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Neuroradiologic Aspects of Cerebral Disseminated Intravascular Coagulation

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Six patients with neurovascular disseminated intravascular coagulation (DIC) illustrate the diversity of neuroradiologic changes in that condition. Emphasis is placed on the involvement of large cerebral vessels, especially as reflected by cerebral vein or sinus occlusions. The latter occurred in four of the six cases reported; deep or superficial veins, larger lobar draining veins such as the superficial middle cerebral vein, or large bore vessels such as the superior sagittal sinus or jugular bulb were involved. Radionuclide sinography or computed tomography (CT) was useful in revealing large vessel occlusions or their sequelae; in two cases CT showed focal hemorrhage in an unusual location. Although DIC has classically been envisioned as occurring within the arterial microcirculation, these cases attest to the occurrence of large vessel, especially venous, involvement in neurovascular DIC.

DIC is a pathophysiologic reaction occurring in an ever increasing number of disease states (fig. 1). It is triggered by a variety of mechanisms resulting in the presence of thrombin in the systemic circulation. The acute fulminant syndrome is readily recognized, and the diagnosis established, through characteristic laboratory findings. These include decreased platelets and fibrinogen, and increased prothrombin time, usually accompanied by abnormalities in tests for fibrinolysis such as thrombin time, euglobulin clot lysis time, fibrin degradation products, and/or changes in the peripheral smear or clotting factors.

Although DIC usually occurs within the arterial microcirculation and is thus distinguishable from thromboembolic disease involving major vessels [14], an unusual but important manifestation includes both arterial and venous large vessel thromboses [5, 15]. If those vessels are in the central nervous system, the prognosis is poor [4, 10, 16, 17]. Thus, a high index of suspicion and improved methods of diagnosis should permit earlier recognition of neurovascular DIC, which hopefully would contribute to earlier and more effective therapy.

In a review of charts for 3½ consecutive years at our hospital, 98 patients with DIC were found; 74 patients died (75.5%). Nine of the 55 autopsied cases with DIC had cerebral dysfunction as a preterminal event and had some form of neuroradiologic investigation with positive findings in five. These five patients and one older case from our files serve as the basis of our report. We illustrate the diversity of neuroradiologic changes in DIC and draw attention to the presence of large cerebral vessel, particularly venous, involvement in this condition.

Case Reports

Case 1

A 4-year-old boy was admitted for operative treatment of tetralogy of Fallot. At 2 days after operation, he began having episodic fever, pneumonia, vomiting and poorly defined

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- A. Infections [2, 3]
1. Viral: herpes, rubella, cytomegalic inclusion disease, others
 2. Rickettsial: Rocky Mountain spotted fever, others
 3. Bacterial
 - a. Gram-negative septicemia
 - b. Gram-positive septicemia, especially in asplenic patients
 4. Plasmodium falciparum malaria
- B. Obstetrical and neonatal complications
1. Abruptio placentae
 2. Septic abortion and chorioamnionitis
 3. Amniotic fluid embolism
 4. Intrauterine fetal death
 5. Severe toxemia
 6. Severe respiratory distress syndrome
 7. Others: degenerating hydatidiform mole and fibromyomata, intraamniotic saline injections, abdominal pregnancy, tetracycline-induced hepatorenal failure, fetomaternal blood transfusion
- C. Neoplasms [4]
1. Carcinoma: prostate, pancreas, breast, lung, stomach, colon, ovary, diffuse metastases, others
 2. Acute leukemia: stem-cell, promyelocytic, others
 3. Sarcomas: rhabdomyosarcoma, neuroblastoma, others
- D. Vascular [5]
1. Vascular malformations: giant hemangioma: Kasabach-Merritt syndrome, aneurysms of the aorta and other large vessels, cyanotic congenital cardiac lesions
 2. Collagen vascular disorders: acute vasculitis, polyarteritis, systemic lupus erythematosus
 3. Hypoxia and hypoperfusion: congestive heart failure with pulmonary emboli [6], cardiac arrest, shock, hypothermia
- E. Massive tissue injury: trauma, including cerebral [7-9], extensive burns, heat stroke, fat embolism, surgical complications (surgical procedures involving extracorporeal circulation, prostatic surgery, procedures involving extensive manipulation of lung)
- F. Miscellaneous: diabetic acidosis [10-13], amyloidosis, snake bite, anaphylaxis, drug reactions, purpura fulminans, Cushing disease, acute pancreatitis

