Septic pulmonary embolism following necrotizing fasciitis of the upper limb

Sir,

A 25-year-old man was first admitted in another hospital with a diagnosis of acute viral illness, septic shock, and acute lung injury. He developed hyperpigmentation of the right thumb and index finger, a day after his right radial artery was cannulated for invasive monitoring, and hence the intra-arterial cannula was removed. A color doppler study revealed normal color flow. He received noninvasive ventilatory support, vasopressors, antibiotics, pentoxifylline, and supportive care. His general condition was improving until the ninth day, when he acquired necrotizing fasciitis with clinical deterioration.

He took discharge from that hospital on the tenth day and presented to us on the same day, with chief complaints of fever, breathlessness, and swelling in the right hand and forearm with excruciating pain since two days and gangrenous changes in right thumb and index finger since eight days. There was no history suggestive of peripheral vascular disease.

On examination, he was conscious, febrile, and appeared toxic. He had a heart rate of 162/min, blood pressure 138/100 mmHg, respiratory rate 56/min, and SpO₂85% on room air. Arterial blood gas analysis (room air) showed PaO₂ 49.5 mmHg, PCO₂ 30.8 mmHg, O₂ saturation 88.5%, and pH 7.496. The chest had bilateral crepts. On local examination, a dry gangrenous change in the right thumb and index finger and blackish discoloration on the dorsum of the hand extending to the forearm were seen. There was swelling, tenderness, erythema, and raised temperature of the overlying skin. Sensation was intact; brachial, radial, and ulnar artery pulsations were palpable. On grey scale ultrasound, extensive myofascial edema was seen in the forearm and hand. X-ray chest showed bilateral infiltrates. A diagnosis of necrotizing fasciitis with acute lung injury was made and patient was shifted to the intensive care unit. Although the previous hospital records conveyed these morbidities; we investigated and reconfirmed the findings in our setting as well. He received ventilatory support (SIMV+PS+PEEP), parenteral antibiotics (meropenem, amikacin, and teicoplanin), and subcutaneous enoxaparin along with supportive care. An urgent fasciotomy and wound debridement were done on the dorsum of the right hand to relieve the compartment syndrome.

Laboratory investigations revealed hemoglobin 10 g/dL, total leucocyte count 22.3×10^{9} /l (neutrophils 90%), blood sugar (random) 342 mg/dl, serum sodium 128 mmol/l, and serum potassium 3.5 mmol/l. Liver and kidney function tests were normal. Wound and blood cultures showed growth of *Pseudomonas aeuroginosa*. No growth was seen in tracheal and urine cultures. CT angiography (right upper limb) was normal.

Following good surgical care and broad spectrum antibiotics, his wound healed. However, he continued to have spikes of high grade fever with chills and complained of chest pain while lying on his side. On the fifth day in our hospital, X-ray chest [Figure 1] showed multiple nodules (some with cavitation) and bilateral pleural effusion. CECT chest revealed bilateral, multiple, variable-sized, well-defined, nodular lesions in varying stages of cavitation, feeding vessel sign [Figure 2], right hydro-pneumothorax [Figure 3], and left pleural effusion. A diagnosis of septic pulmonary embolism (SPE) was made. An intercostal tube was inserted in the right fifth-intercostal space. Pleural fluid examination was sterile. Echocardiography was normal. He was weaned off the ventilator by the 10th day in our hospital and transferred to the ward on the 15th day. He continued to receive parenteral antibiotics based on culture sensitivity and supportive care in the surgical ward for two more weeks and was discharged after a total stay of four weeks. His progress was satisfactory on OPD follow-up.

The patient initially had septic shock followed by development of dry gangrene following intra-arterial cannulation and administration of high dose of vasopressors. A week later, he acquired pseudomonas aeruginosa necrotizing fasciitis. Other micro-organisms causing this infection include *Streptococci, Staphylococcus aureus, Escherichia coli,* and *Bacteroides*.^[1] Reisman *et al.* have reported a case of monomicrobial pseudomonas necrotizing fasciitis and reviewed 37 cases in literature.^[2] Our patient eventually developed SPE. Probably secondary to a local septic thrombophlebitis which could have been the source of emboli. Hematogenous spread of emboli containing microorganisms to the lungs caused metastatic abscesses and infarction in

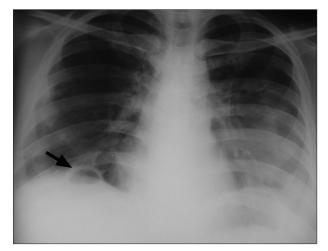


Figure 1: Chest radiograph showing multiple nodules (some with cavitation) and bilateral pleural effusion

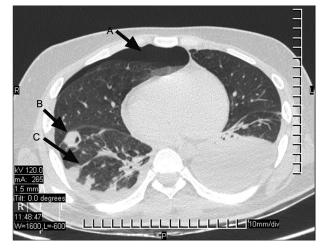


Figure 2: Axial CT image of chest in lung window depicting (a) pneumothorax, (b) cavitating nodule, and (c) feeding vessel sign in right lung



Figure 3: Axial CT image of chest depicting (a) right-sided hydro-pneumothorax with (b) loculation at base and (c) cavitating nodule in left lung

the pulmonary vasculature. The same micro-organism was isolated both from necrotizing fasciitis wound and blood culture. The diagnosis of SPE is usually suggested by the presence of a predisposing factor, febrile illness, and CT findings of multiple, nodular lung infiltrates peripherally, with or without cavitation.^[3] "Feeding vessel sign" (distinct vessel leading into the center of a nodule) on CECT chest [Figure 2] is highly suggestive and may be seen in 67-100% cases of SPE.^[4] Our patient too had a febrile illness, an extrapulmonary source of infection, a positive blood culture, and typical radiological findings suggesting SPE. Management comprises prompt administration of appropriate antibiotic therapy for 4-6 weeks and control of infectious source along with good surgical and supportive care. Anticoagulation may be appropriate in certain circumstances.^[5]

To conclude, an early diagnosis is the key to improve patient outcome and clinicians should be aware of radiological findings in SPE which serve as an invaluable diagnostic tool.

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Quick Response Code:	Website: www.joacp.org
	DOI: 10.4103/0970-9185.117080