

wound healing perspectives®

A CLINICAL PATHWAY TO SUCCESS

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➔ VASCULOPATHY VS VASCULITIS

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Vasculopathy vs. vasculitis: a primer

In this issue of *Wound Healing Perspectives*, we shed light on two disorders that are often misunderstood and misdiagnosed—**vasculopathy**, a disorder that is characterized by occlusion of the blood vessels, particularly due to hypercoagulable states, and **vasculitis**, which is generally characterized by inflammation of the blood vessels. Leg ulcers, caused by vasculopathy, are sometimes misdiagnosed as venous ulcers.

When a wound is vasculopathic, patients typically have pain, necrosis, and ischemia. Patients with vasculitic ulcers typically suffer from inflammation in arteries which results in symptoms of palpable purpura, urticaria, papules, or nodules. It is important to note that biopsies should be performed on patients with suspected vasculitic ulcers or lesions.

Treatment of vasculopathy involves controlling coagulation, while treatment for vasculitis focuses on controlling the etiology of the inflammatory response.

We hope this issue will help you better understand and recognize these types of wounds so that prompt treatment can follow. Thank you for your interest in National Healing Corporation and Wound Healing Perspectives. We welcome your comments.



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Vasculopathy and hypercoagulable states

Hypercoagulability, which is caused by a blood clotting disorder, is characterized by decreased blood flow due to clotting in the blood vessels, as well as intrinsic alterations in the nature of the blood itself, according to a 2003 study by Deitcher et al.

Hypercoagulability can lead to large and small vessel thrombosis. Venous blood clots can potentially travel through the bloodstream and cause a pulmonary embolus. Blood clots in the arteries, meanwhile, can increase a person's risk for stroke, heart attack, severe leg pain, difficulty walking, or even the loss of a limb, according to information from the Cleveland Clinic web site.

Hypercoagulability is a condition that can be genetic or acquired. Inherited hypercoagulable conditions include: Factor V Leiden (the most common hereditary blood coagulation disorder in the United States); prothrombin (coagulation factor II) gene mutation; elevated levels of fibrinogen, a protein that plays a key role in blood clotting; deficiencies of anticoagulant proteins such as antithrombin, protein C, and protein S; "sticky" platelets;



MOST HYPERCOAGULABLE STATES ARE ASSOCIATED WITH A SPONTANEOUS ONSET OF DEEP VEIN THROMBOSIS AT AN EARLY AGE.

abnormal fibrinolytic system, including hypoplasminogenemia, dysplasminogenia, and elevation in levels of PAI-1; elevated levels of factor VIII which is still being investigated as an inherited condition; and other disease states such as malignancy, nephrotic syndrome, myeloproliferative disorders,

(continued on page 3)



HIGHLIGHTS INSIDE

Diagnosing and treating vasculopathy	2-3
Symptoms of hypercoagulable states	3,7
Vasculitis	4-6
Indications for hyperbaric oxygen.....	4
Working with hyperbaric oxygen.....	5
Heparin vs. warfarin	7
Working with a Wound Healing Center	8

Who should be tested?



Selected testing of hypercoagulable state should be considered in the following circumstances:

- Idiopathic (i.e., spontaneous) venous thromboembolic event (VTE)
- VTE at young age (<50 years old)
- Recurrent VTE
- VTE in unusual sites
- VTE in the setting of a strong family history of VTE
- Recurrent pregnancy loss (>3 consecutive first-trimester pregnancy losses without an intercurrent term pregnancy) ■

SOURCE: BARGER ET AL, 2003

Clotting cascade

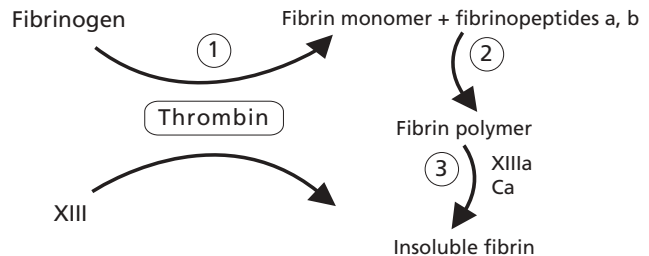
A series of three steps (thus cascade)—both intrinsic and extrinsic—culminate in fibrin clot formation.