Fig. 1.—Disorders associated with disseminated intravascular coagulation (modified from Wintrobe et al. [1]).

pain in the legs and feet. One month later he suffered two focal motor seizures involving the left foot. Shortly thereafter, he experienced a third seizure that began in the left foot but spread to the left hand and arm. A radionuclide brain scan 4 hr later revealed increased uptake in the right hemisphere. Cerebral angiography demonstrated only hydrocephalus in the arterial phase. However, in the venous phase, there was occlusion of subependymal veins, most cortical veins, and the major dural sinuses (fig. 2). Pneumoencephalography revealed minimal hydrocephalus and no evidence of a mass lesion. Examination of the cerebrospinal fluid obtained at pneumoencephalography revealed xanthochromic fluid containing 233 red blood cells of which 10% were crenated, 90 white blood cells of which 92% were polymorphonuclear cells, 320 mg/dl protein, and 66 mg/dl glucose. Coagulation studies showed progressive drop of platelets and fibrinogen, with a prolongation of prothrombin and partial thromboplastin time; fibrinogen split products were markedly positive. Early the next morning, the patient was comatose with bilateral fixed and dilated pupils, and he exhibited numerous focal left-sided seizures. He died 2 days later.

Autopsy revealed the tetralogy of Fallot, Gram-negative coccid pericardial empyema, and massive aspiration pneumonitis. There was thrombophlebitic thrombosis of the innominate and left jugular veins with thrombosis of all the major dural sinuses, all the major superficial and deep cerebral veins, and many smaller and branch veins. Some of these thromboses were associated with foci of subarachnoid hemorrhage, others with hemorrhagic infarctions of the thalami, lenticular nuclei and lateral geniculate bodies. The thrombus in the left lateral sinus showed the most advanced stages of organization. The arterial system was intact throughout the body.

Case 2

An obese 37-year-old woman complained of generalized headaches and weakness. On admission elsewhere, she was found to have a severe Coombs-positive hemolytic anemia and was placed on steroids; during that hospitalization, she also suffered pulmonary

