and forehead is said to be characteristic. Others have described the skin as smooth and rubbery similar to that of an infant [25]. Other signs and symptoms may be specific to the individual hormones that are lacking and whether their deficiency is complete or partial. The most common manifestations of hypopituitarism are sexual dysfunction; easy fatigability; and lack of resistance to stress, cold, and fasting. Diagnosis is by specific hormone levels, MRI of the brain, and provocative tests [2]. Treatment is with replacement of individual hormones, and prognosis varies widely according to etiology.

Hyperpituitarism

Hyperpituitarism is generally secondary to a pituitary tumor resulting in secretion of one specific hormone. Rarely is it caused by hyperfunctioning hypothalamus. GH and ACTH are the usual hormones affected. Excess ACTH was covered in the section regarding Cushing’s syndrome. This section focuses on increased GH secretion. In children, increased GH results in gigantism, and acromegaly in adults. GH increases the mass of all internal viscera and the bones and skin. The skin thickens and feels smooth or rubbery because of an increase in the dermal collagen, and becomes sweaty or oily presumably from direct GH stimulation of the sweat glands. Furrows and ridges are seen about the face, neck, and scalp and are referred to as “cutis vertices gyrata.” Hyperelasticity of the skin resembling Ehlers-Danlos syndrome has been reported [6]. Approximately 50% of patients have increased body hair, and 40% of patients have hyperpigmentation. Nondermatologic manifestations include deepening of the voice, headaches, amenorrhea, visual field loss, and weakness. Diagnosis is by glucose challenge followed by GH measurement 1 hour later. MRI shows a pituitary tumor in 90% of acromegalics. Treatment is by surgery, and prognosis is generally good.

Parathyroid disease

Hyperparathyroidism

Hyperparathyroidism is divided into primary and secondary. Primary hyperparathyroidism is generally diagnosed in infancy and not seen in the geriatric population. Secondary hyperparathyroidism is most commonly caused by chronic renal failure or parathyroid adenomas [33]. No cutaneous lesions are seen in hyperparathyroidism caused by adenomas, but two external manifestations caused by subsequent hypercalcemia can suggest the diagnosis and are worth mentioning. White lines on the lateral margins of the cornea are called band keratopathy. Bone cysts especially in the mandible can grow quite large and produce subcutaneous tumors, which can be seen readily and palpated by an examiner. Neither of these manifestations is specific to hyperparathyroidism, so a high level of suspicion is needed. Chronic renal failure increases serum phosphate levels causing the kidneys to excrete calcium along with the phosphate-inducing hyperparathyroid secretion to replace the lost calcium [29]. This results in bone resorption and cyst formation. Grossly visible nodules can develop from metastatic calcifications in the dermis and subcutaneous tissue. Calcification of vessels may occur causing gangrene of the fingers and toes. Cutaneous infarction of the lower abdomen and legs may also be seen. Treatment is with large fluid intake and bisphosphonates in mild cases, and surgically in severe cases [2]. Most adenomas that are surgically removed result in complete cure, but there can be unexplained exacerbations and partial remissions.

Hypoparathyroidism

Hypoparathyroidism is seen most commonly in the geriatric community secondary to inadvertent removal of the parathyroid glands during thyroid surgery or radical neck dissection for cancer [34]. Other causes are congenital or idiopathic, usually occurring in infancy, but may appear in the 30s or 40s.

Postthyroidectomy syndrome is seen following inadvertent removal of the parathyroid glands during thyroidectomy. Patients have attacks of hypocalcemia and tetany. Thinning of the hair or complete alopecia may be seen in association with these episodes. The nails commonly develop Beau’s lines, horizontal grooves on the nails that appear at the base of the nails approximately 3 weeks following a tetracycline attack. An interruption of keratinization is suggested to be the cause of these manifestations. The skin becomes dry and scaly. Enamel defects and hypopla-
sia of the teeth may be mistaken for poor dental hygiene especially in debilitated seniors. Generalized skin eruptions have been reported. Cataracts develop in approximately 50% of patients with chronic hypocalcemia. Other nondermatologic signs and symptoms include muscle cramps; irritability; carpopedal spasm; convulsions; and tingling of the hands, feet, and circumoral area [2]. Chronic disease manifests as lethargy, personality changes, anxiety, visual blurring caused by cataracts, Parkinson’s disease, and mental retardation. Chvostek’s sign and Trousseau’s phenomenon may be seen. Diagnosis is generally suspected from history of neck surgery, and confirmed with low parathyroid hormone levels. Treatment is with calcium, vitamin D, and magnesium replacement. Transplantation of cryopreserved parathyroid tissue may restore normal calcium levels [35]. Prognosis is good if diagnosis is made, and treatment instituted early.