1. Thrombin splits fibrinogen into fibrin monomers (single molecules) and into the enzymes, fibrinopeptides a and b.
2. Simple molecules (monomers) of fibrin are polymerized

(formed into a compound) into fibrin strands.

3. Coagulation factor XIII and calcium ions

act together to crosslink the fibrin strands, thus forming fibrin. ■



SOURCE: BROWN AND TOWNE

Diagnosing vasculopathy

Since few patients show any signs or symptoms of vasculopathy it is often hard to diagnose. There are some clinical indicators of a hypercoagulable state, including family history of thrombosis and thrombosis at a young age (generally 50 years old or younger) among other indicators.

Furthermore, when a patient presents with a venous thromboembolism, several factors must be considered to determine whether the patient should undergo an evaluation for vasculopathy (Deitcher et al, 2003).

Before laboratory testing begins, a thorough patient history should be completed to accurately document any previous thrombotic events and to identify any acquired prothrombotic conditions. Secondly, the physician

should ascertain if the patient has any clinical indicators of vasculopathy. If findings are suggestive of vasculopathy, laboratory testing should be performed. Laboratory evaluation, according to Barger and Hurley,

should begin with baseline hematologic and coagulation studies. Testing for acquired and inherited hypercoagulable states uncovers an abnormality in more than 50% of patients presenting with an initial venous thromboembolic event (VTE) but may have minimal actual impact on management in most of these patients, according to Deitcher et al (2003). Furthermore, these tests are expensive and their results are frequently misinterpreted. More importantly, focusing solely on such testing for hypercoagulable states may undermine the

performance of age- and gender-specific cancer screening in patients with idiopathic VTEs.

Once a patient has been identified as a candidate for testing for a hypercoagulable state, testing should be performed in stages. Highest-yield assays (screening tests) should be performed first, and if results come back positive, then appropriate confirmatory tests should follow. If screening test results are negative and sufficient suspicion exists, less common disorders can be tested for, such as factor V Leiden or prothrombin G20210A mutation. ■

Vasculopathy and hypercoagulable states *(continued from page 1)*

congestive heart failure, heparin-induced thrombocytopenia with thrombosis, paroxysmal nocturnal hemoglobinuria, and Behçet's disease (Cleveland Clinic web site).

A patient's hypercoagulable state may be unmasked by many acquired conditions such as immobility due to bed rest following surgery, illness, heart attack or stroke, or trauma. Other acquired risks for hypercoagulability include pregnancy, oral contraceptives, hormone replacement, or

antiphospholipid syndrome also known as "sticky blood", myeloproliferative disorders such as polycythemia vera, thrombocytosis, heparin induced thrombocytopenia as well as cancer. According to a study by Barger and Hurley in September 2000, the recent discovery of several common inherited abnormalities, including factor V Leiden, the prothrombin 20210 mutation, and hyperhomocysteinemia is believed to have helped spark an interest in these conditions.

Although patients with hypercoagulable states are at greater risk for developing a thrombotic event, not all persons will develop an overt thrombosis. Similarly, not all patients presenting with thrombosis have an identifiable hypercoagulable state. In fact, according to Deitcher et al (2003), testing for an inherited hypercoagulable state is likely to uncover an abnormality in more than 60% of patients presenting with idiopathic (i.e., spontaneous or unprovoked) venous thromboembolic events

(continued on page 7)

Treating vasculopathy

Once a patient is properly diagnosed with vasculopathy, anticoagulation is the main form of treatment. In serious situations, both heparin and warfarin may be used, however warfarin is used in less urgent circumstances. Heparin pretreatment should be considered to avoid risk of warfarin necrosis, according to Gottlieb (2000).