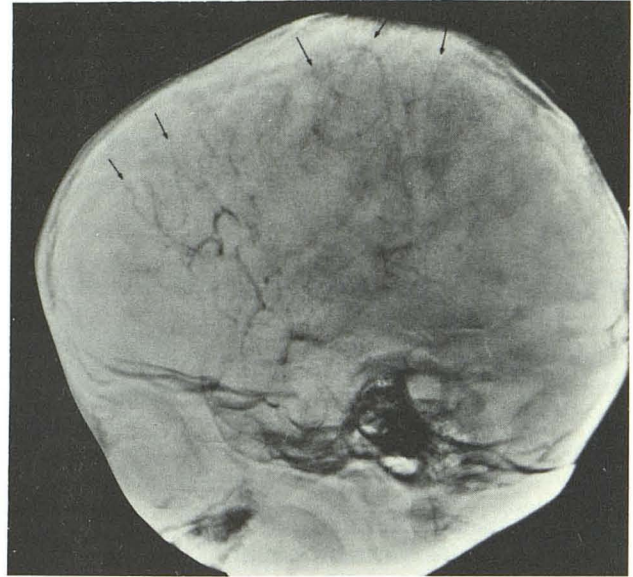


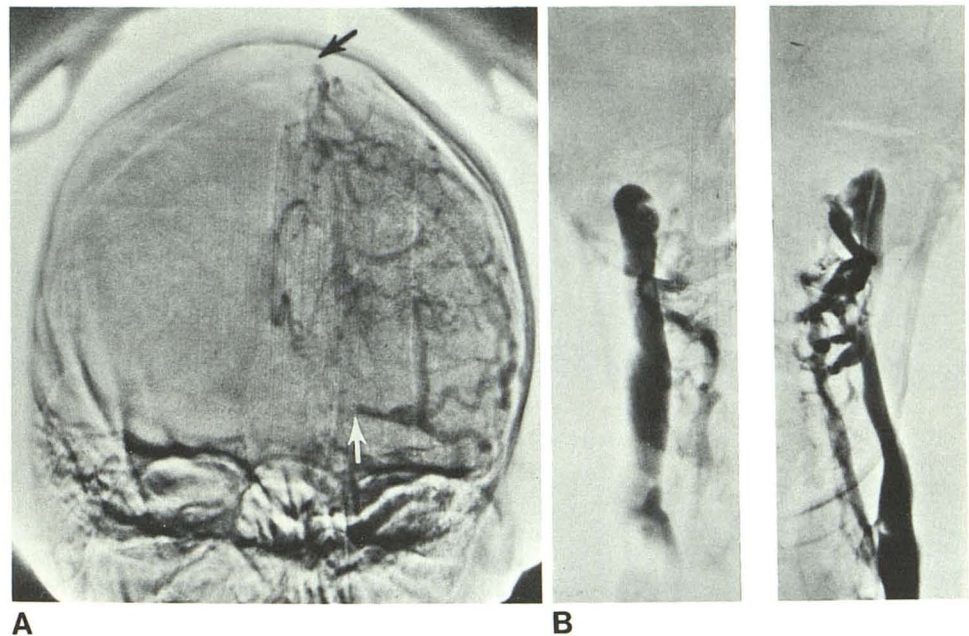
Fig. 2.—Case 1. Massive cerebral venous thrombosis. Lateral angiogram, venous phase, 9.75 sec after contrast reached brain. Occlusions of cortical veins (arrows); subependymal veins and major dural sinuses did not opacify.

infarcts confirmed by radionuclide lung scan for which she was treated with anticoagulants. Three months later, she developed severe occipital headaches, nausea, and epigastric pain accompanied by vomiting and progressive weakness. On admission to our hospital, she complained of severe headache, stiff neck, generalized weakness, and abdominal discomfort. On examination she was noted to be severely obese with hepatosplenomegaly. Neurologic examination revealed nuchal rigidity, bilateral papilledema with a flame hemorrhage in the left eye, and bilateral Hoffman signs. Laboratory studies were compatible with hemolytic anemia. The patient was given intravenous steroids and low dose mannitol. Chest and skull radiography, midline echoencephalography, and radionuclide dynamic and static studies were all within normal limits. Early on hospital day 2, the patient began vomiting, followed by confusion and disorientation. Emergency arteriography revealed filling defects with the superior sagittal and transverse sinuses (fig. 3A). Bilateral selective retrograde jugular venography confirmed sinus thromboses (fig. 3B). A right frontal subdural pressure monitor was placed; initial cerebrospinal fluid obtained with a pressure of 40 mm Hg. Coagulation studies showed progressive drop in platelets, prolongation of the prothrombin and partial thromboplastin times, and an increase in fibrin split products. The patient was anticoagulated with heparin, transfused with packed red cells, and treated with cyclophosphamide, steroids, and mannitol. The next morning she was comatose, responding only with decerebrate posturing on the left, semipurposeful movements on the right, and bilateral Babinski responses. She had spiking temperatures to 40°C, followed by hypothermia. She entered intractable status epilepticus. Platelets continued to decrease, with prolongation of prothrombin time and partial thromboplastin time. She died 9 days after admission. Autopsy was not permitted.

Case 3

A 17-year-old boy with a long history of right leg deep venous thrombotic episodes was transferred to our hospital after sustaining

Fig. 3.—Case 2. Complete dural sinus thrombosis in DIC. **A**, Oblique angiogram, later venous phase. Occlusion of superior sagittal sinus (*black arrow*) and transverse sinus (*white arrow*). **B**, Composite of left and right selective retrograde jugular venography with catheter in jugular bulb. No filling of dural sinuses. Catheterization above bulb was attempted without success.



a generalized motor seizure and remaining stuporous with left hemiparesis 11 days after successful right femoral thrombectomy. He had left hemiparesis, bilateral clonus, and aphasia. He rapidly deteriorated, lapsed into coma, dilated and fixed his left pupil, and exhibited decorticate posturing on the left, decerebrate posturing on the right, and continuous tonic-clonic activity of the left limbs. CT revealed a left frontal hematoma (fig. 4).

