Deep Vein Thrombosis with Staphylococcus aureus Septicemia

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Deep vein thrombosis (DVT) in children is usually associated with inherited or acquired hypercoagulable state, mechanical obstruction, fractures of long bones, central venous catheterization and prolonged immobility. We report DVT in 4 children with culture proven staphylococcal septicemia. One child died, while other three survived with appropriate antibiotics and anticoagulation therapy.

Key words: Deep vein thrombosis (DVT), Staphylococcus aureus, Septicemia.

Deep vein thrombosis (DVT) is uncommon in children and its occurrence suggests an inherited or acquired hypercoagulable defect. There are few studies on DVT with *Staphylococcus aureus* septicemia in children(1,2). We report 4 children who developed DVT with fever, unilateral thigh swelling and rapid clinical deterioration, of which 3 recovered and one died due to pulmonary embolism.

Case Report

Four boys, who were admitted in children ward with fever, cough, refusal to feed, swelling of lower limbs and single/multiple healing boils over body developed DVT. The baseline characteristics are mentioned in *Table I*. All children had swelling of thigh; 3 on right side and one on left side, positive Homan's sign and difference of girth from joint line (knee) of more than 2.5 cm. Case no. 1 and 3 had clinical features suggestive of bronchopneumonia. Cardiac examinations in all patients were clinically normal. Liver and spleen were not enlarged.

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Aerobic blood culture of all patients revealed Staphylococcus aureus, which was sensitive to cefotaxime, gentamicin and cloxacillin in case no. 2-4. Case no. 1 was sensitive to only ceftazidime and vancomycin. Doppler flow study results are also summarized in Table I. Platelet count, prothrombin time and KCCT were normal at admission in all patients. Patients were treated with antimicrobial therapy for 6 weeks as per culture and sensitivity and low molecular weight heparin during first 7 days followed by warfarin. The patients were monitored regularly with prothrombin time and KCCT maintaining INR between 2-3 and followed for 6 months for any adverse events. Antithrombin III, Protein C, Protein S and antiphospholipid antibodies measured were within normal limit.

One patient (case no.4) died on day 12 of hospitalization due to pulmonary thromboembolism after 3 days of mechanical ventilation and case no. 1 developed osteomyelitis of right femur that recovered completely with conservative measures. Case no. 2 developed multiple intracranial hemorrhages at 6 weeks, required withdrawal of warfarin therapy and improved with conservative measures. After 6 months, repeat Doppler studies showed absence of thrombus in survivors without any incompetence of ileofemoral valve.

Discussion

Staphylococcal infection causes a wide spectrum of diseases and carries a mortality of 13-27%(2,3). DVT following staphylococcal septicemia is rare in children and is usually life threatening. The process usually begins in veins of calf around valve cusps or within soleal plexus. It occurs due to release of various exotoxins; alphatoxins by many strains that act on cell membranes and produce aggregation of platelets and spasm of smooth muscle. In addition, coagulase enzyme released by staphylococci also interacts with fibrinogen and causes plasma to clot and subsequently results in DVT(3). The early color doppler flow study is important for diagnosis of DVT. The criteria for detecting DVT are failure to compress the vascular lumen and absence of normal phasic Doppler flow signals arising from changes to venous flow. The diagnostic sensitivity and

Case no.	Age (yrs)	Presenting features (duration)	Girth difference of both thigh	Color Doppler USG	Outcome
1	5.5	Pain rt.thigh-3 days Fever-3 days Resp. distress-1 day	2.8 cm	Thrombosis of common femoral & superficial femoral vein	Osteomyelitis of right femur
2	11	Fever-2 days, Swelling rt. Lower limb-1 day	g 2.1 cm	Right deep femoral thrombosis extending up to junction of right superficial vein	Intracerebral hemorrhage at end of 4th week of warfarin
3	1	Swelling of rt. Thigh- 5 days, Fever-5 days Cough-2 days	4.8 cm	Partial thrombosis of right femoral vein	Recovered completely
4	0.5	Fever-3days Swelling of lt. Thigh- 2 days Refusal to feed- 1 day	2.3 cm	Partial thrombosis of left deep femoral vein	Died on 7th day due pulmonary thormboembolism

TABLE I- Characteristics of Four Patients with Deep Vein Thrombosis

specificity of color Doppler study for DVT are approximately 98%(4,5).