Cutaneous manifestations of nutritional deficiencies in the elderly

Vitamin and nutritional deficiencies are much too common in the elderly population. It has been estimated that as many as 55% of hospitalized elderly and up to 85% of institutionalized elderly persons are undernourished. As many as 50% have a vitamin and mineral intake less than the recommended dietary allowance and as many as 30% of the elderly population have subnormal levels of vitamins and minerals [36]. In this author’s opinion, this is a truly tragic problem. By and large, nutritional food is abundant in this country. Many foods are fortified with vitamins and minerals, and supplements in pill, liquid, and powder form are readily available and inexpensive. Monitoring weight loss and cutaneous lesions, such as the presence of decubitus (grade II or higher), is a quick, cheap, and convenient way of detecting poor nutrition and is of prognostic value in older persons [37,38].

Vitamin C deficiency

Scurvy is a disease caused by a deficiency in ascorbic acid (vitamin C). Most cases in the United States are seen in the urban poor, elderly, and alcoholics. Patients with chronic disease, such as cancer or chronic renal failure, are also at risk. Vitamin C cannot be synthesized endogenously; humans are dependent on intake with food. Vitamin C is found in an abundance of foods, and only severely restrictive diets do not provide adequate amounts. The most clearly established function of ascorbic acid is the activation of prolyl and lysyl hydroxylase from inactive precursors, providing for hydroxylation of procollagen [2,23]. Collagen is inadequately cross-linked, and is poorly secreted from fibroblasts. Vessel walls with poor collagen support are pliable and rupture easily. Scurvy manifests as a vascular disease in which hemorrhages and poor healing cause significant morbidity. Early manifestations are nonspecific, like malaise and weakness. Perifollicular hemorrhages, petechiae, gingival bleeding, and splinter hemorrhages are the most striking cutaneous manifestations. Hemorrhages and subperiosteal hemorrhages may also be seen. Anemia is common, and wound healing is impaired. Diagnosis can be made clinically on findings of skin lesions and suggestive history. Adult scurvy can be treated with 300 to 1000 mg of ascorbic acid per day, and improvement is generally seen in less than a week.

Niacin deficiency

Pellagra results from a deficiency of the B vitamins, most notably B3 (niacin). It can also be caused by a tryptophan deficiency, which is a precursor of nicotinic acid. Niacin and its products (NAD, NADP) are essential components of many redox reactions and general cellular metabolism. Most patients with a niacin deficiency are alcoholics or chronic disease sufferers. It may also be seen in patients with chronic diarrhea, diets grossly lacking protein, and long-term use of isoniazid or 6-mercaptopurine. Pellagra may also be seen in populations who receive most of their calories from corn, in which the niacin is highly bound and unavailable for use by the human body [25]. The classic clinical triad of pellagra (dermatitis, dementia, and diarrhea) is seen only in advanced cases. The initial manifestations are very nonspecific and include anorexia, weakness, irritability, weight loss, mouth soreness, glossitis, and stomatitis. The dermatitis is bilateral, symmetric and is seen in sun-exposed areas secondary to photosensitivity [11]. The disorder begins with symmetric itching and painful erythema on the back, neck (Casal’s necklace), face, and dorsum of the hands (gauntlet of pellagra). Vesicles and bullae may erupt leaving crusted, scaly lesions. Eventually the skin becomes indurated, lichenified, rough, and covered by dark scales and crusts. There are cracks and fissures and affected areas are sharply demarcated from normal skin [12]. Diagnosis can be made by detection of decreased metabolites in the urine, or more commonly by clinical presentation and response to replacement therapy.
Prognosis is generally good, and complete resolution is seen with proper treatment.

Zinc deficiency

Zinc deficiency in children is an autosomal-recessive genetic disorder of absorption resulting in a condition called acrodermatitis enteropathica. In adults and the elderly, the manifestations are identical to acrodermatitis enteropathica and can be caused by alcoholism; malignancies; chronic kidney disease; certain drugs (penicillamine, diuretics); and long duration of total parental nutrition therapy. Because these conditions occur more often in the elderly population, they are at a higher risk for zinc deficiency. The pathogenesis of the cutaneous effects is unclear, but a defect in conversion of linoleic acid to arachidonic acid best explains the manifestations [39]. Lesions initially appear in the perioral and anogenital areas, with later involvement of the scalp, hands, feet, flexural regions, and trunk. Lesions are noted as patches and plaques of dry, scaly, sharply defined, and deep red, eczematos dermatitis evolving into vesicobullous, pustular, erosive, and crusted lesions [11]. Poor wound healing is often noted. Hyperkeratosis, parakeratosis, perleche, paronychia, nail dystrophy, and alopecia may also be seen. Sequelae from immunologic impairment may be seen as parasite, fungal, or bacterial superinfections [3]. Nondermatologic manifestations include photophobia and an irritable and depressed mood.