Hyperbaric oxygen (HBO) may also benefit patients with wounds related to vasculopathy. As with any wound, once the wound is under control, (e.g., no inflammation, no sepsis, no eschar, no necrosis, no edema, no symptoms,

nor risk to the patient) longer-term control of the ulcer should begin. For hypercoagulable and other microthrombotic ulcers, anticoagulants are essential; HBO therapy acts as a valuable adjunct. Surgery must be considered carefully, and artificial skins are valuable options. Due to risk of pathergy, it is preferable to anticoagulate patients prior to doing any surgery. Re-ulceration is a risk in recently healed wounds, and anticoagulation should continue until the wound and scar are sufficiently matured (Gottlieb, 2000).

If a patient presents with venous thromboem-

bolism with a precipitating cause such as trauma, he/she should be treated with anticoagulation for three months, according to Barger et al, (2000). Meanwhile, the first episode of idiopathic venous thromboembolism in which a precipitating cause is not identified should be treated for at least six months with anticoagulation. Increasingly, evidence suggests that idiopathic venous thromboembolism should be considered a chronic disease, since there is a 25-30% risk of cumulative recurrence after four years (Barger et al, 2000). ■

Symptoms of hypercoagulable states

Information from the Cleveland Clinic web site indicates there are no specific signs or symptoms associated with vasculopathy. The most common clinical manifestation of an underlying hypercoagulable state is lower-extremity deep venous thrombosis with or without pulmonary embolism (Deitcher et al, 2003).

Although most inherited conditions appear to increase only the risk of venous thromboembolic events (VTEs), some of the acquired conditions—including cancer, myeloproliferative syndromes, antiphospholipid antibodies (APA), hyperhomocysteinemia, and heparin-induced thrombocytopenia—have been associated with both VTEs and arterial thrombosis.

According to Barger et al (2000), under normal hemostatic conditions, the relationship between the coagulation cascade (see page 2) and the mechanisms designed to regulate or limit coagulation is

(continued on page 7)

Vasculitis

Cutaneous vasculitis is the inflammation of the blood vessels in the skin. This disorder can affect capillaries, venules, arterioles, and lymphatics, according to a 2003 report by the New Zealand Dermatological Society. Chung and Fiorentino in a 2005 study define cutaneous vasculitis as a histopathologic entity characterized by neutrophilic transmural inflammation of the vessel

wall associated with fibrinoid necrosis.

There are several causes of cutaneous vasculitis, and the disorder has a wide range of clinical presentations depending on the organ systems involved (New Zealand Dermatological Society, 2003). In fact, vasculitis can range in severity from a disease limited just to the skin to more severe, potentially life-threatening

systemic involvement, which is known as systemic vasculitis. Systemic vasculitis can include widespread involvement of internal organs (e.g. renal, heart, lung) and may result in complications such as acute renal failure. Therefore, physicians should learn the symptoms and signs of systemic vasculitis when evaluating patients so that proper treatment can quickly follow. ■

Indications for HBO



Medicare has approved reimbursement for HBO therapy when the following diagnoses are made:

- Actinomycosis
- Acute carbon monoxide intoxication
- Acute peripheral arterial insufficiency
- Acute traumatic peripheral ischemia
- Chronic refractory osteomyelitis
- Crush injuries and suture (reattachments) of severed limbs
- Cyanide poisoning
- Decompression illness
- Diabetic wounds of the lower extremities
- Gas embolism
- Gas gangrene
- Osteoradionecrosis
- Preparation and preservation of compromised skin grafts
- Progressive necrotizing infections
- Soft tissue radiation injuries ■

Symptoms of vasculitis

There are various ways of classifying vasculitis, which typically depend on the size of the vessel involved, clinical and histopathologic features, and etiology (Chung et al, 2005). Examples of large vessels include the aorta and large arteries and veins; medium-sized vessels include the medium- and small-sized arteries and veins; while small vessels include arterioles, venules, and capillaries.

