Vascular Emergencies

Expert Management for the Emergency Physician

EDITED BY:
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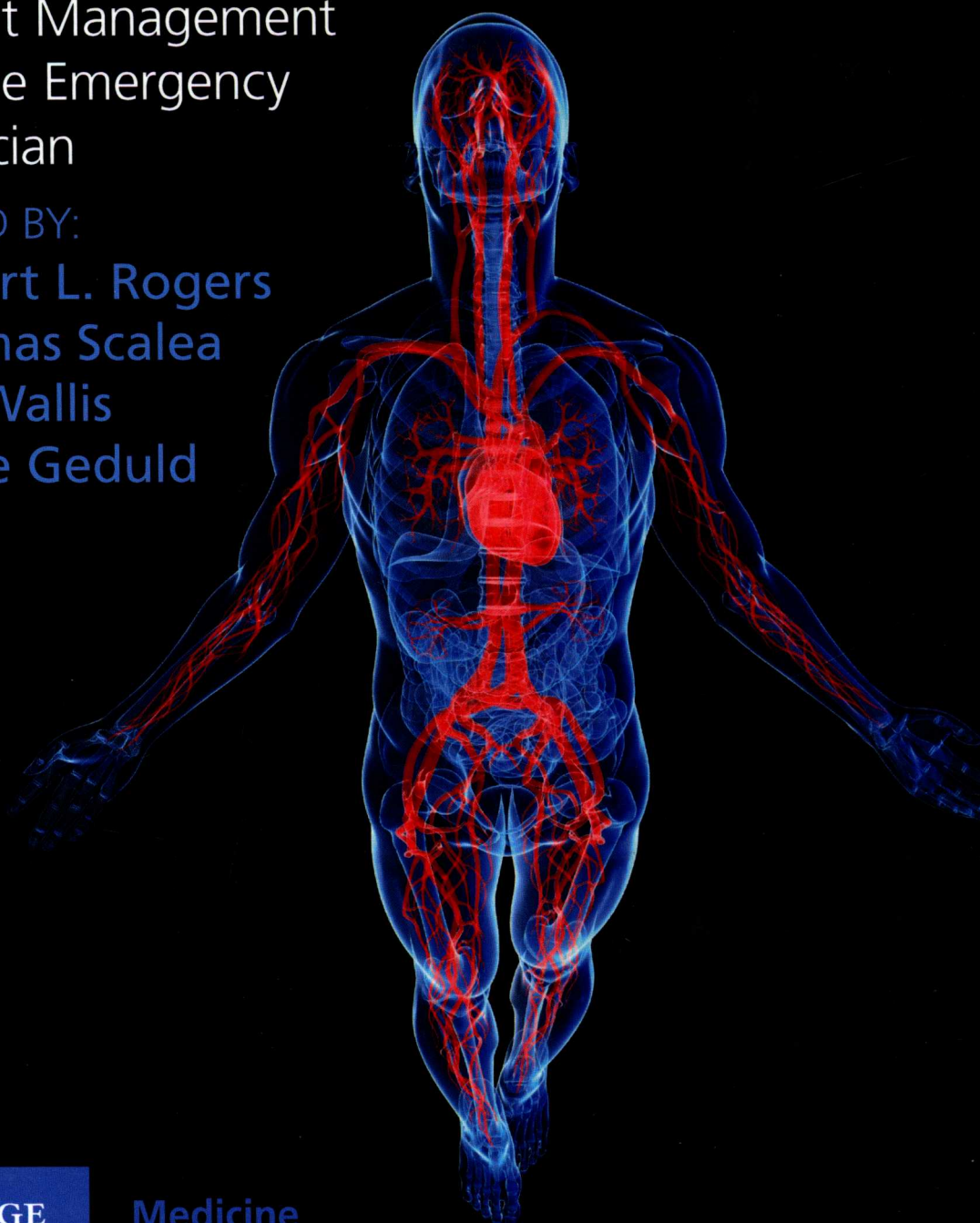

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Vascular Emergencies

Expert Management for the Emergency Physician

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Preface

Vascular emergencies are common in the practice of emergency medicine, and emergency care providers will no doubt encounter these entities on a day-to-day basis in the emergency department. Of all of the clinical entities in the house of medicine, vascular emergencies typically are the most time sensitive, and the patients with these conditions tend to be the sickest. Vascular emergencies by their very nature are limb and life threatening, and emergency physicians and other acute care providers should be expert in the care and disposition of this group of patients in order to ensure an optimal outcome. This book, developed by emergency physicians, vascular surgeons, and trauma surgeons who know what it is like to see patients day in and day out, focuses on the acute presentation of vascular emergencies in the emergency department. The overall aim of the book is to provide practical, useful information that will allow for the delivery of excellent medical care.