Other vitamin deficiencies

A few other vitamin deficiencies may result in cutaneous lesions [36,40], and are reviewed briefly as follows:

- Vitamin E deficiency has been associated with poor wound healing and decrease in immune function.
- Vitamin D deficiency can result in calcium disturbances discussed previously in the section on parathyroid dysfunction.
- Vitamin A has been associated with dry skin, follicular hyperkeratosis, and possibly poor wound healing.
- Vitamin K deficiency is associated with disorders of the coagulation cascade and may manifest as increased tendency of bleeding.
- Vitamin B2 (riboflavin) has been associated with seborrheic dermatitis; cheilosis (red, fissured lips); magenta-colored glossitis; and inflammation of the oral mucosa.
- Vitamin B12 may exacerbate peripheral neuropathy increasing the incidence of trauma.

Effect of menopause on the skin

As the baby boomers approach retirement age, the focus is more and more on the health and well-being of the largest segment of the population. Over half of this demographic are women. The average life expectancy of women in the United States is 81 years; this means that many women will live over a third of their lives in a postmenopausal state. When one thinks about estrogen and menopause, the focus tends to be on the issues recently covered by the Women’s Health Initiative [41]: cardiovascular health, bone density, cancer risk, and symptom reduction. Often forgotten is the enormous effect of estrogen on the integumentary system. Problems postmenopausal women encounter include atrophy, dryness, pruritus, loss of resilience and pliability, dry hair, alopecia, and easily traumatized skin [42]. The skin of the postmenopausal woman is thinner and less lubricated than her premenopausal counterpart increasing the risk for ulceration and infection, including HIV transmission [43]. A decrease in the amount of type I collagen is a well-documented reason for this [44,45]. This decrease correlates not with chronologic age, but with postmenopausal age, and the effects can be reversed with oral estrogen replacement therapy [46]. Cutaneous wound healing is impaired in the elderly, especially the postmenopausal woman, and this too is reversible with oral estrogen replacement [47]. Sebum levels fall in the aging person, but it has been shown to decrease more in women [48]. Decreased sebum production is associated with dry skin and can exacerbate pruritus. It has been postulated that altered hormone levels in aged individuals is responsible for a sebaceous gland hypertrophy, which in turn is responsible for decreased sebum production. One group of researchers found that the sebum follicular pore size was markedly increased, and that sebum production was markedly decreased in non–estrogen supplemented women compared with women on hormone replacement therapy. Interestingly, hormone replacement therapy did mitigate the process of progressive enlargement of the follicular pore size; however, there was no significant difference in sebum excretion rate [49].

Topical estrogen supplementation has been shown to be of benefit. Studies evaluating estrogen creams have shown clinical improvement in skin elasticity,
firmness, and moisturization [50]. Other studies have found increased skin collagen content and less wrinkling with topical estrogen use [51]. Unfortunately, these studies were relatively small and used different types and dosages of estrogens. Also, no literature was found comparing the efficacy, safety, and cost effectiveness of topical versus systemic estrogens. For the many women who were previously taking systemic hormone replacement therapy only for the purported cardiovascular benefits, topical estrogen supplementation may be a viable alternative.

Considerations of HIV and the elderly

HIV is not thought of as a problem of the elderly. But in fact, people in their 50s, 60s, and beyond do have sex, do use intravenous drugs, and do contract HIV. According to the Centers for Disease Control and Prevention, 10% of all new AIDS cases reported in 1996 occurred in people older than age 50 [52]. With the advent of new antiretroviral drug regimens there will be seen a phenomenon never dreamed of in the early 1990s: managed HIV patients growing old eventually to die from unrelated causes.

Early HIV symptoms are nonspecific and may be attributed falsely to another disease or to normal aging [53]. Without a high index of suspicion, many physicians do not test older patients for HIV [54]. This may delay diagnosis until a later stage of the disease. Many cutaneous lesions are highly suspicious of HIV infection, and commonly seen lesions in nonimmunocompromised individuals present differently in the HIV-positive population.

References

[31] Zargar AH, Laway BA, Masoodi SR, Bashir MI, Wani AI, Salahuddin M. A critical evaluation of signs