Cutaneous vasculitis can be acute, subacute, or chronic. In all forms, however, the rash usually presents on the limbs—especially the lower limbs. Acute vasculitis may be referred to as allergic or hypersensitivity

vasculitis. It may present with bleeding under the skin known as purpura. Subacute vasculitis starts off less dramatically and usually results in mild purpura, along with wheals, flat red patches, and small bumps, often resembling urticaria. Chronic vasculitis typically presents with macules and papules but purpura and urticaria may be present (Stanway, 2003).

Dermatologic signs of vasculitis are present only when small- and medium-sized vessels are involved (Chung et al, 2005). According to Sa Cabral (n.d.), the small vessels (post-capillary venules) are involved in leukocytoclastic or hypersensitivity or allergic

vasculitis and frequently present as palpable purpura. It also can manifest as urticaria, pustules, vesicles, petechiae, or erythema multiform-like lesions. The medium-size vessels are involved in the polyarteritis nodosa, while the large arteries are involved in giant cell arteritis. Cutaneous signs of the disorder in medium-size vessels include livedo reticularis, ulcers, subcutaneous nodules, and digital necrosis (Chung et al, 2005).

These skin lesions often initially start on the lower legs, back, and buttocks, before becoming generalized. The overall duration and course of these skin

(continued on page 6)

Diagnosing vasculitis

Routine investigations should be performed on the patient to confirm that he/she indeed has vasculitis, but these tests will also rule out other disease processes that mimic leukocytoclastic vasculitis (Chung et al, 2005). These investigations also will help determine the predominant cell infiltrate by skin biopsy; screen for any extra-cutaneous organ involvement; determine systemic nature of vasculitis; help to exclude an offending causative agent such as a drug, and then remove it; and screen for and treat



et al, 2005). Other tests include blood tests (see related chart on page 6); throat swab for culture; urinalysis for proteinuria to ensure there is no occult renal involvement; red blood cell, white cell, and casts; stool for occult

pain, or paresthesias. What's more, a physical examination can help determine whether the vasculitis primarily involves small or medium vessels. Also, if systemic vasculitis is expected, a complete cardiopul-

PHYSICAL EXAMINATIONS ARE THE PRIMARY METHOD OF DIAGNOSING THE EXTENT AND TYPE OF VESSEL INVOLVEMENT.

any underlying disease processes such as infection, connective tissue disease, or malignancy (Sa Cabral, n.d.).

In many cases, however, a vasculitis diagnosis can be made solely based on appearance, where no further tests are needed. (Stanway, 2003) Sometimes a skin biopsy with direct immunofluorescence is performed to confirm the diagnosis. Meanwhile, if medium vessel involvement is suspected, a wedge biopsy may be required (Chung

blood (systemic); chest X-ray; and nerve conduction studies and/or nerve biopsy.

After confirming the diagnosis of cutaneous vasculitis, Chung and Fiorentino report that the clinician must determine the extent and severity of organ involvement. A complete history on the patient will often reveal symptoms of systemic involvement such as sinus congestion, hemoptysis, shortness of breath, hematuria, abdominal

monary, abdominal, and neurologic exam should be performed. If a physician/clinician suspects there is systemic involvement or the cutaneous signs have persisted for more than six weeks, additional laboratory studies should be administered to evaluate for systemic disease. ■

working with hyperbaric oxygen

The Wound Healing Center is staffed with physicians specially trained in hyperbaric medicine. Every precaution is taken to deliver HBO treatment in the safest and most effective manner possible. While HBO is very useful for many patients there are some contraindications.