Most books covering the topic of vascular emergencies are usually aimed at surgeons and tend to focus on in-depth surgical management and other inpatient issues. This book has been written for you – the practicing emergency physician. What you do on a day-to-day basis truly does matter, and it is our hope that this book helps you better care for your patients.

Acknowledgments

I would like to thank my wife, Tricia, and my wonderful children, Harrison and Gabriella, for tolerating me while I put this very important book together. They are my sole inspiration.

To Linda Kesselring, who helped me to organize my cluttered brain and develop a truly wonderful book that is aimed at helping emergency physicians throughout the world.

To our patients, who teach us every day that we still have so much to learn.

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Chapter 1

Cerebral venous sinus thrombosis

Jonathan C. Roberts and Christopher M. Fischer

Cerebral venous sinus thrombosis (CVST) is an uncommon cause of stroke. Arterial strokes are much more common. CVST more commonly affects younger adults and children and can be associated with significant rates of morbidity and mortality.

Diagnosing CVST requires a high index of suspicion, as the presenting symptoms can be highly variable and their onset can be subacute. Few large clinical investigations of CVST have been performed; therefore, much controversy and misunderstanding persist surrounding its clinical presentation, diagnosis, and management.

Anatomy

The cerebral venous sinus network, a series of vascular channels suspended within the dura mater, drains cerebrospinal fluid (CSF) and venous blood from the brain into the systemic venous circulation. CSF bathes the central nervous system (CNS) and provides the brain and spinal column with a protective cushion. CSF is produced by the choroid plexus and modified ependymal cells found within the lateral third and fourth ventricles. It flows through a ventricular system into the subarachnoid space, where it eventually passes through arachnoid villi into cerebral venous sinuses. Venous blood also drains from deeper cortical veins into this same sinus system.

The major cerebral venous sinuses are the superior and inferior sagittal sinuses, the straight sinus, the transverse sinuses, the sigmoid sinuses, the cavernous sinuses, and the superior and inferior petrosal sinuses. Disruption of this vascular sinus network can impair venous and CSF outflow, causing hydrocephalus and catastrophic changes in cerebral perfusion pressure, along with possible infarction and hemorrhage.

Pathophysiology

CVST is a rare form of stroke, caused by a blockage of this drainage system. Thrombosis may be confined to the large cerebral venous sinuses or may involve the deeper cortical veins. Isolated thrombosis of a large cerebral venous sinus impairs venous drainage and CSF absorption, which results in an elevation of intracranial pressures. This can cause abrupt alterations in consciousness and impaired vital functions by affecting perfusion of the brainstem. Thrombosis of draining cortical veins results in cytotoxic and vasogenic edema with possible subsequent capillary rupture, hemorrhage, and infarction from impaired perfusion.

If collateral circulation remains intact, patients may present with extremely subtle symptoms and physical examination findings. Thrombus progression to multiple venous sinuses and cortical veins with impaired collateral circulation may cause more focal neurologic signs, seizures, and altered mental status.[1,2]

Epidemiology and risk factors

CVST has an annual incidence of two to four per million[3] and an overall mortality rate approximating 8%.[4] Although the exact incidence is unknown, CVST is thought to account for 1 to 2% of all strokes. While arterial stroke tends to affect older patients, with a slight male predominance,[5] CVST affects predominantly younger patients, with peaks in childhood and in the fourth and fifth decades of life, and with a 3 : 1 female predominance.[4]

There are numerous risk factors for the development of CVST (Table 1.1). Genetic or acquired prothrombotic states are found in more than 85%

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Table 1.1 Causes of and risk factors for development of cerebral venous sinus thrombosis

Medications
Oral contraceptives
Genetic prothrombotic tendencies
Factor V Leiden mutation
Protein C and protein S deficiency
Antithrombin deficiency
Prothrombin mutation
Acquired prothrombotic states
Pregnancy and peripartum period
Nephrotic syndrome
Dehydration
Hematologic conditions
Polycythemia
Thrombocytosis
Leukemia
Mechanical causes
Head trauma
Neurosurgical procedures
Lumbar puncture
Infection
Mastoiditis, otitis, sinusitis
Meningitis
Inflammatory disease
Inflammatory bowel disease
Systemic lupus erythematosus
Sarcoidosis

