Chief Complaint

- Recurrent severe cramping pain over bilateral calf muscles following walking for two weeks

Present Illness

- This 49-year-old man has a past history of aortoiliac occlusive disease and had received aortobifemoral bypass graft (intra-abdominal approach) & right 5th toe amputation at NTUH in 2002

- In May, 2004, he was admitted in WFH because of intermittent claudication with chronic ulcerative wounds over right foot.
  - Angiography: (2004/5/5)
    - Atherosclerotic changes with severe stenosis of the lower abdominal aorta at the level of L3 vertebral body
    - PAOD of right lower extremity (right popliteal a., right posterior tibial a.)
  - Operation (2004/5/10)

    Arterial endarterectomy with re-do artobifemoral bypass graft and left lumbar sympathectmy (retroperitoneal approach)
  - Pathology

    Atheroma with fibrous cap and central necrosis

Second Admission at WFH, Sep, 2005

- During regular OPD follow-up, the patient complained of cramping pain of calf muscles after walking for a while, especially the left legs. The pain would relief after resting, but the symptom progressed day by day.
- Physical examination
  - Bilateral pulselessness of femoral arteries, cold skin, ulcerative wound, cyanosis over the dorsal side of left foot
- ABI
  - Right: 0.13 Left: 0.19
  - PAOD of both lower extremities

Second Admission at WFH, Sep, 2005

- CT at TMUH (205/9/8)
  - Remarkable peripheral thrombotic partial occlusion of the proximal abdominal aorta at about bil renal arterial level.
  - Complete occlusion of Lt branches of SMA, IMA, and involvement along bifircation of common iliac, bil internal and external iliac arteries.
  - Very thin and weak caliber arterial circulations of bil lower extremities still noted, probably arise from collateral circulations.

Second Admission at WFH, Sep, 2005

- Because of the poor condition of the vessels, only medical therapy were given.
  - PGE1, heparin injection
- Discharged with OPD follow-up and medication
  - Aspirin 100mg 1# PO QD
  - Warfarin 5mg 1# PO QD
  - Cilostazol (Pletaal) 50mg 1# PO BID

