Pressure Ulcers in the Elderly

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Faculty Disclosures

None.
- Thinning of the epidermis
- Flattening of the dermal-epidermal junction
- Disorganization of collagen and elastin
Changes in Aging Skin

INTRINSIC vs EXTRINSIC causes

Both have profound genetic & ethnic differences
Intrinsic Changes of Aging Skin (I)

- Increased Reactive 02 Species (ROS), decreased antioxidative capacity
- Increased matrix metalloproteases (MMPs)
- Decreased Langerhans Cells, aberrant function of T, B Cells [immunosenescence]
- Decreased melanocytes
- Flattening of the dermal-epidermal junction (rete ridges)
- Reduced keratinocyte proliferation and turnover time
• Dermis: reduced fibroblasts, macrophages and mast cells
• Reduced vascularity
• Loss of Extracellular Matrix (ECM) components: collagen and glycosaminoglycans
• Collagen: increased type III, decreased type I, altered morphology with higher degree of disorganization
• Decreased amount & morphology of elastin
• Surface pH: epidermis less acidic, affects enzymes processing surface lipids
- Diminished sensation to light touch and pressure (Meissner & Pacini corpuscles)
- Reduced sebum secretion
- Decreased ability to produce Vitamin D3
- Decreased pilosebacious units, sweat glands and subcutaneous fat
- Reduced stratum corneum lipid biosynthesis
- Advanced glycation end products (AGE’s) and increased fibroblast death (apoptosis)
Extrinsic Changes of Aging Skin (I)

- Environmental insults through oxidative stress
- Generation of free radicals and reactive oxygen species (ROS)
- ROS stimulates the lipid peroxidation reaction cascade and the release of pro-inflammatory mediators
- Most important: UV radiation, Cigarette Smoke, Ozone (O3), Airborne particulate matter
Extrinsic Changes: Photo Aging

• Decreased atmospheric O_{3} has increased UV exposure (UV-A worse than UV-B)
• Directly initiates photochemical generation of ROS overwhelming natural anti-oxidant defenses
• Mitochondrial DNA mutations lead to dysfunctional oxidative phosphorylation
• Shortens telomeres, leads to apoptosis, senescence, cell cycle arrest
• Increased MMPs not compensated by inhibitors
Cigarette Smoke (CS) has over 4,000 chemicals including pro-oxidants, free radicals, and nitric oxide.

Directly induces oxidative stress and other adverse chemical reactions.

Ozone (O₃) is a gaseous oxidant that also directly induces oxidative stress, decreases antioxidants such as Vitamin C, E, and Glutathione (GSH).

Polycyclic aromatic hydrocarbons (PAHs) adsorbed to airborne particulate matter (PM) may activate xenobiotic metabolism and induce ROS and MMPs.
Comorbidities that Impact Skin (I)

- Altered nutritional status
- Altered hormone levels (Estrogen, Testosterone, GH)
- Anemia
- Atherosclerosis, decreased perfusion
- Venous insufficiency
- Diabetes with microvascular and neurologic changes
- Any source of edema: CHF, Venous stasis, hypoalbuminemia
Comorbidities that Impact Skin (II)

• Any source of hypoxia: COPD, OSA, etc.
• Low output state: CHF, shock
• Incontinence with Moisture Associated Skin Damage (MASD)
• Colonization of skin with fungus and pathogenic, multiple resistant bacteria
• Pharmacologic compromise: corticosteroids, immunomodulators
• Obesity, lymphedema
Cumulative Result of Comorbidities & Age

• Xerosis (dry skin), pruritis
• Decreased reserve: Homeostenosis, affects thermoregulation and H2O balance
• More susceptible to injury including shear forces, ischemia, pressure related trauma, maceration
• More susceptible to infection
• Prolonged wound healing
Mgmt of Geriatric Patients with PrU

- Clinical assessment of “at risk” status
- Offloading: repositioning and surfaces
- Maintain awareness of devices
- Atraumatic wound dressings
- HOB elevation: consideration of priorities (i.e. ventilators and TFs require >30 degrees but PU prevention requires <30 degrees)
- Document your wounds and interventions!
- Consider palliative care principles