Current Management of Acute Cutaneous Wounds
Evaluation of Non-Visible Hematuria in Primary Care
Eosinophilic Esophagitis: Asthma of the Esophagus!

Fact File
Procedure in Practice
News from Internet
April 1, 2009

Dear Doctor,

Every year our heart fills with joy when the auspicious moment of announcing the new volume of Medical Newsletter arrives. This is the moment to thank you for strengthening the bondage of love, reliability and trust between us. Your faith is our inspiration to proceed closer to perfection. Cutaneous wound is something which people have to deal more or less in their every day life. Although some conditions are life threatening, they can be handled quite perfectly if the remedies are well informed. For this purpose, we have discussed 'Current Management of Acute Cutaneous Wounds' in this issue. Our desire is to update you with the latest management options for cutaneous wounds.

With the advancement of Medical Science, the health professionals have now managed to identify several new forms of diseases and their treatment strategies. One such type of disease is eosinophilic esophagitis. This disease condition is seldom discussed in our community. Therefore, many may remain in the dark about its diagnosis and management. We have highlighted 'Eosinophilic Esophagitis' this time. Non-visible hematuria is a disease condition that bothers the physicians quite often. Sometimes this may become difficult to diagnose resulting in delay in treatment. In order to enlighten this topic we have discussed 'Evaluation of Non-Visible Hematuria in Primary Care'. This may help to understand the nature of this condition and its latest available management options.

In 'Procedure and Practice' we have described the 'Placement of a Femoral Venous Catheter'- their usefulness for certain treatments. We hope that this will provide you with well equipped information and will help to enhance further sophistication in your treatment procedures. Tobacco smoking has emerged as the modern epidemic of human world. Although seen as a harmless practice, this is one of the major causes of several deadly diseases like cancer as well as some like asthma and COPD which may completely hamper the daily life of a person. It is the duty of every health related authority to create awareness specially among the youth to abandon cigarette smoking in order to create a healthy and active society of tomorrow’s world. This concept has encouraged us to highlight this habit as lethal in our community also. That is why we have highlighted hazards of tobacco smoking in our 'Fact File'. 'News from Internet' has also brought the latest ongoing research news with their outcomes.

We thank again for your support and wish you ‘Shuvo Noboborsho’.

With regards,

Prof. Farida Huq
MBBS, M.Phil, FCPS, Ph.D.(London)
Medical Director
Beximco Pharmaceuticals Ltd.

Dr. Selina Akhtar
Senior Manager
Medical Department
Beximco Pharmaceuticals Ltd.
The primary function of the skin is to serve as a protective barrier against the environment. Loss of integrity of large portions of the skin as a result of injury or illness may lead to major disability or even death. Every year, in the United States more than 1.25 million people have burns and 6.5 million have chronic skin ulcers caused by pressure, venous stasis or diabetes mellitus. In 2005, 11.8 million wounds were treated in emergency departments in the United States. More than half a million burns and 7.3 million lacerations are treated annually and wounds caused by cutting or piercing instruments are responsible for an additional 2 million outpatient visits each year.

The primary goals of treatment of wounds are rapid wound closure and a functional and aesthetically satisfactory scar. Recent advances in cellular and molecular biology have greatly expanded the understanding of the biological processes involved in wound repair and tissue regeneration. At the same time several technological improvements in acute wound healing give the physicians a promising outcome not only in successful wound closure with satisfactory scar but also in preventing infection and further trauma as well as by providing an environment that optimizes healing of the wound. This article describes the various recent approaches in the management of acute cutaneous wound.

**General Principles of Care**

All wounds should be thoroughly cleansed with tap water or normal saline. For heavily contaminated wounds, high-pressure irrigation can be achieved with the use of a 10 to 50 ml syringe and splatter shield. The patient’s tetanus-immunization status should be ascertained, and standard recommendations followed to ensure that the patient is protected against tetanus.

A moist environment for the wound accelerates healing by preventing cellular dehydration and stimulating collagen synthesis and angiogenesis, thus improving cosmesis and reducing pain, the risk of infection, and the costs of care. A moist environment may be created by covering the wound with a topical antimicrobial agent or by applying an occlusive dressing that reduces the loss of fluid through evaporation. Topical antimicrobial agents have been shown to reduce rates of infection of traumatic lacerations but not in cases of wounds caused by elective surgery.

Occlusive dressings have also shown to reduce rates of infection. Cyanoacrylate liquid bandages are effective for clean, simple wounds. The choice of dressing depends on the cause, size, depth, location, degree of exudation, and level of contamination of the wound, as well as on cost. There is no clinically directive evidence to support the choice of one dressing over another. Occlusive dressings are less painful and more convenient for patients and may speed healing, although they are more expensive than topical antibiotics and gauze dressings. Wet dressings that promote maceration of the tissue and proliferation of bacteria should be avoided. Prophylactic systemic antibiotics should not be used routinely.

**Abrasions**

Abrasions are skin scrapes that do not fully penetrate the epidermis. These are evaluated, cleansed, and debrided like it is done in case of lacerations. Abrasions are harder to anesthetize, which is particularly problematic when large amount of dirt, stones, or glass are embedded as this occurs frequently. Regional block or intravenous sedation may therefore be needed. After the wound has been irrigated and foreign bodies have been removed, abrasions that are limited to the superficial dermis should be treated with a topical antibiotic or an occlusive dressing.

Deep abrasions that extend below the dermis (especially if they have a surface area larger than 1cm² or involve underlying structures) and those that have not healed in 2 weeks may require more advanced care like grafting, and consultation with a plastic surgeon should be considered.
Natural Biological Process of Wound Healing

Wound healing is a dynamic, interactive process involving soluble mediators, blood cells, extracellular matrix, and parenchymal cells. The complete healing of a wound has three phases— inflammation, tissue formation, and tissue remodeling which overlap in time.
Post-traumatic Tattooing: Post-traumatic tattooing is abnormal skin pigmentation due to embedded foreign particles. If particles embedded in the injured skin are not removed, post-traumatic tattooing develops. This condition is most commonly seen with injuries from explosions or fireworks and also in 'road rash'- an abrasion of the skin from contact with a surface containing asphalt, tar or dirt, with embedding of these particles. Initial treatment consists of the meticulous removal of all particles with standard surgical scrub brushes.

During this procedure, a topical lidocaine, local infiltrative anesthetics, or regional anesthetics should be used for abrasions of small to moderate size, and systemic opioids or procedural sedation for extensive abrasions. Removal of particles within 24 hours after injury has been shown to have the best cosmetic results. Treatment of established post-traumatic tattooing has had disappointing results, and requires referral to a plastic surgeon.

