

Fig. 2. Microscopic examination of the surgical specimen showing a monotonous plasma cell population with eccentric atypical nuclei. (Hematoxylin and eosin stain, 600x).

monly includes cognitive changes, headache, cranial nerve deficit, motor deficit and seizure. Treatment includes radiotherapy and chemotherapy, which may be administered either systemically or intrathecally.

The patient with solitary intracranial plasmacytoma should undergo systemic evaluation, including bone marrow examination, skeletal survey and bone scan to exclude multiple myeloma. In one study, multiple myeloma was diagnosed in four of eight patients, who initially presented with intracranial plasmacytoma.³ In this study, the strongest predictor of diagnosis of multiple myeloma from a single known plasmacytoma was cranial base involvement. Plasmacytoma can involve either the dura or the parenchyma; or occasionally both.

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MRI scan is the most sensitive tool in the diagnosis of intracranial multiple myeloma,⁴ however, it may be negative in the initial stages. It commonly manifests as a dura thickening or an intraparenchymal lesion. However, the MRI scan appearance is nonspecific for differentiation from other malignancies.

The prognosis for patients with intracranial multiple myeloma is very poor. The overall median survival from the time of diagnosis is 1.5 months.⁵ However, long-term survival of patients with solitary plasmacytoma following complete excision and post-operative adjuvant therapy has been reported.³ In a case report by Hirata,⁶ leptomeningeal myelomatosis was diagnosed in a patient with well-controlled multiple myeloma.

We conclude that leptomeningeal involvement should be considered in the differential diagnosis of an intracerebral lesion in a patient with multiple myeloma, even if the systemic disease is well-controlled.

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Traumatic internal carotid artery dissection

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Abstract

Traumatic internal carotid artery dissection is a serious condition that may cause ischemic stroke in young patients. It has been underdiagnosed in the past. We present three cases of traumatic internal carotid artery dissection. The clinical manifestations include hemiparesis, hemiparesis, partial Horner's syndrome and cranial nerve palsy. Diagnosis is with carotid color Doppler ultrasound, CT angiography of the neck and conventional angiography. The outcome may be poor with hemiparesis, persistent vegetative state and

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death. We review the literature and discuss the clinical presentation, diagnosis, grading and treatment choices for traumatic internal carotid artery dissection and stroke.

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Keywords: Carotid artery dissection; Ischemic stroke; Craniofacial trauma

1. Introduction

Traumatic internal carotid artery dissection (TICAD) is a common cause of cerebral ischemic stroke in young adults, accounting for approximately 20%.^{1,2} The mean age of patients with TICAD is 45 years.^{1,2} The average annual incidence of TICAD is 3/100,000.^{1,3,4} Bilateral internal carotid artery (ICA) dissection is seen in 3–28% of affected patients.^{1,5} Overall mortality ranges from 0–40%, with long-term morbidity occurring in 40–80%.^{6–8} We present three cases of TICAD, review the literature, and discuss diagnosis, classification, treatment, and prognosis for this type of stroke.

2. Case reports

2.1. Patient 1

A 22-year-old man was admitted to our hospital with a change in consciousness noted by his family after his daily work. He had fallen down some stairs at work 2 days before admission. He had complained of a mild headache, which he considered minor compared with the degree of muscle strain associated with the accident.

On admission, vital signs were stable with a blood pressure of 128/98 mmHg. Physical examination revealed an abrasion on the right neck and a bruit. The patient obeyed

