

9 Tropical Infections in ICU

Abstract: Many tropical infectious diseases present with acute onset illness in previously healthy adults who are usually young and who work to support the family and society. Any serious consequence of such a short lasting illness creates a big gap in the family. At the same time, timely diagnosis and prompt interventions can make a world of change and ensure good recovery. While many articles describe isolated attempts at describing the pathophysiology and management in individual illnesses, a consolidation of the available information is attempted in this article. Major clinical syndromes like bleeding, respiratory distress, hypotension, shock, alteration of consciousness, etc. are analyzed. Some common illnesses like leptospirosis, malaria, dengue fever, Japanese encephalitis, rabies, tetanus, varicella and HIV disease are discussed with special reference to the emergency management of the contingencies that come with them. That all diseases and all details about them cannot be discussed in a small session like this may be accepted. It is emphasized that the specific management of these illnesses should follow the general guidelines. But an attempt is made to include the interventions which may be life saving in many instances. The need for proper and timely communication regarding the prognosis and the need for regular monitoring and periodic review of diagnosis and management needs to be stressed. The usual topic of nosocomial infections in the ICU has been omitted.

Intensive care (critical care) involves taking care of patients with serious life threatening illnesses. The speciality of critical care is unique in that it tries to involve other subjects and fields and now includes almost all major surgical and medical groups with the anesthetist playing a major role. As our country is facing newer tropical diseases in the form of new and re-emerging infectious diseases in sporadic and epidemic proportions, many of the Intensive Care Units are getting filled with patients with these type of illnesses, particularly during epidemics. These patients develop the illness when they are usually otherwise healthy, in their prime years of activity. At that juncture, any unfavorable outcome is unacceptable and hence the need for critical life saving care is of paramount importance. These patients are likely to have compensated organ systems as a result of the acute toxic / microbiological onslaught and the homeostatic mechanisms may be at fault. Organ failure in one system may contribute to damage in other systems. For example, a person with respiratory failure may have cardiac, renal and central nervous systems getting affected. Renal / Hepatic involvement may produce metabolic disturbances affecting other organs. The complex interplay of these organ systems are also complicated by the ongoing microbial replication, which may continue inspite of our best efforts. The diseases with multisystem involvement like leptospirosis, dengue fever or malaria pose diagnostic confusions in deciding the etiological factors as well. The diagnosis is usually confirmed by serology, which may not be conclusive in the first few days. The usage of antimicrobials in patients with multisystem dysfunction may fail to achieve the desired therapeutic effect, leave alone the adverse drug reactions that are likely. The fact that these infectious diseases are common in the lower socioeconomic strata and that it is usually the breadwinner who is bedridden, makes matters worse. The economic loss measured in DALYs (Disability adjusted life years) lost will be augmented by the time and money spent in the ICUs. Considering all the factors, it becomes evident that proper management of infectious disease related problems in the ICU is extremely important.

In this discussion, the problems associated with hemodynamic modifications, renal, pulmonary and central nervous system involvement and hematological alterations will be discussed first followed by a few select diseases like leptospirosis, dengue fever, hanta fever, malaria, tuberculosis, tetanus, varicella and rabies will be discussed.

Fever with Bleeding Disorders

In our country, the commonest in this category would be thrombocytopenia associated infections. These include dengue fever, leptospirosis, malaria, rickettsial infections and common viral fever. Exotic infections like Yellow fever, Ebola, Marburg, Lhassa, Hanta virus, Crimean-Congo HF and Omsk fevers also produce well known hemorrhagic fevers. Other viral infections that have been associated with thrombocytopenia include mumps, rubella, rubeola, varicella, disseminated herpes simplex, CMV infection and infectious mononucleosis. Septic vasculitis, as in gram-negative bacterial sepsis (i.e., meningococemia) may also cause hemorrhage. Thrombocytopenia can occur due to defective production, excessive destruction or sequestration. In many of the above conditions, a combination of all these may occur. The associated destruction of capillary endothelium paves the way for early development of purpurae, bruises and bleeds.