Anticoagulation therapy remains the mainstay of initial treatment for DVT. Low molecular weight heparin (LMWH) prevents extension of the thrombus, significantly reduces but does not eliminates the incidence of fatal and nonfatal pulmonary emboli as well as recurrent thrombosis. Heparin therapy has little effect on the risk of developing postphlebitic syndrome. Though thrombolytic therapy (streptokinase, urokinase, rTPA) causes prompt resolution of symptoms, prevention of pulmonary embolism, restoration of normal venous circulation, preservation of venous valvular function, and prevention of postphlebitic syndrome; it does not prevent clot propagation, rethrombosis, or subsequent embolization. Furthermore, heparin therapy and oral anticoagulant therapy must always follow a course of thrombolysis(6). We used antimicrobials, heparin and warfarin in all patients. Thrombolytic agents were not considered because of high cost and nonaffordability by parents. Intracranial hemorrhage in one patient could have developed due to interaction of warfarin with macrolide (erythromycin) during follow up therapy.

Surgical therapy for DVT may be indicated when anticoagulation therapy is ineffective or contraindicated. The rationale for thrombectomy is to restore venous patency and valvular function. Thrombectomy alone is not recommended because of high risk of rethrombosis and it should be always followed by heparin therapy.

Deep vein thrombosis should be considered in children with unilateral swollen, tender limb, edema, positive Homan's sign and local musculoskeletal focus of infection. Early diagnosis by Doppler flow study and treatment with appropriate antibiotics and anticoagulation may be life saving.

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Pericardial Tamponade in Neonate Following Migration of a Sialastic Central Venous Catheter

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Central venous catheters constitute an essential part of most neonatal intensive care units (NICU). However, they are known to be associated with several complications. We here with report a rare lethal complication of pericardial effusion with cardiac tamponade occurred in a term neonate following central venous line.

Keywords: Central venous catheters, Pericardial effusion, Cardiac tamponade.

In Neonatal intensive care units (NICU), central venous catheters (CVC) play an important role in the management of an extremely preterm neonates and neonates undergoing surgical intervention. CVC are commonly used for monitoring central venous pressure, administering medications, total parenteral nutrition and for long term vascular access. However, the use of CVC have been

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associated with complications such as sepsis, thrombosis, embolism, migration of catheter tip leading to pericardial effusion (PCE), cardiac tamponade, hydrothorax and ascites(1).

Case Report

A term male baby born by normal vaginal delivery with a birth weight of 3.5 kgs was referred at 40 hours of life to our NICU in view of progressively increasing abdominal distension, bilious vomiting and non-passage of meconium since birth. Plain and contrast X-ray abdomen were inconclusive. In view of progressive decline in the clinical status, laparotomy was done, which revealed colonic atresia and was managed with resection and end-to-end anastomosis. A CVC (Sialastic 2 Fr per Q cath) was inserted electively by right femoral cut down and the position of catheter tip in inferior vena cava confirmed by check -ray and total parenteral nutrition (TPN) was started. Baby remained initially stable for 72 hrs then there was a sudden desaturation, bradycardia and cardiac arrest. With effective resuscitation neonate was revived back. A rare complication of cardiac tamponade due to CVC migration was considered and TPN was stopped immediately. Chest X-ray did showed migration of catheter tip from inferior vena cava to right ventricle, but no change in cardio thoracic ratio. The catheter was immediately pulled out and tip repositioned outside the cardiac silhouette. Echocardiography (ECHO) confirmed significant pericardial effusion with tamponade and it was intervened immediately by ECHO guided percutaneous pericardiocentesis subxiphoid resulting in 18 mL of milky white fluid. Biochemical analysis of the fluid was very much