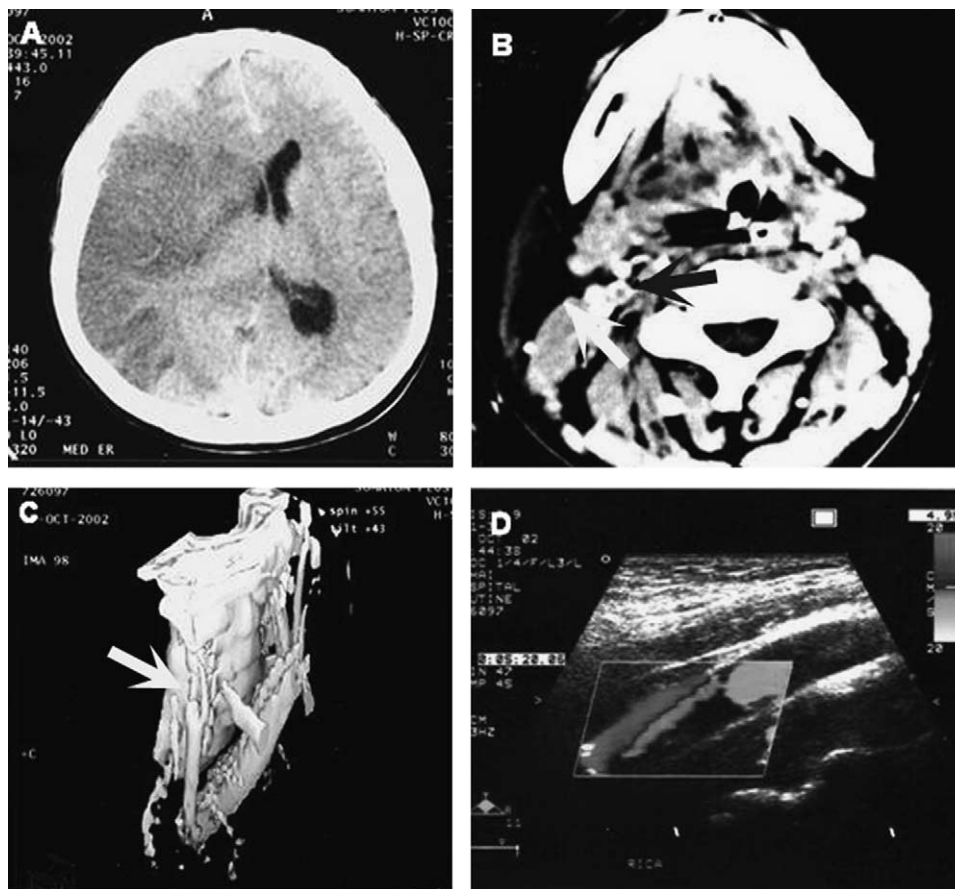


Fig. 1. Patient 1 **A**: Cranial CT showing hypodensity in the right middle cerebral artery territory, with severe mass effect. **B**: CT scan of the neck showing a filling defect in the right internal carotid artery with a crescent-shaped enhancement (black arrow) and surrounding soft tissue swelling (white arrow). **C**: 3D CT angiography of the neck showing near total occlusion of right internal carotid artery (arrow). **D**: Doppler ultrasound of the internal carotid artery with two significant findings (available in colour at www.sciencedirect.com). The red area reveals normal blood flow within a sting-like residual lumen. The blue area demonstrates a false lumen resulting from dissection. The black region is thrombus.

commands, but was confused. A left hemiparesis was found on neurological examination. Brain CT scan revealed a low-density area in the right middle cerebral artery (MCA) territory, with severe mass effect (Fig. 1A). An emergent decompressive craniectomy was performed due to deterioration in his neurological status.

Neck CT angiography and carotid color Doppler ultrasound were performed postoperatively. The neck CT revealed a filling defect in the cervical segment of the right ICA (Fig. 1B). CT angiography revealed near total occlusion of the right cervical ICA (Fig. 1C). The ultrasound examination demonstrated a thrombus in the right ICA and a string-like residual lumen with a trickle of flow (Fig. 1D). The patient died 3 days after admission due to progressive neurological deterioration.