Lacerations

Skin Tears: Skin tears are particularly common among patients who are receiving long-term corticosteroid therapy and among the elderly, who tend to have fragile skin. Frail skin presents a clinical challenge for the practitioner, as even the simplest movement can result in damage of the skin. According to a retrospective study, skin tears occur most often in the upper extremities. Although nearly 80% of such laceration occur on hand and arms, this may sometimes appear on the back and buttocks often mistaken as pressure ulcer.

For category I tears (without tissue loss), the wound edges can be approximated with surgical tapes, and the area covered with a non-adherent dressing. In one study, the healing rate of skin tears with the use of this treatment was 66%, as compared with 33% with the use of a thin-film dressing. Category II skin tears (partial tissue loss) and category III skin tears (complete tissue loss) can be managed with one of a number of absorbent dressings such as petroleum-based gauzes, hydrogels, foams, hydrocolloids, nylon-impregnated gauzes, and silicone-coated dressings. These dressings usually remain in place for 5-7 days and are covered with a secondary absorbent gauze dressing that can be changed daily as needed. Elastic tubular nets should be used to support the underlying dressings. Skin tears of all types that are treated within 8 hours after injury can also be closed with cyanoacrylate-based topical adhesive.

Plantar Puncture Wounds: The rate of superficial infection (cellulitis) ranges from 2-10% among patients who present to the emergency department with planter puncture wounds. Most of the infections are caused by *Staphylococcus aureus* or *Streptococcus pyogenes*. The incidence of osteomyelitis, chondritis, and septic arthritis is considerably lower. Puncture wounds in patients who were wearing tennis shoes that were saturated with sweat at the time of injury may be associated with pseudomonas osteomyelitis. A prospective observational study of 63 adults who presented to the emergency department within 24 hours after receiving a plantar puncture wound suggests that cleansing alone may be adequate therapy. If this approach is used, close follow-up is recommended, and antibiotics should be administered immediately in patients who have signs and symptoms of infection.

If the presence of a foreign body is suspected, computed tomographic imaging or ultrasonography should be used to detect non-radiopaque objects. Some studies suggest that deep wounds, especially those occurring over the forefoot, have an increased likelihood of infection, and patients with such wounds should receive prophylactic antibiotics. Since most infections are caused by streptococcus or staphylococcus, or occasionally pseudomonas species, antibacterial agents that target these species should be used. (e.g. dicloxacillin and ciprofloxacin). Frequent cleansing of the puncture wound and application of a topical antibiotic are also indicated.

Mammalian Bites: The risk of infection after dog, cat, and human bites ranges from 3-18% for dog bites to 28-80% for cat bites. Whereas most cat bites are deep puncture wounds, many dog bites cause open lacerations. Large observational studies and limited clinical trials suggest that after high-pressure irrigation of the wound, it is safe to close most bite wounds (even on the extremities) up to 12 hours after injury (healing by primary intention). Puncture wounds and scratches should be allowed to heal by secondary intention. These wounds should be covered with a topical antimicrobial agent and an absorbent dressing. Close follow-up and daily changes of the dressings are required.

For large, heavily contaminated lacerations, delayed primary closure, after an observation period of 3-5 days healing by tertiary intention may be considered. Human bites that are sustained over the metacarpophalangeal joints (clenched-fist bites) are
particularly prone to infection. These bites require aggressive irrigation and treatment with antibiotics (e.g., amoxicillin-clavulanate) and should not be closed. This commonly occurs as a result of a person punching another person in the mouth and hitting a tooth. The tooth may lacerate the extensor tendon and joint capsule in the hand, inoculating the joint with saliva. As patients may be reluctant to disclose the circumstances resulting in such injury, it should always be suspected when patients present with lacerations that are located over the metacarpophalangeal joints. These injuries generally require specialized consultation. Although prophylactic antibiotics are widely used for mammalian bites, a systemic review has demonstrated significant reductions in the rates of infection only in cases of bites to the hands and in human bites.

Subungual Hematomas: A subungual hematoma is a painful condition that results from a collection of blood under the fingernail. Previously, hematomas involving more than 50% of the nail bed were recommended for removal of the nail by the physicians. This is because the incidence of underlying lacerations was found to be quite high especially in association with underlying tuft fractures. However, in a study involving 45 patients who presented to the emergency room with subungual
hematomas, simple nail trephination resulted in healing without any nail deformities or other complications in all the patients. Thus, simple nail trephination with the use of a handheld portable cautery is recommended for most subungual hematomas. Nail removal should probably be reserved for subungual hematomas that are associated with disruption of the nail or surrounding nail folds.

Burns

Burns are dynamic injuries that may progress over the first 2-3 days. Therefore, frequent reassessment of the wound is required to ensure optimal management.

Managing Burns According to Their Classifications: First-degree burns are limited to the epidermis and are erythematous and painful. They generally heal within several days. Second-degree burns involve all of the epidermis and part of the underlying dermis and are classified according to the depth of dermal involvement. Superficial second-degree burns usually heal within 2 weeks, with minimal scarring. Deep second-degree burns are often difficult to distinguish from third-degree, or full-thickness burns.

Deep burns are characterized by hemorrhagic blisters and are covered with layer of white or red injured dermis that does not blanch. These burns usually does not heal for at least 3 weeks and often result in hypertrophic scarring and contractures, especially in children. It is very important to distinguish between superficial second-degree burns and deep burns (deep partial-thickness and full-thickness burns).

Full-thickness burns may be dark brown or tan and have a leathery texture that is insensitive to touch. Circumferential burns (burns that completely encircle a limb, the neck or the torso) can compromise perfusion, and it may be necessary to relieve the pressure by means of an escharotomy, in which an incision is made over the lateral and medial aspect of the involved areas down to the subcutaneous tissue. The depth of the burn is often difficult to assess immediately after injury and is often underestimated. The true depth becomes more obvious with time, therefore, careful surveillance of the wound and reassessment of the treatment are necessary.

The size of a burn is described by an estimation of the percentage of the total body-surface area that has sustained second- or third-degree burns. Errors in estimating the size of the burn, often results in overestimation. Cooling of Burns: Cooling of burns with the use of cold (15-25°C) tap water within 30 minutes after injury has been shown to reduce the pain, the depth and extent of the injury, the need for surgical excision of the burn, scarring and mortality. Cooling of burns should continue until the pain is substantially reduced or resolved. The use of ice or ice water may increase tissue injury and should be avoided. Concern that cooling of large burns may result in hypothermia has been based on a single study in small animals with extensive burns.

