Peripheral Venous Hypertension Related to Arteriovenous Fistula for Hemodialysis

- A Case Report -

Wen-Yu Chang  Jiann-Woei Hwang*  Hong-Tien Kuo**  Gwo-Shing Chen  Hsiu-Hui Chiu  Chieh-Shan Wu

Venous hypertension is a significant problem for patients under chronic hemodialysis. Arterialization of the outflow venous system may induce this condition, and result in impairment of arteriovenous access function, disabling upper extremity edema, violaceous discoloration and hyperpigmentation, ulceration of the skin, and in advanced cases, gangrene formation of the fingers and neuralgia. Histologic features show thick-walled capillaries with plump endothelial cells in upper dermis, which are compatible with the pattern of stasis dermatitis by chronic venous insufficiency. Early awareness of these problems is crucial for dermatologists to prevent the extreme result of gangrene formation.(Dermatol Sinica 24: 145-149, 2006)

Key words: Venous hypertension, Pseudo-Kaposi’s sarcoma, Arteriovenous fistula

From the Departments of Dermatology, Cardiovascular Surgery,* Nephrology,** Kaohsiung Medical University Hospital
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Reprint requests: Chieh-Shan Wu, M.D., Department of Dermatology, Kaohsiung Medical University. No. 100, Shih-Chuan 1st Rd., Kaohsiung, Taiwan, R.O.C.
TEL: 07-3208214   FAX: 07-3216580
INTRODUCTION

Venous hypertension of the hand caused by arteriovenous access in patients receiving hemodialysis was first described in 1975.1 It causes swelling, induration, hyperpigmentation, and even ulceration distal to the arteriovenous access. We herein report a case of venous hypertension which developed thirteen months after arteriovenous access creation, as a rare complication in patients involved in chronic hemodialysis program.

CASE REPORT

A 69-year-old female visited our outpatient-clinic in November 2004 for progressive swelling and tingling sensation over her right hand for 4 months. She had an autologous arteriovenous access placement over her left wrist and has been receiving hemodialysis at a local hospital for 9 years for underlying end stage renal disease with undetermined etiology. Past medical problems included hypertension, previous gastric ulcer bleeding, and right ureteral and renal transitional cell carcinoma. Due to gradual thinning of the arteriovenous shunt, she had a right brachial artery expanded polytetrafluoroethylene (PTFE) arteriovenous shunt built over right upper arm at the local hospital and kept receiving hemodialysis since June 2003. In July 2003, she underwent radical cystectomy, bilateral nephroureterectomy, and paraaortic lymph node dissection and received regular hemodialysis via the new PTFE shunt. Swelling, local heat and erythema over her right hand and forearm developed after hemodialysis. The patient then abandoned the use of her right upper arm shunt and returned to receive hemodialysis via the previous left wrist graft. Erythema gradually subsided with less swelling. However, she started to notice progressive swelling, violaceous change, and tingling sensation of right hand since July 2004.

On physical examination, severe hand swelling, purple patches, and violaceous change of right hand were observed (Fig. 1). The skin temperature of right hand was warmer than left hand. In addition, several brownish papules over forearm were also noted. Cellulitis or herpes zoster was initially considered. Laboratory examination revealed slightly elevated C-reactive protein, 29.2 µg/ml (normal range <5 µg/ml), and leukocyte count 9.38 x 10^3/ul (normal range 4-10 x 10^3/ul). During the admission in November 2004, we prescribed intravenous acyclovir 125mg along with prostaphyllin 2 g

Fig. 1
Swelling, induration, violaceous discoloration and ulceration of right hand caused by arteriovenous access for hemodialysis

Fig. 2
Thick-walled capillaries with extravasated erythrocytes, hemosiderin deposits, and interstitial mononuclear inflammatory infiltrate in the dermis (H&E stain, 200X).
per day. We consulted cardiologists for vascular survey and peripheral vascular echography of upper extremities, which showed adequate right arm flow and no evidence of deep vein thrombosis. The patient requested discharge after 3 days due to personal preference for hemodialysis at previous hospital.