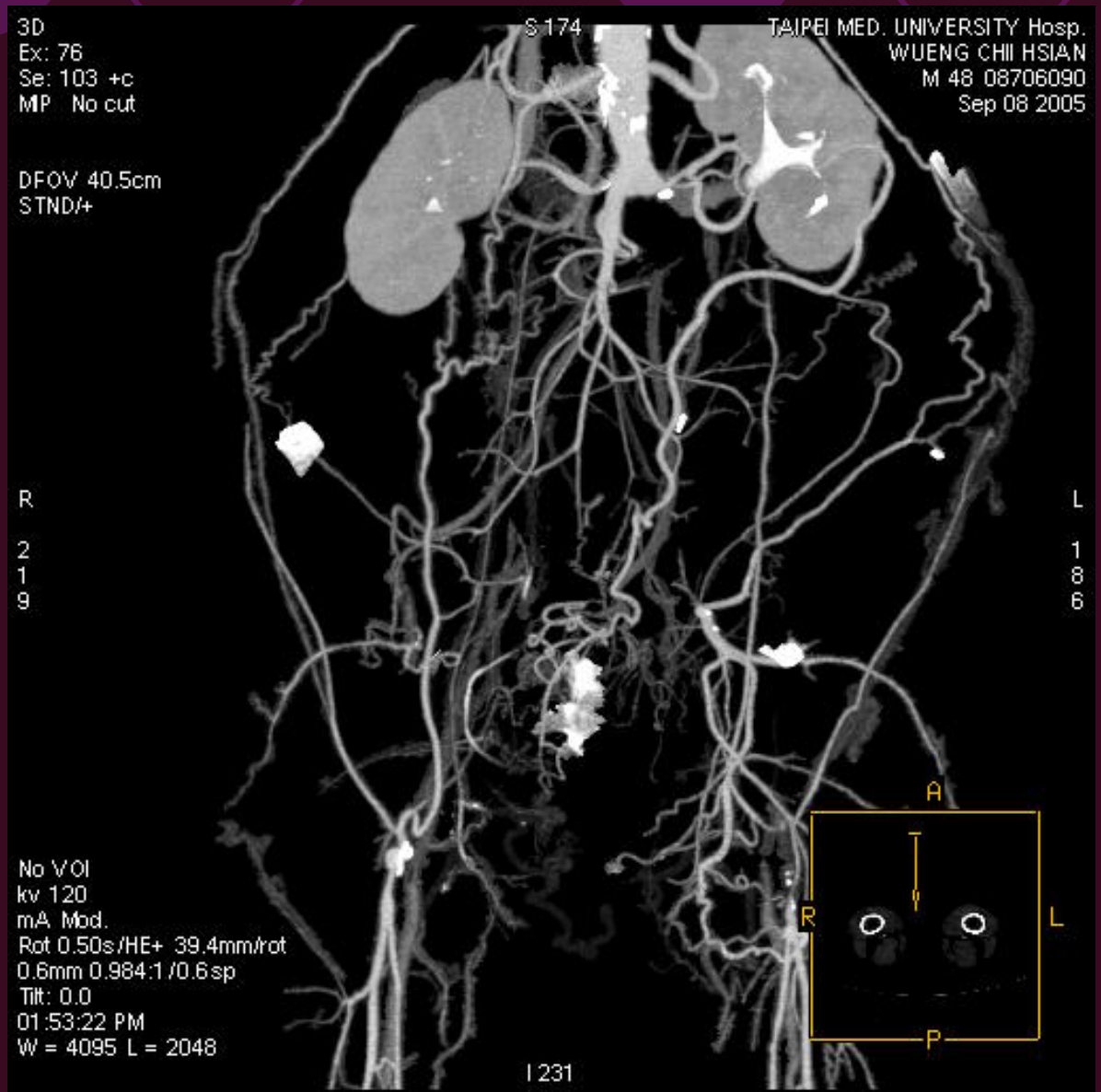
Personal & Family History

- Tobacco: + 20+years, 1PPD/day
- Alcohol: + 烈酒
- Betel nut: -
- Allergy: -
- HTN:+ without treatment (BP: 140/90)
- DM: -
- Heart disease: -
- Asthma: -
- Old TB 7-8 years ago
- Family history: DM- HTN -

Lab data (2005/9/7)

- Cholesterol 195
- HDL-C 30
- LDL-C 116
- Triglyceride 293
- Uric acid 8.6
- Fibrinogen 489 mg/dL
- Protein C 100.4 %
- Protein S 107.0 %

Image Study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Rot 0.50s/HE+ 39.4mm/rot
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W = 4095 L = 2048
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Aortoiliac Occlusive Disease

- Chronic
  - advanced peripheral atherosclerotic disease
  - Inflammatory arteritides
    - Takayasu's arteritis: initially steroids followed by cytotoxic agents
- Acute
  - arterial embolism
    - 85% of such emboli are of cardiac origin: heparin injection
  - a consequence of thrombosis at the site of advanced atherosclerotic plaque
- Acute aortic occlusion requires prompt recognition and emergent surgical intervention. Mortality is greater than 75%, if not treated.

Pathophysiology

- Risk Factors: cigarette smoking, hypertension, diabetes mellitus, and hypercholesterolemia
- Men being afflicted more commonly than women
- Afflicts the abdominal aorta more commonly than the thoracic segment

Clinical Presentation

- Pain in the calves, thighs, and buttocks brought on by exercise that usually subsides shortly after the cessation of exertion.
- Some patient may present only weakness
- Progress to pain at rest
- Extremity pain that awakens the patient from sleep and is relieved by dangling a foot over the side of the bed or relieved by sleeping in a chair or by standing.
- Bilateral aortoiliac occlusion in men->sexual impotence (Leriche's syndrome)

Clinical Presentation

- Permutation of pain, pallor, paresthesias, paralysis, poikilothermia, or pulselessness in the lower extremities can combine with ischemic pain in the buttocks and lumbosacral region
- Irreversible ischemia of the distal tissue begins within approximately 6 hours of occlusion

Diagnosis

- The strength or **absence of pulses** in the femoral, popliteal, posterior tibial, and dorsalis pedis arteries in either leg should be noted and compared with pulses in the upper extremities, although the presence of pulses does not rule out disease.