Management of Blisters: The debate regarding the removal of burn blisters has been confusing due to conflicting data regarding the in vitro effects of blister fluid. Two clinical trials involving patients and volunteers with superficial burns demonstrated that intact blisters healed faster and were less likely to

---

**Burns**

Burns are dynamic injuries that may progress over the first 2-3 days. Therefore, frequent reassessment of the wound is required to ensure optimal management.

Managing Burns According to Their Classifications: First-degree burns are limited to the epidermis and are erythematous and painful. They generally heal within several days. Second-degree burns involve all of the epidermis and part of the underlying dermis and are classified according to the depth of dermal involvement. Superficial second-degree burns usually heal within 2 weeks, with minimal scarring. Deep second-degree burns are often difficult to distinguish from third-degree, or full-thickness burns.

Deep burns are characterized by hemorrhagic blisters and are covered with layer of white or red injured dermis that does not blanch. These burns usually does not heal for at least 3 weeks and often result in hypertrophic scarring and contractures, especially in children. It is very important to distinguish between superficial second-degree burns and deep burns (deep partial-thickness and full-thickness burns).

Full-thickness burns may be dark brown or tan and have a leathery texture that is insensitive to touch. Circumferential burns (burns that completely encircle a limb, the neck or the torso) can compromise perfusion, and it may be necessary to relieve the pressure by means of an escharotomy, in which an incision is made over the lateral and medial aspect of the involved areas down to the subcutaneous tissue. The depth of the burn is often difficult to assess immediately after injury and is often underestimated. The true depth becomes more obvious with time, therefore, careful surveillance of the wound and reassessment of the treatment are necessary.

The size of a burn is described by an estimation of the percentage of the total body-surface area that has sustained second- or third-degree burns. Errors in estimating the size of the burn, often results in overestimation. Cooling of Burns: Cooling of burns with the use of cold (15-25°C) tap water within 30 minutes after injury has been shown to reduce the pain, the depth and extent of the injury, the need for surgical excision of the burn, scarring and mortality. Cooling of burns should continue until the pain is substantially reduced or resolved. The use of ice or ice water may increase tissue injury and should be avoided. Concern that cooling of large burns may result in hypothermia has been based on a single study in small animals with extensive burns.

Management of Blisters: The debate regarding the removal of burn blisters has been confusing due to conflicting data regarding the in vitro effects of blister fluid. Two clinical trials involving patients and volunteers with superficial burns demonstrated that intact blisters healed faster and were less likely to
become infected than blisters that were ruptured. Blister larger than 3 cm in diameter and those over mobile areas usually rupture spontaneously and also they may be aspirated under sterile conditions. When blisters rupture, they should be washed with soap and water, and the non-adherent necrotic epidermis carefully removed. In order to relieve pain while the burn is being thoroughly cleansed, the patient often requires analgesia with oral or perenteral opioids.

**Local Therapy for Burns**: Although first-degree burns do not require any specific therapy, topical nonsteroidal anti-inflammatory drugs or aloe vera may be used to reduce the pain. Superficial second-degree burns should be treated with a topical antimicrobial agent or an absorbent occlusive dressing. Studies suggest that occlusive dressings are more convenient and less painful than treatment with topical antimicrobials and also result in more rapid healing. Deep second-degree burns and third-degree burns should be covered with a topical antimicrobial agent, and the patient should be referred to a burn specialist for consultation regarding the need for excision and grafting. Routine use of systemic antibiotics is not supported by the evidences.

The optimal treatment for heavily contaminated or infected burns and those with a large amount of exudates is application of topical antimicrobial agents and absorbent gauze dressings.

Traditional topical antimicrobial agents that contain silver, such as silver sulfadiazine, confer wide antimicrobial coverage and are most useful for deep second-degree burns and third-degree burns. However, the use of these antimicrobial agents is associated with cellular toxicity and delayed healing. Synthetic dressings that reduce the cytotoxic effects of silver by slowly releasing it in small amounts are now available. They are more effective than silver sulfadiazine in reducing pain and improving healing and are also cost effective.

A large number of synthetic and biological occlusive dressings have been evaluated for the local management of burns. Absorbent hydrocolloid dressings can be used for weeping burns, although they tend to become malodorous. As compared with silver sulfadiazine, these dressings have been shown to result in less pain, better acceptance and compliance, and more pleasing cosmetic results in patients with superficial partial-thickness burns. They are also as effective as and less expensive than collagen based dressings.

Method of performing an escharotomy

The polyurethane films are not recommended since they do not absorb exudates. A silicone mesh dressing that adheres gently to the wound bed and allows wound exudates to escape onto a secondary dressing is also available and results in faster healing than treatment with silver sulfadiazine. Biological dressings based on collagen or skin cells should be reserved for deep burns and should be applied by burn specialists.

**Chemical Burns**: Chemical burns cause tissue injury through the interaction of the chemical agent with the tissue. Initial treatment consists of copious water lavage commencing at the scene and removal of any particles. The important exception to the treatment of a chemical burn with water lavage involves injury from elemental metals (lithium, sodium, magnesium, and potassium), which spontaneously ignite with water. Exposure to hydrofluoric acid, which is used in etching and rust removal, leads to intense pain and tissue damage.
Treatment includes copious irrigation followed by the application of calcium gluconate gel or subcutaneous injection of calcium gluconate, with the goal of relieving the pain. A burn from hydrofluoric acid that involves more than 5% of total body-surface area, or more than 1% of total body surface area if the concentration of hydrofluoric acid is greater than 50%, requires hospital admission for electrocardiographic monitoring and serial measurements of calcium levels since life-threatening arrhythmias and hypocalcemia can occur. The patient should be referred to a burn specialist because immediate excision of the wound may be necessary in cases of hypocalcemia that is unresponsive to intra-arterial or intravenous calcium gluconate.

Frostbite: Frostbite occurs when exposure to cold results in the freezing of tissue. It usually affects the most exposed appendages. Fingers, toes, nose and ears are among those most affected parts. Initial treatment consists of rapid rewarming over a period of 20 minutes in water that is at a temperature of 40 to 42°C. The appearance of the skin after rewarming and over the next 24-48 hours is the main clinical factor that is used to determine the level and extent of the injury. The use of radio-imaging with technetium-99m at 1 week is helpful in predicting the ultimate level of tissue injury and the extent of amputation that may be required. Opioids are used to control pain. Massaging the area is not recommended, since it may increase tissue damage.