2.2. Patient 2

A 47-year-old man had been involved in a traffic accident. His initial neurological examination revealed a change in consciousness, with a Glasgow coma score of 7 (E1, V1, M5). A left frontal scalp laceration was noted with underlying bone exposure. Right radial, patellar and tibial fractures

were noted. Weakness of the right extremities was observed, and was initially thought to be due to the multiple fractures. A brain CT scan (Fig. 2A) revealed pneumocranium of the skull base, subarachnoid hemorrhage and fractures of the left zygoma and temporal bone. X-rays of the cervical spine (Fig. 2B) showed a teardrop fracture of the C2 vertebral body. A craniotomy for compound skull fracture and dural repair were performed soon after admission. Two days postoperative, a brain CT scan (Fig. 2C) revealed a large infarct in the left ICA territory. An emergent decompressive craniectomy was performed and ICA dissection and occlusion was found on intra-operative angiography (Fig. 2D). His neurological condition stabilized after the decompressive craniectomy but there was persistent aphasia and complete right hemiplegia.

2.3. Patient 3

A 48-year-old man suffered a head injury in a traffic accident 7 days prior to admission. He had lost consciousness for about 4 hours. Loss of visual acuity of the left eye and limited range of extraocular movements were noted after he regained consciousness. In the days after the injury, right

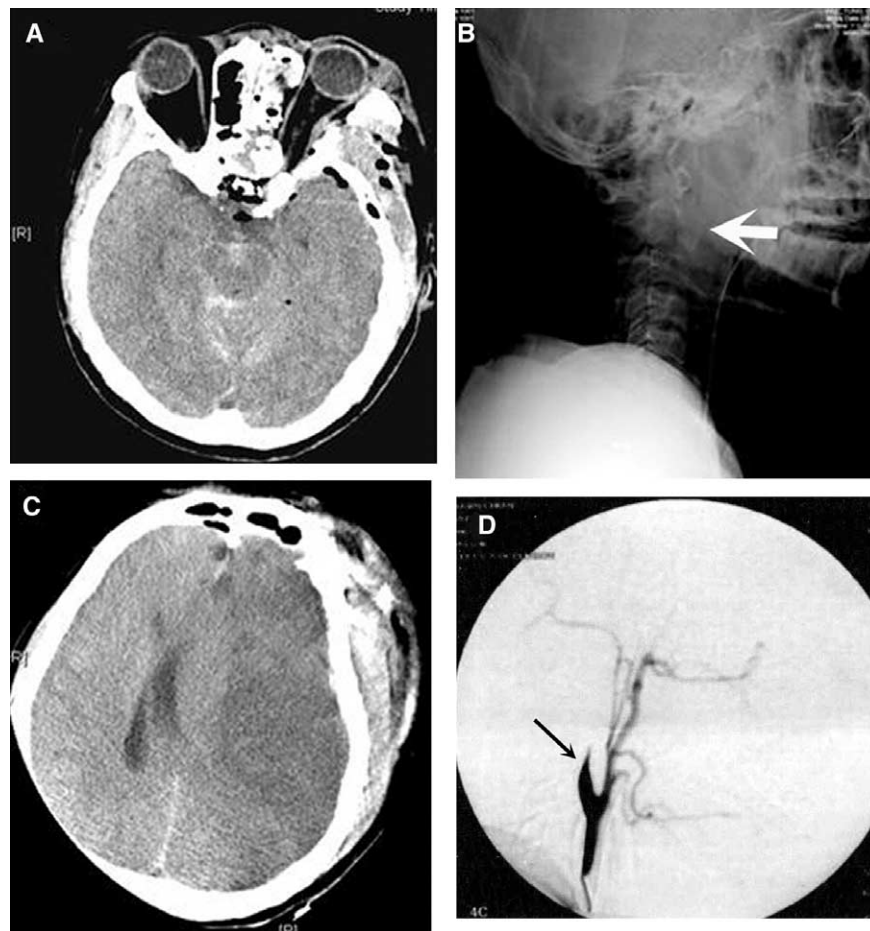


Fig. 2. Patient 2 **A**: The brain CT scan showing subarachnoid hemorrhage, multiple skull fractures and pneumocranium. **B**: Lateral cervical X-rays showing a C2 tear drop fracture (white arrow). **C**: Two days after the initial brain CT, the CT scan was repeated, showing a left internal carotid territory infarct. **D**: Left cervical internal carotid artery angiography showing a tapering of the vessel to occlusion (arrow).