Hyperbaric physicians take no chances with patients in these circumstances:

- Untreated pneumothorax
- History of spontaneous pneumothorax
- Recent use of cisplatin or adriamycin for chemotherapy
- Any use of bleomycin for chemotherapy
- Current use of disulfiram (Antabuse) if your patient needs >1 treatment

Common side effects from HBO therapy are:

- Idiosyncratic cataract growth
- Transient deterioration of far vision as near vision improves. Converts to baseline after 2-3 months.
- Paresthesia, tingling of the fingertips, can be noticed after 30 treatments. Effect disappears about one month after completing treatment. ■

Vasculitis blood tests



Patients can have blood tests to check liver and kidney function as well as a urine test to look for protein or bleeding. Other blood tests that may be requested include:

- Full blood count and ESR—detects some blood disorders and reflects general health
- Anti-nuclear antibodies (ANA) and extractable nuclear antigens (ENA)—may indicate lupus or other autoimmune disorders
- Anti-streptococcal antibodies—indicates recent streptococcal infection
- Hepatitis B and C serology
- Protein and immunoglobulin electrophoresis—detects blood disorders such as multiple myeloma
- Cryoglobulins—detect abnormal antibodies in the blood that precipitate in the cold ■

SOURCE: NEW ZEALAND DERMATOLOGICAL SOCIETY, 2003

Treatment of vasculitis

The best supportive treatment for cutaneous vasculitis is sufficient bed rest; the avoidance of trauma; leg elevation; avoiding tight clothing; and keeping warm (Chung et al, 2005). Anti-inflammatory agents, such as topical steroids and non-steroid anti-inflammatory drugs (e.g. indomethacin), are often prescribed. Meanwhile, antiplatelet agents such as aspirin or dipyridamole are sometimes used (Sa Cabral, n.d.). For chronic or persistent skin-limited vasculitis, dapsone and/or colchicine may be effective.

Approximately 50% of patients with cutaneous vasculitis will have a treatment-associated condition, such as infection, inflammatory disease, or malignancy, report Chung and Fiorentino. Anti-viral therapy is good for patients with known viral-associated vasculitides such as HCV-associated CV and HBV-associated PAN.

For the more severe systemic vasculitis, such as in the ANCA-associated vasculitides, therapy should include prompt treatment with high dose

corticosteroids in combination with immunosuppressives such as cyclophosphamide which are required to reduce morbidity and can save lives. (Sa Cabral, n.d.) According to Chung and Fiorentino, intravenous immunoglobulin or plasmapheresis may be useful in the treatment of severe, refractory vasculitis on patients who have contraindications to traditional immunosuppressive therapy. ■

Symptoms of vasculitis (continued from page 4)

lesions depend on the etiology and, in an acute case, could resolve within a few days to a few weeks or persist for months or even years, if the case is chronic and recurrent (Sa Cabral, n.d.).

Approximately 50% of cases of vasculitis are primary disease (idiopathic). Meanwhile, some of the major secondary causes of cutaneous vasculitis seen in small and medium vessel vasculitis include infection (15-20% of patients); drugs (10-15%); and malignancy (<5%). Patients with streptococcus, hepatitis B and C, or such infectious

agents as bacteria, parasites, and fungi can also be associated with the disease and can present with associated cutaneous vasculitis. Also, various inflammatory diseases such as rheumatoid arthritis, systemic lupus, erythematosis, and inflammatory bowel disease (IBD) can also present with associated cutaneous vasculitis (Chung et al, 2005).

Cutaneous vasculitis can also be associated with such drugs as aspirin, penicillins, sulfonamides, thiazides, and oral contraceptives; chemi-

cals such as insecticides, petroleum; and foodstuff allergens like milk products or gluten. It can also be associated with malignancy (typically a paraproteinemia or lymphoproliferative disorder) and abates with the underlying cancer treatment (Chung et al, 2005). ■

Vasculopathy and hypercoagulable states *(continued from page 3)*

(VTEs). Although the remaining 30% to 40% will have unremarkable test results, this cannot rule out a complete absence of vasculopathy. Some of these individuals may have an acquired condition such as cancer or antiphospholipid antibodies (APA), which interfere with the normal function of blood vessels, while others may have a disorder or defect that has not yet been discovered or properly characterized. Prior to 1993 (before the discoveries of factor V Leiden and the prothrombin G20210A

mutation), an inherited predisposition to hypercoagulability was identified in only 15% to 20% of patients presenting with idiopathic VTEs (Barger et al, 2000).