Frostbite is classified in the same way as burns - that is according to the size and depth of the injury within 24-48 hours after rewarming. Other than rapid rewarming, there are no unified treatment protocol for frostbite. General principle for treatment include splinting and elevation of the injured area. Treatment with ibuprofen, 400mg twice daily, is recommended to decrease the levels of prostaglandin and thromboxane, since elevated levels of these lead to vasoconstriction and platelet aggregation, resulting in progressive tissue injury. Although some recommends routine debridement and application of aloe vera because of the high concentration of prostaglandin $\text{F}_2\alpha$ and thromboxane $\text{B}_2$, there is a risk of desiccation. So sterile aspiration of tense blisters is recommended that are larger than 3 cm in diameter.

Recently, it has been shown that tissue plasminogen activator given intravenously or intra-arterially within 24 hours after injury and within 6 hours after rewarming significantly decreases the extent and level of amputation. Other treatment includes sympathectomies, hyperbaric oxygen, and pentoxyfylline. In general, the treatment remains conservative until the wound is completely healed or clearly demarcated. This process usually takes more than 3 weeks, at which time, final debridement and grafting, flap coverage, or amputation is performed as needed basis.

**Conclusion**

Many types of cutaneous wounds are managed by different specialties of health care practitioners in multiple clinical settings. However, wound irrigations, debridement, protection from further trauma and bacterial contamination, creation of a moist wound environment, and judicious use of antibiotics (when indicated) will often help achieve optimal outcomes.

**References**

2. Advances in skin and wound care, 2008; by Baranoski, Sharon
10 Facts About Smoking

1. Tobacco is the leading preventable cause of death in the world. It causes 1 in 10 deaths among adults worldwide. In 2005, tobacco caused 5.4 million deaths, or an average of one death every 6 seconds. The death toll is projected to reach more than 8 million by 2030 if current trends continue.

2. Tobacco kills up to half of its regular users. On average 29% of people around the world are smoking tobaccos. Smoking is more common among men- 47.5% of all men smoke compared to 10.3% of women.

3. More than 80% of the world's, more than one billion smokers live in low- and middle-income countries. Unless urgent action is taken, by 2030, more than 80% of tobacco related deaths will occur in the developing world.
10 Facts About Smoking

4. Tobacco caused 100 million deaths in the 20th century. If current trends continue, there could be up to one billion deaths in the 21st century.

5. The smoke produced by burning tobacco products is known as second-hand tobacco smoke or environmental tobacco smoke. Tobacco smoke in enclosed spaces is breathed by everyone, exposing both smokers and non-smokers to its harmful effects. This is commonly referred to as involuntary smoking or passive smoking.

6. Second-hand tobacco smoke is dangerous to health. There are about 4000 known chemicals in tobacco smoke. Second-hand smoke also causes heart disease and many serious respiratory and cardiovascular diseases in adults which can lead to death.
10 Facts About Smoking

Source: World Health Organization

1. There is no safe level of exposure to second-hand tobacco smoke. Neither ventilation nor filtration, even in combination, can reduce the exposure indoors to levels that are considered acceptable. Only 100% smoke-free environments provide effective protection.

2. An estimated 700 million children, or almost half of the world’s children, breathe air polluted by tobacco smoke, particularly at home. Second-hand smoke causes many serious diseases in children and worsens conditions such as asthma.

3. Exposure to second-hand smoke also imposes economic costs on individuals, businesses and society as a whole, in the form of direct and indirect medical costs and productivity losses.

4. The International Labor Organization estimates that at least 20,000 workers die every year due to exposure to smoke at work. The United States Environmental Protection Agency estimates that second-hand smoke is responsible for about 3000 lung cancer deaths annually among non-smokers in the country.

5. An estimated 700 million children, or almost half of the world’s children, breathe air polluted by tobacco smoke, particularly at home. Second-hand smoke causes many serious diseases in children and worsens conditions such as asthma.

6. Exposure to second-hand smoke also imposes economic costs on individuals, businesses and society as a whole, in the form of direct and indirect medical costs and productivity losses.

7. There is no safe level of exposure to second-hand tobacco smoke. Neither ventilation nor filtration, even in combination, can reduce the exposure indoors to levels that are considered acceptable. Only 100% smoke-free environments provide effective protection.

8. The International Labor Organization estimates that at least 20,000 workers die every year due to exposure to smoke at work. The United States Environmental Protection Agency estimates that second-hand smoke is responsible for about 3000 lung cancer deaths annually among non-smokers in the country.

9. An estimated 700 million children, or almost half of the world’s children, breathe air polluted by tobacco smoke, particularly at home. Second-hand smoke causes many serious diseases in children and worsens conditions such as asthma.

10. Exposure to second-hand smoke also imposes economic costs on individuals, businesses and society as a whole, in the form of direct and indirect medical costs and productivity losses.
Presence of blood in the urine can originate at any point along the urinary tract and both gross and microscopic hematuria may represent serious underlying disease. Microscopic or non-visible hematuria, unlike gross hematuria is often an incidental finding during evaluation of urinary tract infection or during routine health screening. Although remaining asymptomatic, it may be associated with urologic malignancy in up to 10% of adults. Despite this risk, results of a recent study revealed that 39-90% of persons with microscopic hematuria on screening urinalysis received no follow-up testing. Non-visible or microscopic hematuria thus presents a challenging clinical scenario for general practitioners.

Prevalence

Non-visible hematuria is present in about 2.5% of the general population, although it can be as high as 20%, depending on features of the study population, such as age, sex, the presence of risk factors for disease, and the definition used. Within patients with asymptomatic non-visible hematuria detected by screening, the overall incidence of serious conditions such as urological malignancy is <1.5%, so the consensus is that populations screening is not warranted. In contrast, a cause for non-visible hematuria is found in about 15% of cases selected for referral from primary care towards specific health care authorities. But there is currently no evidence to support opportunistic testing for hematuria without a clinical reason.

Causes

The etiologies of microscopic hematuria are numerous and range from clinically insignificant causes to potentially life-threatening neoplastic lesions. In one study of 1930 patients who had completed urological evaluation for hematuria, 982 had microscopic hematuria. Nearly 1 in 5 patients with microscopic hematuria had significant disease compared with about 1 in 3 patients with gross hematuria. In this study, 92 (9.4%) of the patients with microscopic hematuria had cancer. Evaluation of the upper urinary tract followed by cystoscopy fails to identify the source of microscopic hematuria in 19-68% of patients. Finally the younger the patient, the less likely it is the etiology to be identified.