Subtemporal decompression and a left craniotomy were performed with aspiration of clotted blood and necrotic, hemorrhagic brain. Postoperatively his coagulation profile was normal. His scalp wound continued to ooze. On hospital day 3 he suffered a pulmonary embolus; the placement of a Mobin-Uddin umbrella was unsuccessful. Repeated coagulation studies revealed progressive decrease of platelets, prolongation of the prothrombin time, and elevation of the partial thromboplastin time. He died 3 days later.

In addition to extensive systemic venous thrombosis, autopsy revealed thrombosis of the superior sagittal sinus and of various superficial frontal cerebral veins. This was accompanied by considerable subarachnoid blood, bilateral frontal foci of cortical hemorrhagic infarctions, and a large cavitory, hemorrhagic necrotic area involving both cortex and white matter on the left.

Case 4

A 65-year-old woman had been treated with salicylates and physical therapy for left saphenous thrombophlebitis 2 months earlier. One week before her neurologic problems, she had been examined because of dark brown vaginal bleeding and a left adnexal mass. She developed aphasia and was hospitalized elsewhere. The next morning she developed a severe headache and fever of 38.4°C. Lumbar puncture revealed an opening pressure of 215 mm H₂O, 250 red blood cells and 5 white blood cells. Her level of consciousness decreased progressively over the next 2 days; she developed right upper extremity decorticate posturing and right lower facial weakness.

On transfer to our hospital she was febrile (39.4°C) and comatose with a stiff neck, and conjugate deviation of the eyes to the right. Her pupils were slightly anisocoric, with the right larger but both

reactive. She had bilateral Babinski responses. Skull radiography showed mild demineralization of the sella turcica. Echoencephalography revealed no shift and normal-size ventricles. A radionuclide dynamic study was normal; the 3 hr delayed static scan demonstrated an area of increased activity in the right parietal region and a suspicious area in the left parietal zone. The electroencephalogram exhibited bilateral symmetrical theta-delta slowing. Coagulation studies were diagnostic of DIC.

The patient was given Gentamycin for Gram-negative organisms found on sputum culture. Angiography 14 days after admission (transfer) revealed bilateral branch occlusions of the middle cerebral arteries (fig. 5). The patient was treated with low dose intravenous heparin and continued on steroids. During the last week of hospitalization, she continued to have melanotic stools and epistaxes. She died 12 days after angiography. Permission was granted only for an autopsy limited to the abdominal cavity. This revealed an ovarian cystadenocarcinoma metastatic to the contralateral ovary, to the mesocolon, and to the peritoneum. Also noted were multiple renal interlobular artery and pancreatic arteriolar thrombi; multiple renal infarcts; a large splenic infarct with a splenic abscess.

Case 5

A 68-year-old markedly obese man with a long history of chronic obstructive pulmonary disease, cor pulmonale, and erythrocytosis complained of headaches and hallucinations for several days before admission. He drank heavily for 2 days and was admitted because of progressive lethargy. On admission, he had a temperature of 38.6°C, was mildly disoriented and lethargic, and demonstrated asterixis and bilateral Babinski responses. During hospital day 2 he developed sudden right leg pain, and the leg and foot became cold and pulseless. A right femoral embolectomy was performed; coagulation studies revealed a serial decrease in platelets and elevation in split products.

Three days later, he suffered a generalized motor seizure followed by coma. A lumbar puncture revealed the opening pressure to be 310 mm H₂O, with xanthochromic fluid containing 550 red blood cells, 3 white blood cells, 85 mg/dl glucose, 65 mg/dl protein. CT revealed a large hemorrhage in the right parietal lobe (published as fig. 4 in Buonanno et al. [18]). He died 8 days later.