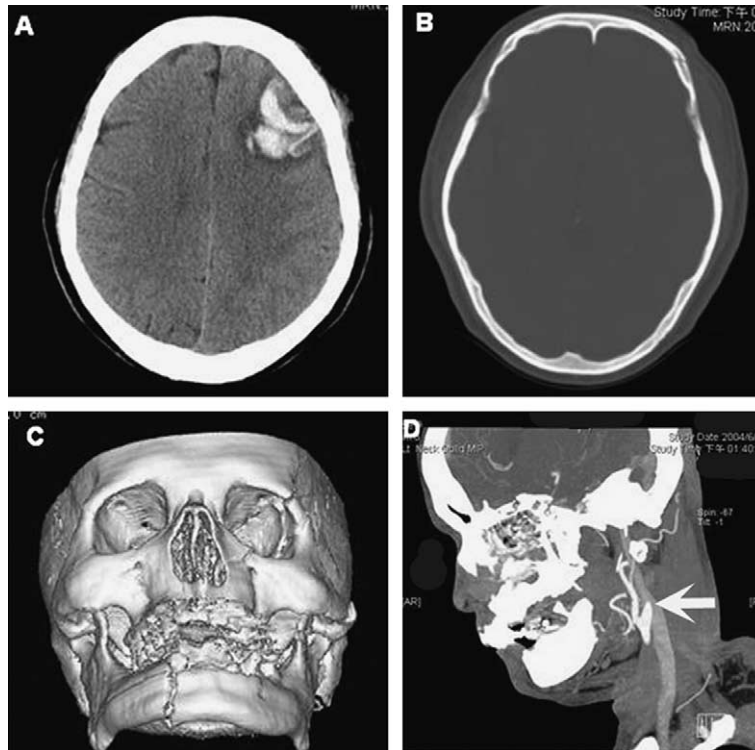


Fig. 3. **A:** CT scan showing a watershed hemorrhagic infarction between the left anterior cerebral and middle cerebral artery territories. **B:** CT scan with bone windows showing left skull vault fractures. **C:** 3D reconstruction of the CT scan showing multiple craniofacial fractures on the left. **D:** Cervical CT angiography showing a left cervical internal carotid artery dissection and occlusion (arrow).

hemiparesis developed. Initial brain and facial bone CT scan (Fig. 3A) revealed a left anterior cerebral artery and middle cerebral artery watershed hemorrhagic infarct. There were multiple craniofacial fractures including of the left lower frontal bone, left orbitozygomatic bone, left orbital floor and hard palate and a mandibular parasymphiseal fracture (Fig. 3B, C). There was no cervical spine fracture. The hemorrhage was managed conservatively and the craniofacial fractures were surgically repaired after the clinical neurological status was stable. Postoperatively, hypohidrosis of the left hemifacial region was found. CT angiography revealed a left ICA dissection and occlusion (Fig. 3D). This was managed conservatively and followed regularly, maintaining a stable neurological condition.