Investigation of Non-Visible Hematuria in Primary Care

Urine Dipstick: Chemical dipsticks detect intact red cells (RBC), free hemoglobin, or free myoglobin.

Medications That Can Cause Hematuria

- Aminoglycosides
- Amitriptyline
- Analgesics
- Anticonvulsants
- Aspirin
- Busulfan
- Chlorpromazine
- Cyclophosphamide
- Diuretics
- Oral contraceptives
- Penicillins
- Quinine
- Vincristine
- Warfarin
Algorithmic Approach to Microscopic Hematuria in Adults

Microscopic evaluation of urine to confirm presence of RBCs

- Signs or symptoms of infection (e.g. dysuria, frequency, flank/CVA pain, leukocyte esterase, nitrites, white blood cells, bacteria)
  - Yes
  - Treat infection; confirm resolution of microscopic hematuria with follow-up urinalysis six weeks after completion of therapy
  - No
  - Findings in support of glomerular cause (e.g. proteinuria, elevated creatinine level, red cell casts, dysmorphic RBCs)
    - Yes
    - Refer to nephrology subspecialist
    - No
    - Other etiology probable (e.g. vigorous exercise, trauma to urethra, menstruation, offending medication)
      - Yes
      - Proceed with upper urinary tract radiographic evaluation
      - No
      - Stop and retest urine after possible contributing factor stopped

Intravenous Urography
Strengths: detecting transitional cell carcinoma of kidney or ureter in renal masses larger than 3 cm in diameter
Limitation: detecting renal masses smaller than 3 cm in diameter or lesions of the bladder or urethra

Renal Ultrasonography
Strengths: inexpensive and safest detection of solid masses larger than 3 cm in diameter and hydronephrosis
Limitation: detection of small solid tumors less than 3 cm in diameter

Computed Tomography
Strengths: detection of renal calculi, small renal and pararenal abscesses
Limitation: high cost and limited availability in some cases

Proceed with assessment of lower urinary tract

Urine cytology

- Negative findings; low-risk patient
  - No further assessment needed
- Positive findings or all patients older than 40 years or younger patients with risk factors for urothelial tumors
  - Referral to urology subspecialist for cystoscopy

Note: High risk = smoking, history of urothelial neoplasm, age older than 40 years, occupational exposure to benzenes or aromatic amines.
CVA = costovertebral angle. RBC = red blood cell.
They provide an instant result. Although the efficiency of this test is difficult to estimate, analysis of data sets indicates that it is a reasonable way to detect non-visible hematuria in primary care.

**Urine Microscopy:** Red blood cell counts have been used to define microscopic hematuria, and cut-off points have varied (including ≥2 cells per high power field and ≥5 cells per power field). Microscopy provides an accurate measure of red blood cells when assessed by an expert in fresh voided early morning midstream specimens of urine. However, time to analysis affects the integrity of RBC. In a prospective multicenter study, RBC counts dropped by 5-9% at five hours, 11-28% at 24 hours, and 29-35% at 72 hours. Because immediate microscopy is not feasible in primary care, the accuracy of quantitative RBC microscopy is questionable.

**References**

2. American Family Physicians, 2006; 73: 1748-1754

---

<table>
<thead>
<tr>
<th>Causes of Isolated Microscopic Hematuria*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Origin</strong></td>
</tr>
<tr>
<td>---</td>
</tr>
<tr>
<td><strong>Glomerular</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Nonglomerular</strong></td>
</tr>
<tr>
<td><strong>Upper urinary tract causes</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Lower urinary tract causes</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Uncertain</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

* Disorders causing microhematuria are presented roughly in order of descending frequency of presentation, according to available data. HIV denotes human immunodeficiency virus.
Eosinophilic esophagitis is an increasingly recognized cause of a variety of esophageal symptoms, including dysphagia, food impaction, atypical chest pain and heartburn that does not respond to therapy. Its cause is unknown, but allergic and immune-mediated mechanisms similar to those of asthma and other atopic diseases are implicated.

Eosinophils invade the epithelium of the esophagus, possibly in response to allergens in food and the air, in a process mediated by type 2 helper T cells, which release the cytokines interleukin 5, interleukin 13, and eotaxin-3.

Barium studies may show focal narrowing and subtle concentric rings (trachealization).

Endoscopy may reveal mucosal fragility, rings, strictures, linear furrows, and a narrow caliber.

Biopsy findings demonstrate eosinophil migration into the esophageal epithelium.
Until recently, the cause of intermittent or progressive difficulty in swallowing solids was thought to be a mechanical problem such as a stricture, ring, or cancer of the upper gastrointestinal tract (GIT). Also motility disorders such as achalasia or diffuse esophageal spasm were implicated in difficulty in swallowing of both solids and liquids. But now health professionals are becoming aware of a relatively new disease, eosinophilic esophagitis (EE), as a cause of dysphagia in both adults and children.

Over the last several years, EE has emerged as the topic of an ever-increasing number of articles in the adult and pediatric gastrointestinal as well as allergy journals. EE is an increasingly recognized cause of a variety of esophageal symptoms, including dysphagia, food impaction, atypical chest pain, and heartburn that does not respond to medical therapy. Although the exact cause is still not well known, allergic and immune-mediated mechanisms similar to those of asthma and other atopic diseases are implicated.

**When and how was Eosinophilic Esophagitis Detected?**

Abundant eosinophils in the esophagus were first described in 1977 in a 51-year-old man with dysphagia, chest pain, and a personal history of severe asthma and marked peripheral eosinophilia. In 1983, a similar case was reported in an adolescent with dysphagia. In both patients, large numbers of eosinophils were also noted in the duodenum, suggesting that these findings were part of a systemic hypereosinophilic syndrome. Increased numbers of eosinophils in the GIT have also been described in a number of diseases, including Crohn’s disease, connective tissue disorders, malignancy, various infections, and drug hypersensitivity reactions.

However, not until 1993, EE was described as a distinct clinical entity, consisting of isolated esophageal eosinophilia (typically more than 15 eosinophils per high-power field) in patients with dysphagia. What was originally a case reportable disease ten years ago is now one that is appearing in numerous gastrointestinal and allergy journals on a regular highlight.