Adapted from Stam.[1]

of patients with CVST.[1,6] Oral contraceptive use is the most commonly identified risk factor for CVST; several case-control studies demonstrated a greater than 10-fold increase in risk among women using those medications.[7,8] Infections, including otitis media and mastoiditis, may also predispose individuals to CVST. Trauma can disrupt the normal vascular channel anatomy and lead to thrombosis. The risk of CVST increases during pregnancy and the immediate postpartum period.[1] Other risk factors include disease processes that induce a prothrombotic state. These include factor V Leiden mutation, protein S and C deficiency, antithrombin III deficiencies, inflammatory bowel disease, and malignancy. Despite extensive evaluation, no clear cause is identified in approximately 20% of cases. Case reports have described rare associations between CVST and paroxysmal nocturnal hemoglobinuria,[9] heparin-induced thrombocytopenia,[10] and even lumbar puncture.[11]

Clinical presentation

CVST is a very rare form of stroke with variable presentations. The variable nature of the symptoms and the subtle presentation can often delay diagnosis. In one study, investigators found the median delay between symptom onset and diagnosis of CVST was seven days.

History

Headache is the most common presenting symptom of CVST, present in more than 75% of patients. The nature of the headache can be highly variable and is not helpful in distinguishing CVST from other causes of headache. Up to 15% of patients may report sudden onset of a thunderclap headache more typical of subarachnoid hemorrhage.[12] Other neurologic symptoms may be associated with increased intracranial pressure and include dizziness, nausea, and visual disturbances. Focal or generalized seizures may be part of the initial presentation and occur in as many as half of patients.[13] Seizures may be even more common in peripartum women with CVST.[14] Other possible focal neurologic symptoms include focal weakness, sensory deficits, aphasia, and visual field deficits. Coma and stupor may result from dramatic increases in intracranial pressure and are a poor prognostic sign.

CVST can mimic other conditions. The combination of headache, visual disturbances, and papilledema can also be found in idiopathic intracranial hypertension (pseudotumor cerebri). In one study, 10% of 106 patients diagnosed with presumed idiopathic intracranial hypertension were ultimately found to have CVST.[15]

Physical examination

Careful neurologic examination is important to elicit the sometimes subtle findings that can be associated with CVST. Cranial nerve examinations may demonstrate papilledema, nerve palsies, or visual field deficits. Focused neurologic examination may elicit focal weakness or signs that accompany increased

intracranial pressure, including gait instability and abnormal reflexes.

Diagnosis

Diagnosing CVST depends on a high index of suspicion and appropriate imaging studies. Imaging may also provide prognostic information for patients with CVST. The extent of venous sinus involvement and the presence of intraparenchymal hemorrhage may correlate with functional outcome.[2]

Non-invasive imaging

Unenhanced brain computed tomography (CT) is useful in identifying secondary signs of CVST, including hemorrhagic infarction, brain edema, mass effect, hydrocephalus, subdural effusion, and subarachnoid hemorrhage. It may also infrequently identify primary signs of CVST, including the dense triangle sign and the cord sign. The dense triangle sign or delta sign represents a hyperintense thrombus within the superior sagittal sinus and may be visible on axial images through the superior sagittal sinus. The cord sign represents a hyperintense thrombus within a deeper cortical vein.[16] However, only 25% of patients with CVST demonstrate these signs on unenhanced brain CT.[17] The most common finding on an unenhanced CT scan is a non-arterial distribution of areas of hemorrhage – this finding represents the venous congestion resulting from venous thrombosis.

Enhanced brain CT has emerged as a possible alternative to magnetic resonance imaging/magnetic resonance venography (MRI/MRV) for non-invasive diagnosis of CVST. Computed tomography venography (CTV) imaging protocols use intravenous contrast to highlight draining cortical and dural venous sinuses and can thus readily identify filling defects. The empty delta sign represents an occlusive thrombus within the venous sinus, which prevents contrast-mediated enhancement, and is found in 30% of patients with known CVST.[17] Disadvantages of CTV include exposure to ionizing radiation, exposure to iodinated contrast, and hyperdense bony artifact requiring digital subtraction techniques for optimal image quality. Many authors suggest that CTV's sensitivity and specificity for identifying CVST are equivalent to those for the more time-consuming magnetic resonance (MR) techniques.

