

Symmetrical peripheral gangrene associated with cardiac surgery

The Editor,

A 63-year-old female, with known hypertensive and diabetic, was diagnosed as a case of acute anterior wall myocardial infarction for which thrombolytic treatment with streptokinase was given to her in a peripheral center. As the condition of the patient deteriorated, she was shifted to our hospital for further management. Echocardiography revealed ejection fraction of 45–50% and 12 mm long muscular ventricular septal defect (VSD) at the junction of mid and apical interventricular septum, with the left to right shunt. Doppler study of upper and lower limb arteries was normal. Laboratory investigations were within normal limits except high total leukocyte count 22,700/mm³.

In the presence of VSD and keeping in mind to decrease afterload of heart, we inserted intra-aortic balloon pump (IABP) through the right femoral artery and heparin infusion 1000 unit/h was started. After taking samples of blood and urine for culture and sensitivity test, we started antibiotic injections of cefoperazone, sulbactam, and metronidazole. Coronary artery bypass surgery and closure of VSD with a Dacron patch were done. Patient was weaned off from bypass with dopamine 5 microgram/Kg/min ($\mu\text{g/Kg/min}$) and epinephrine 0.02 $\mu\text{g/Kg/min}$. Total cardiopulmonary bypass time, aortic cross-clamp time, and surgery duration were 73 min, 63 min, and 4 h respectively. Three units of packed red blood cell and four units of platelets concentrates were used during the surgery. After finding no flow across VSD patch by a careful transesophageal echocardiography examination suggesting successful operation, the patient was shifted to the Intensive Care Unit (ICU). Arterial blood gas (ABG) was normal except a serum lactate value of 3.6 mmol/L, which also showed a decreasing trend.

At this stage, we noticed bluish discoloration of all upper limb fingers [Figure 1], which was soon noticed in lower limb toes also. All peripheral pulses were palpable and of normal volume. Patient has a borderline cardiac output (3.8 L/min) and cardiac index (2.1 L/min/m²). A clinical diagnosis of systemic peripheral gangrene (SPG) was made. We started milrinone 0.3 $\mu\text{g/Kg/min}$ at this point for its inodilatory effect. This improved the cardiac output. Affected parts were covered with cotton pads to prevent them from trauma and to keep them warm. Considering sepsis in mind, antibiotics were escalated to meropenem, linezolid, and ciprofloxacin. Thereafter, ABG and cardiac output were repeated every 4th h. Gradually patient was weaned off from ventilator and trachea was extubated after 12 h of ICU admission. IABP tapered off and we were able to remove it at 48 h of its insertion. The fine adjustment was done in administration of fluids bolus and pulmonary artery pressure. With this maneuver, we were able to taper off inotropic support. The patient improved symptomatically with this treatment. The physiotherapist was called to provide gentle exercise of extremity joints. Lower limb ischemia turned to dry gangrene of foot, from tip of all toes to mid metatarsal on the right side, and up to the base of toes in the left side [Figures 2 and 3]. In next few days, clear demarcation line appeared between gangrenous and nongangrenous parts. After 2nd week, amputation of affected toes was done. Gradually patient improved and discharged home after 1 month of admission.

Symmetrical peripheral gangrene is characterized by sudden onset of symmetrical gangrene of fingers, toes, nose, upper lip, ear lobules, and genitals. SPG was first described by Hutchinson.^[1] It manifests as symmetrical distal ischemic damage in two or more extremity without any evidence of obstruction or vasculitis of relevant arteries.^[2] It may result



Figure 1: Bluish discoloration of both upper limb fingers



Figure 2: Systemic peripheral gangrene of anterior aspect of both feet



Figure 3: Systemic peripheral gangrene of ventral aspect of both feet

from (1) vasospastic conditions including Raynaud's syndrome, ergot poisoning, frostbite, etc.; (2) small vessel obstruction as in disseminated intravascular coagulation (DIC), presence of cold agglutinins, primary thrombocythemia, and polyarteritis nodosa; and (3) condition producing very low cardiac output.^[3]

The aggravating factors include asplenia, immunosuppression, previous cold injury to extremity, diabetes mellitus, increased sympathetic tone, and use of vasopressor.^[4]

SPG has been reported with various medical conditions such as DIC, infection (meningococcal, streptococcal, *staphylococcus aureus*, anaerobic bacteria, *Escherichia coli*, *Pseudomonas*, *Salmonella* paratyphi, *Klebsiella*, *Proteus vulgaris*, *Proteus mirabilis*, *Pasteurella multocida*, varicella, rubella, and disseminated tuberculosis), myocardial infarction, congestive heart failure, dog bite, shock, hypertension, coma, pulmonary embolism, paroxysmal ventricular tachycardia, appendicitis, Hodgkin's disease, polymyalgia rheumatica, extracorporeal shock wave lithotripsy, viral gastroenteritis, use of vasopressor (dopamine, epinephrine, and norepinephrine), plasmodium falciparum malaria systemic lupus erythematosus, small cell lung carcinoma, ergotism, CA colon, acquired hemolytic anemia, reaction to the drug (sulfamezathine and penicillin), pulmonary embolism, pneumonia, protein C deficiency, etc.^[4]

The actual reason of vascular occlusion and resulting gangrene is difficult to determine, but a low flow state is present in most of the cases, which result in occlusion of microcirculation of affected part.^[5] Pathological examination of amputated specimen often reveals thrombi concentrated in the small vessels.^[4] Once it is recognized, it is too late and amputation and debridement become inevitable. The intravenous prostaglandin (epoprostenol), tissue plasminogen activator, aspirin, vasodilator (intravenous nitroprusside and topical nitroglycerine), intravenous and local alpha-blockers, and sympathetic blockade have been used but not effective.^[4]

We covered extremities with cotton pads. It kept the extremities warm and prevented further injury to them. We escalated antibiotics and timely removed IABP and tapered off inotropes. Gentle physiotherapy was added to preserve joints mobility. These maneuvers appeared to control sepsis and increased blood flow to the extremities. Probably, it stopped further progression of gangrene and helped us to improve patient condition.

Various multiple conditions may lead to SPG, so every patient in ICU should be monitored closely for any change in the color of hands and foot along with monitoring of other vitals. Any change in color of hands or foot may indicate starting of SPG and should

be taken seriously. The adequate measure should be taken to prevent the progress of this complication at the earliest.

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