**Epidemiology**

Eosinophilic esophagitis predominantly affects men between the ages of 20 and 40, but cases in women and in younger and older patients have also been reported. Recent systemic reviews found male-to-female ratio of approximately 3:1. Epidemiologic studies suggest that EE may be as common as inflammatory bowel disease. In a study of children in Cincinnati, Ohio, the incidence was estimated at 10 per 100,000 children per year, and the prevalence was estimated at 43 per 100,000. Of interest, 97% of cases were diagnosed after the year 2000. EE may occur in isolation or in conjunction with eosinophilic gastroenteritis. While isolated EE was previously thought to be a rare condition, in the last several years, numerous case series have been reported from North and South America, Europe, Asia and Australia. The cause of this dramatic rise is likely a combination of an increasing incidence of EE as well as a growing awareness of the condition.

**The Current Consensus Definition for EE**

- Clinical symptoms of esophageal dysfunction such as dysphagia, food impaction
- At least 15 eosinophils per high-power field
- Either no response to a high-dose proton pump inhibitor or normal results on pH monitoring of the distal esophagus and
- Other features such as basal zone hyperplasia, edema, and papillary elongation are seen to a greater extent in patients with EE than in patients with gastro-esophageal reflux disease (GERD)
Pathophysiology

The growing incidence of EE is quite similar with asthma, eczema, allergic rhinitis, and other atopic diseases, raising the possibility that these disorders share common environmental exposures and similar inflammatory pathways. The pathological mechanisms of EE are unknown, but emerging evidence suggests that like other allergic diseases, it is an immune response mediated by type 2 T helper cells. Recently, eotaxin-3, a potent attractant for eosinophils, was shown to be markedly over expressed in children with EE compared with controls. Acid reflux does not appear to be a causative factor in most patients. However, reflux may play a secondary role, as some patients have experienced symptomatic, endoscopic, and histological resolution of EE after treatment with a proton pump inhibitor.

The role of environmental allergens contributing to EE has also been suggested in humans. There may be seasonal variation of EE, as suggested by a case report of a 21-year-old woman when had EE that worsened symptomatically and histologically during the pollen season but resolved during winter. This is another example of aeroallergens that may play a role in this disease. Biopsies obtained during non-pollen months were normal, suggesting that tissue eosinophilia was triggered by pollen exposure. Studies focusing on children suggest that food allergies are a major contributor to EE. In children, a strict amino-acid elemental diet has led to complete resolution of symptoms and a marked decrease in EE. However, symptoms tend to recur once patients resume a regular diet. It is unclear if dietary modification is effective in adults. In six adults with EE and a history of wheat and rye allergies, symptoms did not improve when these foods were eliminated and did not worsen when they were reintroduced. The most commonly identified food allergens include milk, soy, egg, wheat, peanuts, and shellfish. Evidence of a genetic predisposition to this disease is also growing with a number of case reports describing multiple affected family members spanning generations.

Diagnosis

The diagnosis of EE has often been overlooked with many patients having had endoscopies with alternative diagnoses that included Schatzki's rings or gastroesophageal reflux disease. In many cases, misdiagnosis led to repeated endoscopies, esophageal dilations and delay in the institution of appropriate medical interventions.

Clinical Presentation: More than 90% of adults with EE present with intermittent difficulty in swallowing solids, while food impaction occurs in more than 60%. Heartburn is the only manifestation in 24% of patients. Non-cardiac chest pain, vomiting, and abdominal pain have also been seen, but less frequently. In children, presenting symptoms vary with age and include feeding disorders, vomiting, abdominal pain, and dysphagia. Up to 80% of patients with EE have a history of atopic diseases such as asthma, allergic rhinitis, or allergies to food or medicine. One-third to one-half of patients has peripheral eosinophilia, and up to 55% have increased serum levels of immunoglobulin E (IgE). Children with EE have a higher frequency of atopic symptoms and peripheral eosinophilia than does in adults.

Endoscopic Findings: Although no single endoscopic feature of EE is pathognomonic, the esophagus shows mucosal fragility in 59% of cases, a corrugated or ringed appearance in 49%, strictures in 40%, whitish papules in 16%, and a narrow caliber in 5%. Many of these features, including longitudinal furrows, are subtle and can be missed. Between 9% and 32% of patients with symptoms suggesting EE have normal endoscopic findings. In large clinical series of 381 children, the most common endoscopic features were...
linear furrows (41%), normal appearance (32%), esophageal rings (12%), and white plaques (15%).

**Histological Findings:** While certain endoscopic features are characteristic of EE, this condition is ultimately diagnosed by obtaining biopsy specimens, which demonstrate histological findings of increased intramucosal eosinophils in the esophagus. Eosinophilic infiltration in the stomach or duodenum is typically absent. Another histological feature of EE is superficial layering of the eosinophils. Eosinophilic microabscess, intercellular edema, degranulation of eosinophils, and the presence of other inflammatory cells such as lymphocytes may be seen. Studies have also shown evidence of subepithelial fibrosis in EE patients. Another histological finding in EE is epithelial hyperplasia, defined by papillary height elongation and basal zone proliferation. Epithelial hyperplasia is also a cardinal feature of the histopathology of reflux esophagitis.

**Additional Diagnostic Testing:** Radiographically, EE can appear as a series of concentric rings on barium study- hence the term 'ringed esophagus' appeared. In a study of 14 patients with EE, 10 (70%) had strictures of various length with rings within the strictures.

These findings support the theory that inflammation can lead to submucosal fibrosis, remodeling, narrowing, and eventually symptoms. Furthermore, two recent studies found that children with EE had increased subepithelial collagen deposition in their biopsy specimens suggesting increased potential for fibrosis.

**GERD and EE: What is the Relationship?**

Given the high prevalence of GERD in the general population, much time and effort have been spent on comparing EE with GERD. Both diseases share varying degrees of esophageal eosinophilia, leading to speculation on the relationship of EE and GERD. A recent review article suggested that the mucosal injury caused by acid reflux may allow swallowed allergens to penetrate an esophageal layer that is otherwise impermeable to most proteins, thereby causing mild eosinophilia. Conversely, the intense degranulation of activated eosinophils seen in EE can trigger changes in the lower esophageal sphincter that could predispose to acid reflux. Although the clinical and pathological features may overlap, GERD and EE appear to have different genetic profiles. In a recent pediatric study, it was found that the genes up-regulated in EE were markedly different that those in chronic esophagitis. This suggests that while the two diseases share a constellation of symptoms, they have different pathogenesis. Nevertheless, because of this possible overlap, the diagnosis of EE should be made after acid reflux has been either treated or excluded with pH testing.

