Selcen, *et al.* [10] reported complete recovery with fluconazole in a diabetic child with mucormycosis. There is a role of newer therapies like hyperbaric oxygen, immunotherapy and iron chelation [4,8].

Mucor is angioinvasive hence surgical debridement is necessary for radical cure [1,2,4]. Most patients need repeated surgical debridement. Outcome was seen to be adversely affected in neonates where surgical debridement could not be performed [4,8].

To conclude, zygomycosis is a rare life threatening infection in neonates. It should be suspected in neonates presenting as acute abdomen. Early diagnosis combined with medical and aggressive surgical approach is the mainstay of therapy.

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Inferior Vena Caval and Right Atrial Thrombosis: Complicating Pyogenic Liver Abscess

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Vascular complication of liver abscess are rare but life-threatening. We herein report a 2 year 9 month boy with pyogenic hepatic abscess complicated by inferior vena cava thrombus extending to right atrium. Early clinical suspicion aided by ultrasonography and echocardiography confirmed the diagnosis. The child was treated successfully with timely medical and surgical intervention.

Key words: *Complication, Liver abcess, Pyogenic.*

The common complications of liver abscess include rupture into pleural, peritoneal cavity and pericardial spaces. Unusual vascular complication like inferior vena cava (IVC) thrombosis has been reported only in adult patients of amebic liver abscess but has never been reported in cases of pyogenic liver abscess [1,2]. We herein report a child with pyogenic (S*taphylococcal aureus*) hepatic abscess complicated with inferior vena cava thrombus extending to right atrium.

CASE REPORT

A 2-year-9 months-old boy presented with high grade fever, abdominal distension and cough for 4 days. On

examination, child was febrile, had pallor, with blood pressure of 108/66 mmHg, heart rate of 118/min, respiratory rate of 32/min, and was malnourished (weight <3rd percentile on WHO standards). Abdominal examination revealed tender hepatomegaly (6 cm below right costal margin) while rest of the systemic examination was unremarkable. Hematological investigations showed a hemoglobin of 6.9 g/dL and white blood cell count of 32,700/mm^3 with 87% polymorphs. Peripheral smear showed toxic granules. Liver function test showed bilirubin of 0.8 mg/dL (direct-0.3mg/dL), aspartate aminotransferase 65 IU/L (normal range, <50 IU/L), alanine aminotransferase 31 IU/L (normal range, <50 IU/L) and alkaline phosphatase of 630 U/L. Renal function tests and chest *X*-ray were normal. Ultrasonography (USG) abdomen at admission showed a large (5×4.8×5.8 cm; 120 mL), heterogeneously hypoechoic lesion with internal echoes in the segment IV and VIII of liver, suggestive of liver abscess. 25 mL of thick yellowish pus was aspirated on US guided tap. Routine microscopy of this pus revealed protein of 2.9 gm % with many polymorphonuclear cells. The child was started on intravenous antibiotics ceftriaxone, vancomycin and metronidazole. On day 2 of admission child continued to have fever, developed pedal edema and a repeat USG showed a thrombus in IVC; echocardiography demonstrated a thrombus measuring 2.25 × 1.31 cm in right atrium at the junction of IVC and right atrium with normal cardiac anatomy and function without any evidence of pericardial effusion. As the child's clinical condition deteriorated the child was taken for surgical management. Surgery was performed by cardiothoracic and pediatric surgeons. Peroperative findings were suggestive of around 100 mm^3 partially liquified liver abcess in 4th segment and a organized thrombus adherent to inflamed ostia of left superior hepatic vein extending to IVC and right atrium. Peroperative culture sent from abscess cavity grew S*taphylococcus aureus* that was sensitive to vancomycin, cloxacillin and ciprofloxacin.

Postoperatively child was managed with intravenous fluids and appropriate antibiotics for 4 weeks. Gradually child improved, fever subsided and repeat USG and echocardiography demonstrated resolution of abscess and thrombus, respectively and child was discharged on oral antibiotics for another 2 weeks.