Unenhanced brain MRI is more sensitive than unenhanced brain CT in identifying CVST. Using intravenous contrast and time-of-flight techniques, enhanced brain MRV is able to reliably detect alterations in cerebral venous flow, identifying CVST with an overall sensitivity and specificity similar to those for CTV. In addition, MR techniques may be able to identify deeper cortical vein thrombus more readily than CT.[18]

A proposed CVST management algorithm published by the American Heart Association and the American Stroke Association in 2011 recommends T2-MRI + MRV as the initial diagnostic modality of choice. CT + CTV is the preferred alternative if MR is not readily available or is contraindicated.[18]

Invasive imaging

Cerebral angiography provides detailed images of the deep cortical veins and cerebral venous sinus network. It can serve as an alternative imaging modality when CT and MR prove inconclusive or are unavailable.

Additional diagnostics

Many patients with the symptoms of CVST undergo lumbar puncture during their initial evaluation. The most common finding on lumbar puncture is an elevated opening pressure (>20 cm H_2O), which is found in more than 80% of cases. In addition, the diagnosis of CVST should be entertained if an elevated opening pressure is encountered during the workup of the headache patient. This may very well be the one clue that leads to the diagnosis.

Treatment

Treatment of CVST can involve multiple approaches, including systemic anticoagulation, chemical or mechanical endovascular thrombectomy, and surgical decompression or open clot retrieval.[19]

Few studies have investigated the safety and efficacy of systemic anticoagulation for CVST. Concern that this procedure can precipitate hemorrhage or exacerbate pre-existing hemorrhage in CVST creates barriers to aggressive and effective treatment. This unproven risk of progressive hemorrhage must be weighed against the real risk of withholding anticoagulation and thus promoting venous infarction with hemorrhagic conversion.[20]

Two randomized trials have evaluated systemic anticoagulation in CVST. They were combined in a Cochrane Review meta-analysis,[3] which found a

non-significant trend toward reduced death and disability (relative risk [RR] 0.33 and 0.46, respectively) in patients treated with systemic anticoagulation. No cases of spontaneous or progressive hemorrhage were documented.

The best available evidence supporting the safety and efficacy of systemic anticoagulation for CVST is based on observational cohorts of undifferentiated CVST patients (including those with intracerebral hemorrhage).[3] The International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT) CVST cohort (624 patients known to have CVST and treated with systemic anticoagulation) demonstrated a non-significant reduction in death and disability (12.7% and 18.3%, respectively). The rates of spontaneous and progressive hemorrhage did not increase.

Additional studies support systemic anticoagulation as a safe and effective therapeutic approach for patients with CVST in the emergency department (ED). However, given the rarity of this condition, all clinical trials conducted thus far are underpowered to establish statistical significance.[19]

Not all patients respond to systemic anticoagulation, so alternative methods to re-establish normal outflow have been explored. Systemic or localized fibrinolysis has been evaluated in small trials and has yielded mixed results. At best, thrombolytics provide a safe and effective alternative for CVST that is resistant to systemic anticoagulation. At worst, they might increase the risk of spontaneous or progressive hemorrhage. A handful of small studies have investigated mechanical thrombectomy as an alternative to systemic or localized chemical thrombolysis. Balloon angioplasty, stenting, clot maceration, and rheolytic thrombectomy are promising alternatives for CVST resistant to systemic anticoagulation and might be associated with reduced rates of hemorrhage compared with chemical thrombolysis. Surgical intervention via intracerebral pressure monitor placement or hemicraniectomy for hematoma evacuation may be indicated for management of elevated intracranial pressure.[19]

Multiple studies have evaluated risk factors for and prognostic implications of early seizures in CVST. A prospective observational study of 194 patients found a threefold increase in the mortality rate among patients with CVST who experienced early seizure.[21] A second prospective observational study found that supratentorial hemorrhage, seizures at the time of presentation, and motor deficits were predictive of subsequent seizure activity and clinical deterioration.[22] The use of antiepileptic drugs may be indicated in this subgroup of CVST patients; however, no studies have demonstrated that their use reduces the morbidity or mortality rate.