3. Discussion

Risk factors for TICAD include vigorous physical exercise, blunt head and neck trauma, penetrating neck injury and skull-base fracture. Shearing forces on the stretched artery induced by a rotation-hyperextension or distraction-flexion injury have been described as the main pathophysiological mechanism.^{3,9–11}

Only 10% of patients display immediate symptoms; 55% develop symptoms in the first 24 hours after dissection and 35% have no symptoms until 24 hours or more after injury. Patients with immediate symptoms frequently have a neurological deficit and a normal CT scan.^{12–14} Common symptoms in TICAD include hemicrania, Horner's syn-

drome, hemiplegia, unilateral facial weakness, hemianesthesia, aphasia, amaurosis fugax and seizure.^{12,13} Carotid trauma should be suspected in any alert patient who has a posttraumatic hemiparesis, as other insults causing hemiparesis are most often associated with obtundation.¹⁵ Subadventitial carotid bleeding or expansion of a dissecting aneurysm may also mechanically damage one or more cranial nerves. Generally, TICAD is diagnosed after the development of an unexpected neurological deficit and about 80% of patients progress to cerebral infarction within one week of onset of initial symptoms.^{4,16}

In addition to detailed history and physical examination, several modalities, including duplex ultrasonography, CT/CT angiography, MRI, magnetic resonance angiography (MRA), and conventional angiography, have improved the diagnosis of TICAD. Duplex ultrasonography is a useful screening tool that may show tapering of the carotid artery lumen, a false lumen or thrombus. Some reports have shown that combining ultrasonography with transcranial Doppler increases sensitivity to 95% in TICAD.^{1,10} CT/CT angiography and MRI/MRA provide sensitivity from 80% to 95% in detecting TICAD. The characteristic lesion is a crescent-shaped lesion due to intramural hemorrhage surrounding a narrowed lumen. MRI/MRA has a higher diagnostic rate than CT as it also visualizes vessel-wall irregularity and aneurysmal dilatation. Conventional angiography remains the gold standard for diagnosis of TICAD. It assesses severity of stenosis, the level and extension of the dissection and the presence of aneurysmal dilatation.^{3,6,9,11}

Treatment of TICAD may be surgical or non-surgical. Non-surgical treatment includes observation, anticoagulation or anti-platelet medical therapy and endovascular stenting. Surgical treatment may include endarterectomy/thrombectomy with or without patch angioplasty, extracranial-intracranial bypass, arterial ligation and vascular resection with interposition venous graft.^{4,7,8,14,16–18} The choice of treatment depends on individual conditions including severity of injury, neurological deficit, collateral flow pattern, the anatomic segment involved and the time between injury and treatment.

A new grading scale based on angiographic findings was introduced in 1999.⁸ TICAD is classified as: Grade I, luminal irregularity or dissection with <25% luminal narrowing; Grade II, dissection or intramural hematoma with ≥25% narrowing, intraluminal thrombus, or raised flap; Grade III, a pseudoaneurysm; Grade IV, occlusion; and Grade V, transection with free extravasation. This classification may also guide treatment.⁸ Grade I TICAD should be treated conservatively with anticoagulant therapy as only 7% of cases progress to a higher grade with conservative management. In contrast, 70% of Grade II lesions may progress to pseudoaneurysm or occlusion, and these lesions require aggressive surgical intervention. Surgical treatment results in a good postoperative vessel patency rate ranging from 80–100%.^{7,16,19} Recent improvements in endovascular techniques allow stent placement for Grade II and III lesions with acceptable results.⁴ Grade IV TICAD carries a high stroke rate. There have been reports of recanalization procedures, but these carry high risk in the early post-injury period. Observation and/or anticoagulation are more often recommended. Grade V TICAD is devastating. Surgical ligation and bleeding control should be performed immediately. These patients have a high mortality rate and often die before diagnosis.

Much evidence indicates that conservative, non-surgical management with anticoagulant medication yields results equivalent to surgical intervention. However, in patients with a neurological deficit, there is a risk that anticoagulation may convert a non-hemorrhagic infarct into a hemorrhagic infarct, as is seen with thrombolytic therapy for acute ischemic stroke. Therefore, arterial ligation rather than anticoagulation or revascularization is recommended for patients who have a severe neurological deficit or an early evident hypodense area noted on the brain CT, resulting from TICAD. In cases where the involved segment of the ICA extends into the intracranial portion of the artery direct surgery or stenting are hazardous, and anticoagulation or proximal arterial ligation and/or extracranial-intracranial bypass is the treatment of choice. In this situation, adequate collateral flow is critical in the choice of management. If collateral flow is adequate, observation and/or anticoagulation medication is suggested; if collateral flow is inadequate, proximal arterial ligation and extracranial-intracranial revascularization should be considered.