DISCUSSION

IVC and/or hepatic vein thrombosis are infrequent but life-threatening complications of liver abscess. Budd-Chiari syndrome or pulmonary embolism could cause rapid deterioration of these patients. These vascular complications have been previously described mostly in autopsy studies. In a series of 95 autopsies of amebic liver abscess subjects, thrombosis of hepatic vein and IVC obstruction was observed in 8% of the cases [3]. Apart from this study, there have been anecdotal case reports of IVC obstruction in adult patients with amebic liver abscess [2,4,5]. Hodkinson, *et al*. [1] reported a 50-year-old man with amebic liver abcess where the patient developed IVC obstruction due to a thrombus which extended up to the right atrium [1]. Gupta, *et al*. [6] recently reported ALA complicated by IVC thrombus in a 6-year child. The pathophysiology of vascular thrombosis is uncertain. The proposed mechanisms are external compression coupled with contiguous spread of inflammation over the vessel wall resulting in endotheliitis, which predisposes to stasis and thrombosis [5].This endothelial damage is further accentuated by respiratory movements of the diaphragm and coughing and can contribute to thrombus formation in IVC [7].

While evaluating a child with thrombosis it must be assumed that interplay of multiple factors play a role in evolution of thrombus; in most cases, some insult to endothelium (*e.g.* indwelling vascular catheter, infectious vasculitis) as well as some dysregulation of coagulation (*e.g.* sepsis, congenital or acquired coagulation protein abnormalities) will be present [8]. Therefore most pediatric thrombotic patients should be investigated for congenital or acquired coagulation protein abnormalities (*e.g.* Protein C and S, lupus anticoagulant, total cholesterol, antiphospholipid antibodies). It is also essential to remember that many abnormal results in acute phase (*e.g.* consumption of Protein C and S secodary to thrombotic event) need to be confirmed by repeating atleast 3-6 months after acute thrombotic event [8]. In our case, sepsis-induced endothelitis seems to be the most important factor for thrombosis; it is likely that the inflammatory process in the wall of the abscess spread directly to the adjacent wall of the right hepatic vein which later propagated into the IVC and right atrium. Work-up for procoagulant states has been planned at follow up.

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Hematidrosis

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Hematidrosis is an extremely rare clinical entity characterized by recurrent episodes of skin bleeding mixed with sweat. We report a case of hematidrosis in a 10-year-old girl where detailed laboratory and histopathological investigation revealed no abnormality. The girl was successfully treated with propranolol with no recurrence of bleeding over a follow-up of 3 months.

Keywords: *Child, Hematidrosis , Propranolol.*

Hematidrosis is a extremely rare and enigmatic disorder characterized by recurrent episodes of self limited bleeding from skin. Though, classically hematidrosis means blood in sweat, practically blood is mixed with sweat like material rather than true sweat in this condition [1]. Till date only nine cases are reported in literature. We report a case of hematidrosis diagnosed and treated successfully.

CASE REPORT

A 12-year-old girl was referred to our hospital with complaints of recurrent episodes of spontaneous skin bleeding for last one month. The bleeding was spontaneous from different sites of body including face, limb, palm and sole but not from mucous membrane. The consistency of bleeding was little thinner than blood and stopped as soon as the site was wiped, leaving behind no oozing site. It was occurring both day and night but never after she slept.

During hospitalization she had more than 10 instances of spontaneous intermittent bleeding per day that was evidenced by almost all of our on duty doctors and nurses. Her medical records were non-contributory with no known underlying disease. Her development was normal with normal tanners staging and menarche not yet started.

Complete blood count, and blood biochemistry was normal, with normal coagulation screening tests. Platelet aggregation test and estimation of von-Willebrand factor revealed no abnormality. Assay of anti-nuclear antibody and interleukins was done with a view to exclude underlying autoimmune conditions like vasculitis, but revealed no abnormality. Microscopic examination of bloody exudates from face revealed the same components as of peripheral blood including red blood cells, leukocytes and platelets mixed with epithelial cells. Skin biopsy performed from the bleeding area in palm immediately after the bleeding revealed irregular acanthosis of epidermis with broadening of rete pigs along with hypergranulosis and marked hyperkeratosis with edema of superficial dermis, similar description toin a previous case [3]. There was no abnormality in sweat gland or sebaceous gland.

The girl was treated initially with diazepam without any significant improvement. Finally, based on a previous case [5], she was treated with propranolol in a dose of 1 mg/kg/day in two divided dose with a baseline ECG and monitoring of heart rate. The bleeding episodes started to reduce within 2 days of therapy and stopped completely within 6 days. There was no recurrence of bleeding within three months of follow-up.

DISCUSSION

Hematidrosis is a condition in which capillary blood vessels that feed the sweat glands rupture, causing them to exude blood [2]. The term hematofolliculohidrosis has