In December 2004, she visited our outpatient-clinic again with complaints of persisted swelling and severe neuralgia of her right palm. Incisional biopsy was taken from the violaceous discoloration on the dorsum of her right hand. Under the microscope, the epidermis and papillary dermis was unremarkable. Thick-walled capillaries with plump endothelial cells, extravasated erythrocytes, hemosiderin deposits and mononuclear inflammatory infiltrates in dermis were noticed (Fig. 2). The histological pattern is compatible with stasis dermatitis in patients with chronic venous insufficiency. During readmission, leukocytosis (12 x 10^3/ul) with elevated C-reactive protein (53.1 μg/ml) was found. We prescribed Unasyn® (ampicillin sodium/sulbactam sodium) 3 g per day with supplement after hemodialysis, but the patient's condition did not improve. Skin swabs for aerobic and anaerobic cultures from the ulcerations were negative. We arranged Tc-99m RBCs subcutaneous radionuclide venography and Tc-99m sulfur colloid lymphoscintigraphy for suspicion of poor venous or lymphatic return. The result of venography revealed visualized intact deep venous return without collateral venous formation in the bilateral upper extremity over blood flow (with tourniquet applied in the bilateral elbows and wrists) and blood pool (after removing tourniquet in the bilateral elbows and wrists) study. Lymphoscintigraphy showed no overt evidence of lymphatic obstruction. Patient responded poorly to antibiotic treatment and her condition continued to deteriorate with worsening of swelling, ulceration, neuralgia, and fever (38.5°C). Further elevation of C-reactive protein level (91.8 μg/ml) was also noted. Diagnostic angiography was then arranged due to highly susceptible vascular problems despite the risk of local infection. After puncturing the arteriovenous access, the angiography showed reverse flow, arterialization of the outflow vein, and poor central venous drainage (Fig. 3a, 4). Diagnosis of venous hypertension caused by hemodialysis shunt was then established. Diffuse venous collaterals and poor distal arterial run-off with irregular contour were also noticed over right forearm and hand, possibly related to long-standing edema (Fig. 3b). The
cardiac surgeon then performed horizontal suture ligation of the graft. Two days after operation, progression of swelling halted with formation of wrinkle on her right hand dorsum (Fig. 5). Follow-up C-reactive protein level also decreased to 13.7 μg/ml. She was then discharged with topical silver sulfadiazine for wound care. After 6 months’ follow-up, there has been no recurrence of the swelling or ulceration. Only residual hyperpigmentation was observed over her right hand.

**DISCUSSION**

Proper creation of a well-functioning arteriovenous access for hemodialysis is vital in patients involved in chronic hemodialysis program. Vascular complications of arteriovenous access construction for hemodialysis are rare. However, early recognition is crucial. Venous hypertension occurs in 0.1% to 0.5% of patients and may develop after 1 to 2 years. In our patient, venous hypertension occurred thirteen months after the creation of arteriovenous shunt.

The clinical findings of the complication start with edema of one or more fingers, and the thumb is often affected. The edema may extend to the whole hand and forearm. More developed stages are manifested by bluish discoloration of the fingers, cyanotic-like changes, and pigmentation. In advanced cases, necrosis of skin, gangrene change of the fingers and neuralgia may occur. Eventually ulceration of the skin occurs similar to that seen in chronic venous hypertension related varicose ulceration of legs.

The etiology of central venous stenosis related to hemodialysis is most often initiated by placement of an indwelling catheter. Injury to the venous wall leads to fibrotic reaction and thrombosis that may cause significant stenosis or complete occlusion of the subclavian vein. If the access does not thrombose and the collateral circulation around the area of central venous occlusion is not adequate, signs and symptoms of venous hypertension become apparent.

Anatomically, venous hypertension can occur in all configurations of upper extremity access due to arterialization of the veins. Increased venous pressures in the presence of incompetent venous valves distally in the arm lead to higher distal venous pressure and development of symptoms. This can also happen in prosthetic arteriovenous access when there is reversal of flow in the outflow vein due to valvular incompetence in the vein distal to the venous anastomosis. This may be the possible etiology in our patient.

The erythema, tenderness, swelling, skin necrosis and neuralgia may lead to misdiagnoses related to certain infectious processes such as cellulitis and herpes zoster. Antimicrobial treatment alone may be ineffective, as in our case. It is necessary to distinguish limb ischemia secondary to arterial insufficiency, which induces a similar picture but without the edema. To identify these patients with venous hypertension, a high index of suspicion is required. Physical examinations such as palpation and auscultation may indicate the direction of blood flow. Venous duplex ultrasound is a good screening tool for imaging of the central venous circulation for stenoses, dilated collaterals, and venous reflux. Unfortunately, some proximal lesions are unable to be adequately imaged secondary to poor ultrasound windows.

The definitive diagnostic study remains in upper extremity angiography. Direct puncture of
the arteriovenous access itself should include imaging of the access, with particular attention to the venous anastomosis, and the central venous circulation for detection of central venous occlusion or arterialization of vein.

Skin biopsy of the hyperpigmented macules may be helpful, by revealing the findings of stasis dermatitis-like lesions with thick-walled capillaries with plump endothelial cells, or pseudo-Kaposi’s sarcoma with proliferations of small vascular spaces and narrow vascular channels lined by spindle cells in extreme cases. Extravasated erythrocytes, hemosiderin deposits and mononuclear inflammatory infiltrates are common findings.

The primary goal of diagnosis and therapy of venous hypertension is symptomatic relief while maintaining the functionality of the access. Mild forms of the complications, mostly demonstrated by edema, may be treated by positioning of the limb and employment of bandages. Ligation of the access and replacement at another site has been proposed as a primary mode of the therapy, which decreases the flow into the venous circulation and reduces arm edema and sequelae of venous hypertension. Other choices of managing severe venous hypertension may include percutaneous catheter-based transluminal angioplasty and intravascular stents placement, reconstruction of the central venous circulation with bypassing the occlusive lesion to a patent vein with direct central flow, and adjuncts of anticoagulation and antibiotics.

As the numbers of patients on hemodialysis increases, more arteriovenous accesses are constructed. Venous hypertension will become a complication which dermatologists may encounter with rare but increasing frequency. This report highlights and documents angiographically a case of venous hypertension masquerading as an infection. We would also like to emphasize the importance of diagnostic angiography due to poor window of peripheral vascular echography over proximal shunts and insufficient sensitivity of Tc-99m RBCs subcutaneous radionuclide venography.

REFERENCES