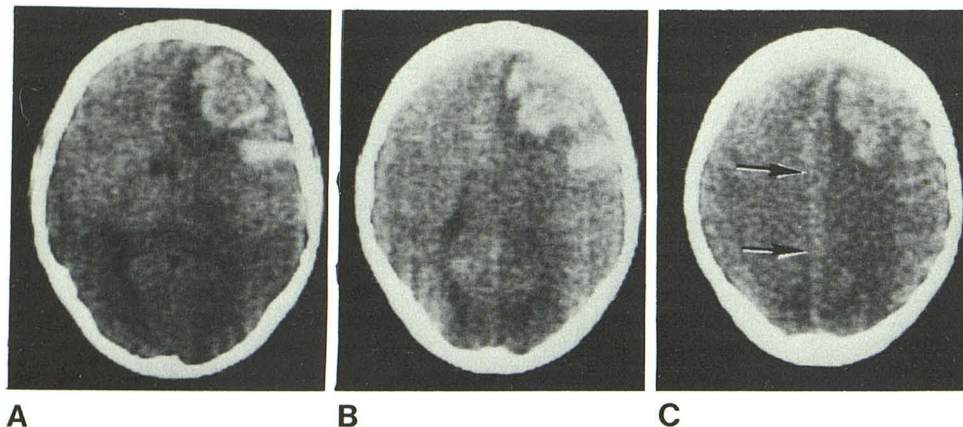


Fig. 4.—Case 3. Superior sagittal sinus and left frontal cortical vein thrombosis. Uninfused CT scans. Frontal hemorrhage. Subarachnoid blood seen in interhemispheric fissure (arrows).

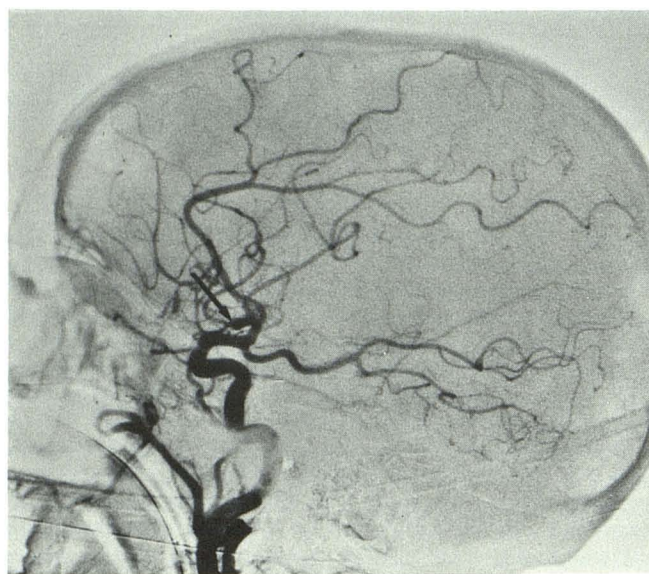


Fig. 5.—Case 4. Major arterial occlusions in DIC. Only one branch of left middle cerebral artery emerges from trifurcation (arrow). On opposite side (not shown) there were also major branch occlusions of middle cerebral artery.

Principal autopsy findings were multiple large arterial vessel thrombi, including the pulmonary artery; hepatic vein and segmental branch thrombi; bilateral superficial middle cerebral vein thrombus with areas of encephalomalacia; and hemorrhage in the right frontoparietal cerebral lobes.

Case 6

A 20-year-old woman, an insulin-dependent diabetic for 11 years, had onset of headaches 3 weeks before admission and was found in diabetic ketoacidosis 2 days before admission. Despite adequate treatment of this condition elsewhere, she remained comatose, with a left hemiparesis, a left Babinski response, and a left gaze paresis.

On arrival in our hospital, coagulation studies were consistent with DIC. Coma deepened and the patient developed bilateral Babinski responses. Echoencephalography demonstrated a marked shift of midline structures from right to left. Radionuclide dynamic study revealed decreased perfusion in the region of the right middle cerebral artery; the 3 hr delayed static study was normal. Right

brachial arteriography revealed a right-to-left shift of the anterior cerebral arteries and occlusion of the right middle cerebral artery (published as fig. 2 in Cooper et al. [19]). She developed repeated bleeding into the urinary tract and blood in nasogastric aspirate. Repeat coagulation studies showed progression of DIC. She was treated with intravenous heparin, followed by normalization of the coagulation studies and control of hemorrhage.