**Treatment**

**Dietary Therapy:** Treatment approaches for EE vary between children and adult patients. In children, the most common approach is to embark on elimination of some diet and introduction of elemental diet. Strict elemental amino-acid diets have resulted in complete symptomatic and histological resolution of EE in children. However, these elemental diets often have to be given by nasogastric tube because they are unpalatable, and the disease tends to return once the diet is discontinued. Elimination diets, based either on avoiding the six foods most commonly associated with allergy (egg, wheat, soy, cow’s milk protein, seafood, peanuts) or on allergy testing such as skin prick testing or atopy patch testing, have shown promise in children. Similar large-scale studies of elimination diets in adults have not been so far conducted.
Allergy Evaluation: The recent recommendations devoted considerable attention to the role of allergy evaluation. Between 50-80% of patients with EE have a coexisting atopic disease such as atopic dermatitis, eczema, allergic rhinitis, or asthma, with a higher prevalence in children than in adults. Evidence suggests that allergy testing may predict response to therapy. Therefore, the current recommendation for all patients with EE is to undergo a complete evaluation by an experienced allergist. Checking the peripheral blood eosinophil count before and after treatment is reasonable, as many patients have elevated eosinophil counts that decrease after treatment. Similarly, many patients with EE have elevated serum total IgE levels suggesting a concomitant atopic disease. So IgE level should also be evaluated and treated. Data on atopy patch testing in EE are currently limited but promising.

Medical Therapy: Swallowed fluticasone is the mainstay of therapy for both children and adults. In one case series, 21 adult patients with EE received a 6-week course of swallowed fluticasone two to four puffs (220 μg/m puff) twice daily. Symptoms completely resolved in all patients for at least 4 months, and no patient needed endoscopic dilation. In another study, 19 patients treated with fluticasone for 4 weeks showed dramatic improvement both symptomatically and histologically. However, after 3 months, 14 (74%) of them had a recurrence of symptoms pointing to the chronic relapsing nature of the disease. Swallowed fluticasone is generally well tolerated, although cases of esophageal candidiasis have been reported.

Acid suppression still has an unclear role in the treatment of EE. Most patients referred for further evaluation of EE have tried twice-daily proton pump inhibitor therapy without success. The impact of concomitant therapy with a proton pump inhibitor has not yet been determined, but the recent guidelines suggest that these drugs are reasonable as co-therapy in patients who also have GERD symptoms. Systemic corticosteroids have been used with success in both adults and children with hypereosinophilic syndromes as well as in patients with refractory EE, but adverse effects limit their routine and long-term use. Cromolyn sodium, a mast cell stabilizer, and montelukast, a leukotriene inhibitor, have been used with limited success. Mepolizumab, a humanized monoclonal antibody to human interleukin 5, decreased the number of eosinophils in the esophagus and peripheral blood and improved clinical symptoms in patients with refractory EE in a recent open-label trial. Further studies with mepolizumab and other biological agents are expected.

Invasive Procedure: Endoscopic dilation with either a guide wire or a balloon technique is often used to treat strictures and a diffusely narrowed esophagus in patients with EE. A common endoscopic feature is mucosal fragility. Although this procedure is safe; the risk of perforation appears to be greater than in those with other indications for dilation. Nevertheless, immediate symptomatic improvement has been reported in 83% of patients after dilation, with symptoms recurring in 20% within 3-8 months. Current recommendations suggest that dilation should be done cautiously in patients who have documented esophageal narrowing for which drug therapy has failed.

Conclusion
Although during the past few years, increased awareness of the prevalence and presentations of EE has developed, the upcoming researches will be helpful in enhancing the understanding of its natural history, pathophysiology and treatment modalities. Further studies delineating the molecular mechanisms behind EE should lead to the development of targeted therapeutic agents. Therefore the results of these ongoing researches and other studies are eagerly awaited.

References
1. Cleveland Clinic Journal of Medicine, 2008; 9: 623-633
2. Northwestern University Feinberg School of Medicine, USA
The insertion of a femoral venous catheter may be necessary when peripheral access to the circulatory system is compromised and no other sites for placement of a central catheter are available.

**Indications**

Such catheter are used to administer large fluid volumes or potentially irritating medicines, to provide temporary access for emergency dialysis, for immediate central access during emergency resuscitation, to facilitate cardiac catheterization, or, in rare instances, for drawing blood, if a patient requires frequent blood sampling and no other access site is available.

**Contraindications**

There are few absolute contraindications to placement of a central catheter, other than the patient's not agreeing to the procedure. As compared with subclavian or jugular catheters, femoral catheters are associated with higher risk of infection, thrombosis, and in the absence of ultrasound guidance, arterial puncture may also be a possibility. Considering these factors, a safer option if exists, then that should be chosen. Uncooperative patients place both the operator and themselves at risk of injury, thus it should be considered with importance. Evident infection at the site where the needle will enter should prompt the operator to seek another site. In addition, complications are more likely to occur if the site is

**General Preparations**

- Appropriate selection of patients suitable for femoral venous catheterization should be confirmed as well as selecting the correct anatomical location.
- The procedure should be explained to the patient and a written informed consent should be taken when possible.
- An assistant should be present with the authorization to halt the procedure if inappropriate technique is applied.
- For optimal exposure of the femoral region, external rotation and abduction of the patient's leg away from midline should be obtained.
- The vein should be localized by palpating the femoral artery or by using ultrasonography (USG) as the femoral vein lies medial to the femoral artery while running distal to inguinal ligament.
- The skin should be prepared with chlorhexidine, and the sterile area should be covered with a sterile drape. Full sterile dress should be used.
- Then the central-catheter kit is prepared and the catheter ports are flushed with sterile saline. If ultrasound guidance is used, then the ultrasound probe should be prepared for sterile use.
- The selected area should be anesthetized with long-acting anesthetic such as lidocaine with epinephrine or bupivicaine. Adequate analgesia will increase the patient's comfort and the operator's likelihood of successful catheter placement.
distorted by trauma or is obscured. In patients with uncorrected bleeding disorders, a central catheter should be placed with caution and only if necessary. Central catheters should not be placed by inexperienced hands.

**Equipments**

Materials required for this procedure include personal protective equipment, a bag of sterile saline for infusion, intravenous tubing, local anesthetic medications, a central-catheter kit, and blood-drawing equipment. The central-catheter kit typically includes a sterile drape, skin preparation solution, sterile gauze, an introducer needle, a guide wire, a scalpel, a dilator, and intravenous catheter, a mechanism for securing the catheter to the skin, and a sterile, transparent dressing.