Regardless if the hypercoagulable state is inherited or acquired, the predisposition to venous thrombosis, including upper and lower extremity deep venous thrombosis with or without pulmonary embolism, cerebral vein thrombosis, and intra-abdominal venous thromboses, or arterial thrombosis, including

myocardial infarction, stroke, acute limb ischemia, and splanchnic ischemia is a major concern for patients and medical practitioners alike. (Deitcher, 2003). In fact, most hypercoagulable states are associated with a spontaneous onset of deep venous thrombosis at an early age. What's more, in 80–90% of people with a thrombosis, a cause can be found, while in 50–80% of these, a hereditary or acquired defect is the culprit, report Brown and Towne in a recent study. ■

Symptoms of hypercoagulable states *(continued from page 3)*

complex. Coagulation is a result of the sequential activation of several serine proteases that ultimately generate thrombin at the site of vascular injury. Circulating anticoagulants that function to limit thrombus formation include antithrombin, activated protein C (APC), and plasmin.

Antithrombin, a serine

protease inhibitor, binds heparin and inactivates a number of serine proteases. Thrombomodulin, an endothelial cell surface protein, binds thrombin and alters its substrate specificity, thus allowing it to convert protein C to APC. APC exerts its anticoagulant effect by proteolytically cleaving and inactivating the activated forms of factors

V and VIII (Va and VIIIa, respectively). Protein S is a cofactor required for protein C activity. Although 60% of protein S circulates bound to a carrier protein, only free protein S has cofactor activity. Plasmin arises from plasminogen and acts as the major fibrinolytic enzyme. ■

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Heparin vs. warfarin



Persons with protein C-related thrombosis are usually treated with the anticoagulant heparin. The standard treatment for acute DVT is with unfractionated heparin for five days by continuous intravenous infusion, with warfarin started on day one or two and continued for three months. Heparin is continued until the international normalized ratio (INR) is within the therapeutic range for two days.

Since there is a risk of recurrence with such patients, long-term therapy with warfarin or low molecular weight heparin is often prescribed. Since the response to warfarin varies, each patient is monitored to prevent underdosing or overdosing. ■

QUESTIONS OR COMMENTS?

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8

Working with a Wound Healing Center

Underlying diseases, like vasculopathy, can cause skin disorders that require a wound specialist's attention. While treating these types of wounds, physicians at Wound Healing Centers work with the primary care physician and other specialists to resolve the underlying disease for their patients.

Specifically, Wound Healing Centers are uniquely equipped to heal necrotic ulcers caused by vascular disorders. Patients with the follow-



ing clinical presentations will benefit from the advanced wound care available at your local Wound Healing Center:

- Cholesterol crystal embolization.
- Cryoglobulinemia—Fifty percent of patients with this disorder will develop vasculitis. Patients with this disorder also

can develop leukocytoclastic vasculitis.

- Calciphylaxis—This life-threatening disorder involves the widespread calcification of small- and medium-sized vessels, which can lead to occlusion, thrombosis, and tissue necrosis.
- Thrombophilias. ■

CONSIDER REFERRING YOUR PATIENTS TO A WOUND HEALING CENTER FOR ADVANCED WOUND CARE IF:

- Your patient has a wound that persists for more than 30 days with conservative treatment
- Your patient has a wound and Reynaud's phenomenon
- Your patient has purpura
- Your patient has a wound and hypertension
- Your patient has gangrene or necrotic tissue in a wound
- Your patient has a wound and diabetes



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