Two days after admission a fixed and dilated pupil developed on the right side. Three right burr holes were placed, but no evidence of intracranial bleeding was found; the brain was soft, swollen, and pale. Two days later the patient had a temperature elevation to 42.2°C rectally, suffered a cardiorespiratory arrest, and died.

Autopsy revealed multiple arterial "hyaline" thrombi within multiple organ sites. A right middle cerebral artery thrombus was associated with cerebral infarction.

Discussion

Disseminated intravascular coagulation (DIC), an acquired thrombotic-hemorrhagic syndrome resulting from the presence of thrombin within the systemic circulation, is an "intermediary mechanism of disease" [2] recognized in an ever-increasing number of disorders and discussed in a rapidly expanding scientific literature (fig. 1). It is manifested clinically as hemorrhage and/or thrombosis at multiple sites: purpura fulminans [20], acrocyanosis [21], hematemesis [22], or a thrombotic episode [23]. The disorder has classically been envisioned as occurring primarily within the microcirculation, with secondary propagation to larger vessels, being thus distinct from thromboembolic disease involving major vessels [14]. Intravascular deposition of fibrin is accompanied by widespread tissue damage which includes the brain [4, 16].

The ultimate consequence of the disorder is determined by the balance between the rate of fibrin formation and fibrin clearance or lysis by the fibrinolytic system. Fibrinolysis is present in virtually every patient with DIC, but plays a homeostatic rather than a pathologic role [1, 3]. Vascular occlusion, if it develops, is presumably the result of "embolic" thrombosis [11]. The localization of these thrombi vary with various agents or factors; for example, corticosteroids favor fibrin deposition in the adrenal gland [1]; transitory or low-grade DIC favors ischemic necrosis of the renal cortex [1]; DIC in diabetic ketoacidosis is often accompanied by cerebral damage [10, 12, 19] contributing to

coma in such patients. Of course, in the latter situation, as in cases of head trauma [7-9], it is possible that brain damage occurs first and, through the release of thromboplastin, leads secondarily to intravascular coagulation. Against this hypothesis is failure to find a cause for focal brain damage in such patients other than the intravascular coagulation itself [4].

The spectrum of neuroradiologic changes is a manifestation of the severity and extent of the intravascular thrombotic process. Often the vessels occluded are not large enough to be identified radiologically. In such an instance, there are no pathognomonic radiologic signs of neurovascular DIC. Cooper et al. [19] described radionuclide dynamic study and cerebral angiographic findings of middle cerebral artery occlusion in a case of DIC in a patient with diabetic ketoacidosis. Subsequently, Weidner and Brennan [24] reported four instances of cerebral arterial occlusion in patients with DIC.

Although large systemic vein thromboses have been reported in pathologic studies of DIC [23], especially when chronic [1], intracranial venous involvement has been underrecognized and its impact not fully appreciated. Pathologic changes in the brain in DIC have been emphasized only recently. Thus Robboy et al. [25] did not find venous pathology in their five cases of DIC with central nervous system involvement; they described fibrin thrombi within the choroid plexi in all of their patients, and, less commonly, anterior spinal or other cerebral arteriolar involvement. Case 1 of the pathologic series by Collins et al. [4] exhibited thrombosis with partial peripheral recanalization of a meningeal vein along with thrombosis of a medium-sized penetrating vein; they found choroid plexus capillary fibrin thrombi in only one of their 12 cases. Although Collins et al. stated that thrombi involved both the arterial and venous circulation, they did not tabulate their data and the incidence of venous pathology in their material cannot be determined. Ten (83%) of their 12 cases had hemorrhagic infarcts, some sharply demarcated and thus possibly resulting from sudden occlusion of penetrating (arterial) vessels [26]. Others had indeterminate borders with gradual transition between necrotic and normal areas, a finding characteristic of venous infarction [27]. Collins et al. [4] did specify, however, that although arteriography was not performed in their patients, the vessels occluded were not large enough to have been identified by arteriography.

Sigsbee et al. [28] recently reported clinical and radiologic findings in some cases of superior sagittal sinus occlusion as a complication of cancer, sometimes associated with DIC. They emphasized the importance of good quality cerebral angiography and the necessity for prolongation of the angiographic sequence to at least 12 sec in order to demonstrate the disorder.