Due to the lack of diagnostic symptoms and signs of TICAD, and the associated facial and other injuries, the

diagnosis of TICAD is often delayed. Delayed treatment contributes limited neurological benefit and may result in catastrophic complications. Revascularization of the infarcted brain carries a high risk of hemorrhagic transformation. Therefore, the time between injury and treatment, and the viability of the ischemic brain, are critical considerations when making management decisions in TICAD.

4. Conclusion

TICAD has been under-diagnosed in the past. Careful history-taking and physical examination may increase the diagnosis rate, and prevent delayed ischemic stroke after blunt head and neck trauma. Recently improved diagnostic tools including duplex ultrasonography, CT/CT angiography, MRI/MRA, and conventional angiography may increase the sensitivity and specificity of diagnosis. Management varies with the individual presentation, however, emergent and adequate therapeutic options should be utilized to restore maximum neurological function and prevent complications.

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Unilateral traumatic posterior fossa subdural effusion in an infant

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Abstract

Supratentorial subdural effusion is common after infection and trauma, but rarely occurs in the posterior fossa, and is even less commonly unilateral. The authors report a rare case of unilateral posterior fossa subdural effusion with secondary hydrocephalus. A 6-month-old female infant presented with lethargy, poor appetite, and persistent vomiting after head trauma 2 weeks previously. A non-enhanced brain CT scan revealed a right posterior fossa subdural fluid collection that displaced the fourth ventricle and brainstem and dilated lateral ventricles. While monitoring the intracranial pressure, the baby was treated with temporary external subdural drainage, followed by a permanent subdural peritoneal shunt. The treatment and likely physiopathology of this unique case are discussed. © 2005 Elsevier Ltd. All rights reserved.

Keywords: Posterior fossa; Subdural effusion; Trauma

1. Introduction

Supratentorial subdural effusion (SDE) is well-recognized, but a unilateral posterior fossa SDE is relatively uncommon, particularly in children. We report an infant with a right unilateral retro-cerebellar subdural effusion, following head trauma. The treatment of this unique presentation is described and the likely pathophysiology discussed.

2. Case report

A 6-month-old female infant presented to our hospital. She had been born to a healthy mother with an uncomplicated pregnancy and delivery. She had undergone unremarkable systemic and neurological development and had been healthy prior to falling from an 80 cm height. Irritable crying, a soft tissue swelling over the right occiput, poor appetite, frequent projectile vomiting and lethargy developed over a

2-week period following the fall. A bulging, tense anterior fontanel and tachycardia were noted on admission. An emergency non-enhanced CT brain scan revealed supratentorial hydrocephalus and a collection of subdural fluid in the right retro-cerebellar space (Fig. 1). The right cerebellar hemisphere, fourth ventricle, and brainstem were compressed and displaced by the effusion. A right frontal external ventricular drain (EVD) was inserted for intracranial pressure (ICP) monitoring, followed by external subdural drainage to evacuate the right retro-cerebellar SDE. The initial ICP was high, but CSF was not drained. The effusion, with a very high initial opening pressure, was clear and colorless. There was no outer membrane. After drainage of the SDE, the cerebellum became slack and the ICP decreased. Fluid from both the ventricle and the subdural space were sent for microscopic, biochemical and microbiological examination; the results are shown in Table 1.

The child was calm and easily aroused postoperatively. The SDE was drained continuously at 5 mL/h. The ICP remained below 5 mmHg, with steady clinical recovery. A CT scan of the brain on the second postoperative day

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