DW1

- A faint, monophasic Doppler signal indicates poor perfusion, whereas a strong triphasic signal is characteristic of a normal pulsatile flow.

DW1 the dorsalis pedis pulse is congenitally absent in up to 10% of the population, more commonly among people of Caucasian descent.

The posterior tibial pulse, on the other
hand, is missing in less than 1% of the population
Dina Wu, 2005/11/9

Diagnosis

- Signs of prolonged ischemia of the lower extremities including muscular atrophy, absence of hair over the dorsal surface of the toes, foot, and across the pretibial area, and thickening of the toenails as a consequence of slow longitudinal growth.
- Pain and pallor are early signs of acute ischemia, and a preserved sensation of light touch is indicative of tissue viability. Complete anesthesia and paralysis are ominous signs of potentially irreversible neuromuscular ischemia
- Ankle-brachial index (ABI): a value of less than 0.9 has a sensitivity and specificity of approximately 95% for the detection of an angiographically demonstrable arterial occlusion.
- An ABI of less than 0.4 indicates extreme occlusive disease and a threat to lower extremity viability

DW2

DW2

Calcification of the lower extremity
arteries, as seen in patients who have diabetes or renal failure, can cause false elevation of the ankle systolic blood pressure and, therefore, a falsenegative ABI

Dina Wu, 2005/11/9

Image Studies

- Clinical acumen is usually sufficient for diagnosis
- Digital subtraction angiography (DSA): invasive and requires the administration of contrast media (gadolinium or carbon dioxide)
- Helical CT scanning
- MRA: a modality of choice in the less acute setting, with a sensitivity and specificity approximating that of DSA
- Transabdominal duplex ultrasonography: most useful in ascertaining whether or not AAA is present.
- Transesophageal echocardiography: thrombus in the thoracic aorta or potential cardiac sources of embolization

Management

- Determine whether their presentation represents an incremental worsening of their atherosclerosis or whether an acute thromboembolic event has been superimposed upon their underlying disease
- Differentiation must then be made between functional ischemia and progression of occlusion to the point where there is an acute threat to limb viability.

- Aortobifemoral bypass grafting has become the surgical procedure of choice for aortoiliac disease
- Percutaneous transluminal angioplasty (PTA) is an alternative that might be appropriate in certain clinical settings, although it is more commonly reserved for disease involving the peripheral vessels.

- If an embolic source of the occlusion is strongly suspected on clinical grounds, direct or Fogarty catheter embolectomy takes precedence over any imaging study that might prolong ischemia.
- If the differentiation between embolic and in situ aortoiliac thrombosis is uncertain, emergent passage of a Fogarty catheter might restore patency temporarily, but recurrence of thrombosis is common.
- Intra-arterial infusion of a thrombolytic agent has emerged as a potential therapy in this setting

Surgical Intervention

- Operative mortality: less than 5% in most major vascular centers.
- Complications
  - Aortoenteric fistula:
    - after the proximal graft anastomosis erodes into the duodenum
    - An abdominal aortic prosthetic graft and gastrointestinal bleeding
  - graft infection
    - commonly they manifest months to years after surgery
    - Periprosthetic fluid collections, failure of graft incorporation, and an increase in the erythrocyte sedimentation rate
    - CT and tagged leukocyte scanning are effective means of diagnosis

Prognosis

- Individuals with systemic small-vessel disease such as vasospastic phenomena (including Raynaud’s syndrome), severe diabetic angiopathy, or angiopathy associated with autoimmune disorders or Leriche’s syndrome may develop compensated DIC, which often becomes fulminant.