Viral and bacterial infections may influence hemostasis and can lead to thrombohemorrhagic complications or syndromes such as DIC, hemolytic uremic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP), or vasculitis. Symptoms and signs may be dominated by bleeding, thrombosis, or both. The patient may present with massive GI, pulmonary or intracranial bleed which necessitates ICU care. The hemorrhagic pneumonitis in leptospirosis and Hanta fever may immediately drown the patient in one's own hemorrhagic pulmonary fluid. The massive GI bleed may precipitate hepatic encephalopathy in a jaundiced patient with leptospirosis. Massive bleed can also aggravate the hypotension and hypoperfusion of the kidneys / brain in leptospirosis.¹⁻³

The underlying mechanisms in dengue associated bleeding are not fully understood. Thrombocytopenia is universal in dengue fever and is one of the diagnostic clinical criteria by WHO. Platelet function is abnormal in dengue infections. In the acute phase of disease, platelet aggregation in response to ADP is impaired, and concurrent increases in plasma thromboglobulin and platelet factor 4 indicate that platelet secretory activity is increased. The duration of survival of transfused platelets is markedly shortened, and immune complexes containing dengue antigen have been found on platelet cell surfaces. Mild prolongation of the prothrombin and partial thromboplastin times and reduced fibrinogen levels have been noted in several studies, but levels of fibrin degradation products are not elevated to a degree consistent with classical disseminated intravascular coagulation. Variable reductions in the activities of specific coagulation factors, including factors II, V, VII, VIII, IX, X, antithrombin have been demonstrated during the acute phase of DHF in small numbers of patients.⁴

Vasculitis may be triggered by infection. The following viruses are known to infect endothelial cells. Dengue virus, herpes simplex virus, ebola virus, poliovirus, marburg virus, adeno-

virus, Hantaan virus, Parainfluenza virus, cytomegalovirus, echovirus, measles, human immunodeficiency virus, human T cell leukemia virus type 1 and mumps. Vascular changes may occur from infarction secondary to occlusion by thrombi of the lumen of small blood vessels. Vascular occlusion may lead to ischemic tissue injury or bleeding. Severity may vary from ecchymosis or oozing from arterial or venous puncture sites to more general complications, such as petechiae, purpura, ecchymosis, gum or gut bleeding, hemoptysis, or even adrenal bleeding (as in Waterhouse-Friderichsen syndrome) producing multi organ failure⁴. The clinical signs are not specific for any one etiology.

In systemic gram-negative and gram-positive bacterial infections, activation of coagulation is mediated via the extrinsic TF pathway. As a rule, coagulation and fibrinolysis occur independently of one another, and the overall result is usually a procoagulant tendency. Discussions on the important roles of endotoxin and specific cytokines have already led to clinical therapeutic trials with selective inhibitors of the TF pathway (monoclonal antibodies, Fab fragments, modified factor VIIa, nematode anticoagulant protein, TFPI). Studies with anti-TF antibodies and TFPI in primates have shown that, in addition to inhibition of coagulation activity, these agents may have significant anti-inflammatory properties and may markedly reduce mortality in otherwise lethal infections.⁵

Bleeding and coagulation abnormalities are seen in less than 5% of patients with severe falciparum malaria.⁶ It can be due to thrombocytopenia and/or disseminated intravascular coagulation. Mature parasitized red cells and cytokines activate the coagulation cascade. Accelerated coagulation cascade, consumption of antithrombin III, increased concentration of FDP and increased splenic clearance of platelets contribute to the coagulopathy and thrombocytopenia in malaria. Hypofibrinogenemia due to DIC occurs in less than 5% of patients. Thrombocytopenia is commonly seen in severe falciparum malaria. It is presumed to be due to increased consumption of the platelets in the periphery, may be in the spleen. Bone marrow shows appropriate megakaryocyte response. However, bleeding due to thrombocytopenia is very rare in malaria.^{7,8} Generally, the platelet count returns to normal with the completion of antimalarial chemotherapy.

Fever with Respiratory Distress

Respiratory distress happens to be a very common indication for transferring a patient to ICU in all our hospitals. Availability of mechanical ventilators and trained staff in the ICU only is one of the reasons for this. The average mortality of ARDS is 50%, and vary from 30 to 70% even with optimal conventional therapies. ARDS is characterized by arterial hypoxemia, bilateral infiltrates on chest radiographs, and a normal pulmonary artery occlusive pressure. This is the uniform

severe inflammatory response to a large number of pulmonary or extrapulmonary insults. The lung response to injury is stereotyped, with transition from acute alveolar capillary damage to a late proliferative phase, quite independent of the initial cause. A direct insult results in alveolar damage that includes edema, fibrin, collagen, neutrophil aggregates, and red cells seen in the alveoli, whereas the "indirect" insult results in prevalent microvascular congestion, interstitial edema and less severe alveolar damage.