Clinical approach when resources are limited

In most cases, CVST can be diagnosed definitively only with the use of advanced imaging modalities, including unenhanced CT, contrast CT, MRI, and magnetic resonance angiography (MRA). When these advanced modalities are not available, the diagnosis might be suspected but cannot be confirmed easily. The diagnosis should be considered in patients with focal neurologic deficits and risk factors for CVST, especially those who are using oral contraceptives or have a genetic predisposition for thrombosis. Close coordination with consulting neurologists, when available, should be part of the management of these patients.

Pearls and pitfalls

- Consider CVST in patients with headache, neurologic findings, and risk factors.
- Consider CVST in patients with a history suggestive of pseudotumor cerebri (idiopathic intracranial hypertension).
- Anticoagulation should be considered in patients with CVST even if there is concurrent hemorrhagic infarction.

Critical actions

- CVST can be associated with subtle symptoms and a subacute onset. Knowledge of the epidemiology and risk factors for CVST can help raise suspicion for this often-overlooked diagnosis.
- Prompt attention to airway, breathing, circulation (ABCs) in patients who present with stupor or coma is essential.
- A thorough neurologic examination is important to elicit findings that indicate CVST (focal weakness, sensory deficits, aphasia, papilledema, visual field deficits).
- Recognition of a non-arterial distribution of hemorrhage, caused by venous congestion, on a brain CT scan suggests the diagnosis and should prompt further investigation and consultation.

- Anticoagulation should be initiated promptly in appropriate patients.
- If a patient does not respond to systemic anticoagulation, more invasive options should be considered.

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Chapter 2

Acute ischemic stroke

Cemal B. Sozener and Phillip A. Scott

Stroke is the third leading cause of death and the leading cause of disability in the United States. In addition, it is an extremely common condition around the world and has become a global health threat, along with diabetes and heart disease. Ischemic stroke afflicts more than 795,000 Americans each year, 610,000 of whom have a first attack. Stroke accounts for 1 in every 18 deaths in the United States, or just over 130,000 deaths, per year.[1] An additional 200,000 to 500,000 Americans experience transient ischemic attack (TIA). *Stroke* refers to any disease process that alters blood flow to a focal region of the brain. This chapter focuses on ischemic causes, which account for 87% of strokes. Timely diagnosis and appropriate management of acute ischemic stroke (AIS) and TIA can reduce morbidity and mortality rates.

Pathophysiology

Ischemic strokes are generally divided into three major categories: thrombotic, embolic, and hypoperfusion associated (watershed strokes). A substantial portion of strokes (30–40%) defy etiologic categorization and are considered cryptogenic. Vasculitic causes of stroke include Takayasu's arteritis, systemic lupus erythematosus, and polyarteritis nodosa. Hypercoagulable states and arterial dissection may also result in cerebral infarction.

Thrombotic strokes, the most common subtype, are characterized by a narrowing of the vascular lumen, typically as a result of atherosclerotic disease, with subsequent platelet adhesion and local clot formation. Thrombosis occurs most commonly in the internal carotid and the middle cerebral and basilar arteries. Clinically, thrombotic stroke symptoms may wax and wane in severity depending on direct flow as well as collateral circulation to the affected tissue.

Embolic strokes, the second most common subtype, account for approximately 20% of ischemic strokes. They result from the release of material into the vascular lumen, which travels distally to occlude a cerebral vessel. The majority of these emboli are cardiac in origin, either from mural thrombi (arising from untreated cardiac dysrhythmias or myocardial infarction) or valvular abnormalities. Other causes include paradoxic emboli (related to right-to-left shunting in patients with a patent foramen ovale or other septal defect), artery-to-artery embolization (resulting from the migration of proximal clots from atherosclerotic disease in larger vessels), or emboli from fat (fractures) or air (injection, diving). Embolic strokes are typically abrupt in onset and manifest maximal deficits early.

Hypoperfusion-related strokes, the least common type of ischemic stroke, are typically the result of systemic disease from cardiac failure, causing diminished blood flow to watershed regions of the cerebral vasculature. The neurologic symptoms of this type of infarct are more diffuse than those associated with strokes of other cause and correlate with the magnitude of reduction in blood pressure.

Epidemiology

In 2006, more than 6.4 million American adults had a stroke.[1] At younger ages, the incidence of stroke is higher among men than women; this trend is reversed in older age groups. Blacks have a higher incidence than whites, particularly in younger populations. The stroke-related mortality rates in the two racial groups are similar. Mexican Americans have a higher incidence of stroke than non-Hispanic whites.[2] Among patients $\geq$65 years of age, stroke has a 30-day mortality of 8.1%.[3]

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