All of our cases had underlying or associated conditions known to be coupled with DIC such as sepsis, congestive failure with pulmonary emboli [6], ovarian carcinoma [29], diabetic ketoacidosis [12], or hemolytic anemia [30]. In four of our six cases, cerebral veins were the principal site of thrombosis, including small, deep or superficial veins, larger lobar draining veins (such as the superficial middle cerebral vein), and large-bore vessels such as the superior sagittal sinus or jugular bulb. In the other two cases, histology

showed that multiple arteriolar or arterial-branch occlusions coexisted with large arterial (e.g., pulmonary, femoral) occlusion. These were usually, but not always, accompanied by evidence of thrombosis elsewhere in the body.

Radionuclide Studies

Recently, radionuclide brain scanning [31, 32] has been shown to be of value in detecting intracranial sinovenous disorders. Posterior radionuclide dynamic and static scanning may reveal images such as the "stump sign," or nonvisualization of the dural sinuses on dynamic study accompanied by increased uptake on static study, images highly suggestive of sinus thrombosis. This technique, while not helpful in visualizing cortical vein thrombosis, may be particularly useful in diagnosing and following the progress of dural sinus thrombosis. Whether caused by arterial or venous thromboembolism, an infarct or hemorrhage may be seen as increased isotopic uptake and is nonspecific.

Computed Tomography

We recently tabulated the CT findings in sinovenous occlusion [18]. While CT is less accurate in evaluating disorders of juxtacalvarial vascular structures, it can detect pathognomonic images of superior sagittal sinus thrombosis (i.e., the dense triangle in an early thrombosis or, later, the "empty triangle" sign). CT, of course, detects associated cerebral changes such as bilateral, parasagittal areas of hemorrhage and/or gyral enhancement, highly suggestive of cortical vein dysfunction secondary to superior sagittal sinus occlusion. Two cases in our series had CT scans, in both revealing cerebral lobar hemorrhage, an image previously cited in reports of sagittal sinus occlusion [18, 31]. Such a location is unusual for a hypertensive cerebral hemorrhage [33] and should be a warning that another etiology may be responsible. A differential diagnosis for nontraumatic spontaneous convexity lobar cerebral (i.e., nonbasal ganglia, noncerebellar, nontemporal lobe) hemorrhage would include arterial thromboembolism, bleeding into primary or secondary tumor, ruptured arteriovenous malformation, ruptured mycotic aneurysm, sinovenous occlusion, coagulation disorder, and occult trauma; hypertension would be a less likely cause.

Angiography

Angiography is the definitive neuroradiologic study to demonstrate major arterial or venous occlusive disease. Acute thromboembolic occlusions may involve multiple, bilateral, large and medium intracranial arteries with predilection for the middle cerebral artery. More commonly, medium-sized vessel occlusions or filling defects may be seen; this may require magnification and/or subtraction angiography. When intermediate vessels are not completely occluded, or small invisible vessels are thrombosed, indirect signs of disturbed circulation may be detected: stasis with slowed circulation or persistence of end-on opacified vessel lumina; perivascular blush possibly representing contrast material extravasation; luxury perfusion [34]; sluggish or absent leptomeningeal collateralization [24].

The angiographic findings in the more subtle venous occlusions have been described in detail by Vines and Davis [35]. Occasionally, the obstructed vessel may not be visualized because of redirected flow, recanalization of the vessel, or dissolution of the embolus with distal progression. In such a case, mass effect caused by edema or hemorrhage may be the only finding.

Conclusions

It must be appreciated that none of the radiographic findings are specific for DIC. Our series is too small and skewed to afford any statements regarding the frequency with which large intracranial vessel thromboses occur within DIC. Large vessel thromboembolic phenomena are more common in chronic DIC and are frequently the cause of initial complaints [1]. Unlike some authors [36], we believe that cerebral dysfunction occurring in DIC is highly correlated with a poor prognosis—75.5% mortality at our institution. Although the diagnosis of DIC, based on laboratory studies described elsewhere [13], is not within the realm of the neuroradiologist, he may play a significant role in alerting the referring physician to this possibility if he is aware of the entity, its clinical setting, and its varied neurologic manifestations.

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