Recent outbreaks of leptospirosis with pulmonary forms have drawn attention, mainly in tropical countries, and respiratory distress or hemoptysis emerge as lethal complications. In India, the fatality rate of complicated pulmonary forms reached 43%. In Brazil, 55% of patients mechanically ventilated for acute lung injury died. In Seychelles, Thailand, and Argentina, rapidly progressive pulmonary hemorrhage was the main cause of death. Hemoptysis originates in alveolar hemorrhage, whose primary cause is diffuse vasculitis with endothelial damage in the septal capillary bed, leading to alveolar flooding and asphyxiation. In such patients with pulmonary hemorrhage, acute respiratory distress syndrome (ARDS), shock, and multiple organ dysfunction, the priorities are to stop alveolar bleeding, restore gas exchanges, and provide hemodynamic support.¹⁻³

Pulmonary edema is a grave complication of severe malaria, with a high mortality (over 80%).⁶ In African children, respiratory distress is an important risk factor for fatal outcome. Metabolic acidosis is a major cause of respiratory distress in these children, but pneumonitis from the sequestered parasitized red blood cells and inflammatory cells in pulmonary microvasculature may also contribute to respiratory distress. In adults, noncardiogenic pulmonary edema and acute respiratory distress syndrome (ARDS) with normal pulmonary artery occlusion pressure are grave complications of falciparum malaria, with a high mortality rate. Pulmonary edema may also arise iatrogenically from fluid overload. In some patients, ARDS is present at admission and is associated with high parasitemia. However, in many instances, ARDS commences few days after treatment has begun, when peripheral parasitemia has decreased or disappeared. Until recently, it was thought that vivax malaria did not cause pulmonary complications; however, there are now many reported cases of ARDS occurring after commencement of therapy for *Plasmodium vivax* malaria and 1 case of ARDS complicating *Plasmodium ovale* malaria. Microvascular sequestration of parasitized red blood cells is thought to be the pathophysiological mechanism underlying most extrapulmonary organ-specific manifestations of severe falciparum malaria. Red blood cells parasitized with *P. vivax* or *P. ovale* do not cytoadhere to endothelial cells, so the occurrence of ARDS in vivax malaria suggests that lung injury in malaria is not caused solely by microvascular sequestration of parasitized red blood cells. Noncardiogenic pulmonary edema often develops rapidly days after starting antimalarial therapy. Gas transfer in *P. falciparum* infection decreased after initiation of treatment, suggesting that alveolar-capillary pathology may be exacerbated by a post-treatment inflammatory response, analogous to ARDS after treatment of bacterial sepsis. Lung ischemia-reperfusion injury may occur after antimalarial drug clearance of sequestered parasitized red blood cells and monocytes in microvasculature. ARDS in vivax malaria also develops after initiation of treatment, which is consistent with a similar treatment-exacerbated inflammatory process.⁸

Patients with Hantavirus pulmonary syndrome typically present in a very nonspecific way with a relatively short febrile prodrome lasting 3-5 days. Within 24 hours of initial evaluation, most patients develop some degree of hypotension and progressive evidence of pulmonary edema and hypoxia, usually requiring mechanical ventilation. HPS has a characteristic radiological evolution, beginning with minimal changes of interstitial pulmonary edema, progressing to alveolar edema with severe bilateral involvement. Pleural effusions are common and are often large enough to be evident radiographically.⁹

Various types of ventilatory support may have to be instituted in all of the above conditions depending on various factors

- Continuous positive airway pressure (CPAP). Oxygen is delivered to the spontaneously breathing patient under pressure via a tightly fitting face mask (noninvasive positive-pressure ventilation, NIPPV) or endotracheal tube. Oxygenation and vital capacity improve and the lungs become less stiff.
- Noninvasive positive-pressure ventilation (NIPPV) has been shown to be of use in patients with hypercapnic respiratory failure secondary to acute exacerbations of COPD who do not require immediate intubation and ventilation. NIPPV should be instituted at an early stage in the hospital admission when the pH falls below 7.35 and the respiratory rate exceeds 30 breaths per minute. NIPPV is usually given for at least 6 hours a day, and oxygen is administered to maintain arterial oxygen saturation above 90%. NIPPV reduces the need for intubation, complications, mortality and hospital stay.
- Intermittent positive-pressure ventilation (IPPV) requires tracheal intubation and, therefore, anesthesia if the patient is conscious. The beneficial effects of IPPV include improved carbon dioxide elimination, improved oxygenation, and relief from exhaustion as the work of ventilation is removed. High concentrations of oxygen (up to 100%) may be administered accurately. If adequate oxygenation cannot be achieved, a positive airway pressure can be maintained at a chosen level throughout expiration by attaching a threshold resistor valve to the expiratory limb of the circuit. This is known as positive end-expiratory pressure (PEEP), and its primary effect is to re-expand underventilated lung areas, thereby reducing shunts and increasing PaO₂.
- Intermittent mandatory ventilation (IMV). This technique allows the ventilated patient to breathe spontaneously between mandatory tidal volumes delivered by the ventilator. These coincide with the patient's own respiratory effort. It is used as a method of weaning patients from artificial ventilation, or as an alternative to IPPV.