**Procedure of Placing the Catheter**

After ensuring proper anesthetization, the position of the femoral vein is reconfirmed by palpating femoral artery or by visualizing directly with ultrasonography. The introducer needle is then inserted at 45° angle from the skin, directed along the course of the artery, while pulling back the plunger. In order to prevent the femoral-artery cannulation, palpation of the artery should be maintained while advancing the needle. Once a flash of blood is seen, the needle is carefully anchored to avoid dislodging it from an intraluminal location. The syringe is detached and the guidewire is to be thread through the needle. It should pass easily without resistance into the lumen of the vessel. While maintaining the grasp of the wire, the introducer needle is removed. The skin is then incised at the wire-entry site with a scalpel keeping the sharp edge away from the wire.

The dilator is advanced over the wire to make a tract through the tissues into the vessel. Larger catheters may have dilators that fit inside them and must be advanced together with the catheter. But unless that is the case, the dilator is to be removed and the catheter is stranded over the wire. Before advancing the catheter past the skin, the guidewire is firmly grasped which is protruding from the proximal end of the catheter. It is often necessary to feed the wire back through the catheter to accomplish this. After the catheter has been threaded into the vessel, the wire is removed. The intravenous location of the catheter is confirmed, the sterile saline is flushed through the port, and the catheter is secured with sutures or staple. A sterile dressing is placed over the site before removing the drapes.

**Complications and Their Remedies**

Potential complications include infection, thromboembolism, arterial puncture, and hematoma. It is important to be aware of these possible problems and to keep them in mind when monitoring the patient. Most of these complications can be prevented by following proper sterile technique, using ultrasonography for placing the catheter, limiting the number of attempts at placing the catheter, and removing it as soon as possible. If the femoral artery has been punctured, pressure is applied to the site for at least 10 minutes. Small hematomas may be managed conservatively, but continuing hemorrhage may require surgical intervention.

*Source: The New England Journal of Medicine, 2008; 358: 26*
23

Clindamycin Phosphate, Benzoyl Peroxide Combination Improves Severe Acne

O
tal antibiotics for severe acne have limitations because of side effect concerns and mounting evidence of antibiotic resistance to Propionibacterium acnes. A topical combination of clindamycin phosphate 1.2% and low-concentration benzoyl peroxide (BPO) 2.5% aqueous gel is effective for severe inflammatory and noninflammatory acne, according to a study. The analysis included 531 patients with severe acne who participated in 2 larger multicenter, double-blind studies which involved patients with both moderate and severe acne. Patients were randomized in a 2:2:2:1 ratio to receive clindamycin phosphate 1.2% plus low-concentration BPO 2.5%, each active ingredient, or vehicle, once daily for 12 weeks. At weeks 4, 8 and 12, the combination showed statistically significant efficacy over vehicle for all lesion types. The median percent change from baseline with the topical combination was 49% in inflammatory lesions and 45% in noninflammatory lesions. The overall median reduction in total lesion counts with the study drug was 44% at week 12. In both treatment arms, mean cutaneous tolerability scores (each for burning, stinging, scaling, erythema, and itching) at weeks 4, 8, and 12 were less than 1 in a scale of 0 (none) to 3 (severe). No patient in either arm dropped out because of burning, stinging, scaling, erythema, or itching.

Desonide Hydrogel in Dermatitis

Use of desonide hydrogel in patients with mild to moderate atopic dermatitis resulted in significant reductions in symptom severity and high patient satisfaction. The surveys included questions about prior medication use for atopic dermatitis, assessments of symptom severity, satisfaction, intent to continue, and recommendation of desonide hydrogel to other patients. A total of 1,025 patients completed the surveys (60% female; mean age, 35 years), with approximately two-thirds having used at least 1 prior medication for atopic dermatitis. Results showed desonide hydrogel reduced redness by 58%, scaling/flaking by 59%, and itching severity was reduced by 60% ($P < 0.05$). Of the patients ($n = 692$) who had used other medications for their condition, satisfaction with desonide hydrogel was nearly twice as high, on average, than satisfaction with their prior medications ($P < 0.05$). Satisfaction with desonide hydrogel for all respondents averaged 7.7 to 9 on a scale of 1 (not at all satisfied) to 9 (very satisfied), with 83% of respondents rating their satisfaction between 7 and 9. Most patients said they would continue using desonide hydrogel if necessary, and most said they would recommend the medication to other patients. The researchers emphasized for further studies to confirm that the effectiveness of desonide hydrogel plus high patient satisfaction may enhance treatment compliance.

Calcipotriol Plus Betamethasone Dipropionate Formulation Effective in Scalp Psoriasis

A
cording to the researchers, a scalp formulation combining calcipotriol plus betamethasone dipropionate markedly decreases itching related to scalp psoriasis. Participants in the trial were aged 18 years or more and had scalp psoriasis of at least moderate severity that covered at least 10% of the scalp with at least 1 clinical sign (redness, thickness, or scaliness) that was considered at least moderate and the other 2 signs at least slight by the investigator. Three hundred and twelve patients were randomized to treatment with a scalp solution containing calcipotriol 50 μg/g plus betamethasone dipropionate 0.5 mg/g once daily or a scalp solution containing calcipotriol alone twice daily to the affected psoriatic lesions on the scalp for up to 8 weeks. Patients assessed the extent of itching at weeks 0, 2, 4, and 8. Results showed that the number of patients reporting itching on the Skindex-16 scale was significantly lower in the 2-compound group compared with the calcipotriol-alone group at all time points (week 2: $P < 0.001$; week 4: $P < 0.001$; week 8: $P = 0.044$). This was also true for scalp burning (week 2: $P < 0.001$; week 4: $P < 0.001$; week 8: $P = 0.002$) and hurting at weeks 2 and 4 (week 2: $P = 0.003$; week 4: $P < 0.001$). After 2 weeks of treatment, a significant effect on itching was achieved in the 2-compound group compared with the calcipotriol-alone group at all time points (week 2: $P < 0.001$; week 4: $P < 0.001$; week 8: $P = 0.002$) and hurting at weeks 2 and 4 (week 2: $P = 0.003$; week 4: $P < 0.001$). After 2 weeks of treatment, a significant effect on itching was achieved in the 2-compound group compared with baseline ($P < 0.001$), and this reduced level of itching remained constant throughout the treatment period. This effect was also true for symptoms of burning and hurting. After 8 weeks, a complete absence of itching was more than twice (57.5% vs. 26.7%, $P < 0.001$) as likely in the 2-compound group.

Source: http://www.pslgroup.com
Pictures used in cover page

*Staphylococcus aureus* bacteria
A person on indirect smoke inhalation
Doctor taking care of a wounded patient

Design & Graphics
DTP Medical Department
Beximco Pharmaceuticals Ltd.

Published in April 2009