Aerosolized surfactant, inhaled nitric oxide and aerosolized prostacyclin are experimental treatments whose exact role in the management of ARDS is unclear.¹⁰

Fever with Shock

Many tropical infections (Leptospirosis, dengue fever, Chikungunya fever, malaria, diarrheal diseases) are associated with hypovolemia, hypotension and shock. During infections, a myriad of mediators induce vasodilatation, increase microvascular permeability, and capillary leakage of serum proteins. This leads to reduced intravascular volume, preload, cardiac output, and results in inadequate tissue perfusion and oxygenation. The predominant hemodynamic feature of septic shock is arterial vasodilation. Diminished peripheral arterial vascular tone leads to dependency of blood pressure on cardiac output, causing vasodilation to result in hypotension and shock if insufficiently compensated by a rise in cardiac output. Factors responsible for myocardial depression of sepsis are myocardial depressant substances, coronary blood flow abnormalities, pulmonary hypertension, various cytokines, nitric oxide, and beta-receptor down-regulation. The arterial-mixed venous oxygen difference usually is narrow, and the blood lactate level is elevated. The basic pathophysiologic problem seems to be a disparity between the uptake and oxygen demand in the tissues, which may be more pronounced in some areas than in others. Sepsis leads to regional changes in oxygen demand and regional alteration in blood flow of various organs. Development of increased systemic microvascular permeability also occurs, remote from the infectious focus, contributing to edema of various organs, particularly the lung microcirculation and development of acute respiratory distress syndrome (ARDS). Maldistribution of blood flow, disturbances in the microcirculation, and, consequently, peripheral shunting of oxygen are responsible for diminished oxygen extraction and uptake, pathological supply dependency of oxygen, and lactate acidemia in patients experiencing septic shock.

Very important points in the management of hypovolemia may be recalled. Mild to moderate blood loss (less than one liter in an adult), should be replaced by colloid (1-1.5 times volume of

blood lost) or crystalloid (crystalloid 2.5-3 times volume of blood lost). Crystalloid of choice is normal saline or lactated Ringer's solution because its osmolality is similar to that of the intravascular volume. In large-volume resuscitation, however, excessive normal saline infusion may produce hyperchloremic metabolic acidosis. Colloid solutions (5% albumin and hetastarch) offer the most efficient intravascular volume expansion, but are expensive and no outcome benefit has been shown. Dextrose 5% in water does not offer significant expansion of intravascular volume because it is quickly distributed throughout body fluid compartments¹¹.

Dopamine is indicated if fluid replacement alone is not enough or when the response is not fast. Dopamine is started in a dose of 5 mcg/kg/min and increased if necessary to 15-20 mcg/kg/min. The addition of dobutamine (5-10 mcg/kg/min) may be beneficial. Use of corticosteroids in the treatment of septic shock has been controversial. Many studies in the 1980s and early 1990s generally showed no significant improvement with corticosteroid therapy, and some even showed increased morbidity. However some investigations recently have focused on use of more modest doses of corticosteroid in patients with refractory shock despite adequate resuscitation. However, the disadvantages must be kept in mind. Other drugs like Activated Protein C (Drotrecogin alpha), Monoclonal antibody to endotoxin, antibody to IL-1 receptor, antibradykinin, antiplatelet activating factor, anti-tumor necrosis factor have not stood the test of time in repeated studies.¹²

Fever with Hepatorenal Involvement

In many parts of India and the tropics, Leptospirosis is diagnosed only when the patient presents with hepatic and renal function derangement. This happens to be the commonest indication for such a patient in the ICU as well. However, malaria, scrub typhus and fulminant hepatitis also must be considered in these cases. Occasionally, patients with typhoid fever also presents with similar manifestations. While elevated bilirubin and creatinine values are the clues, oliguria, hematuria, altered consciousness, irritability and presence of visible hemorrhages in the skin and mucous membranes (including conjunctiva) must also alert the physician to think of this situation. Hemolytic uremic syndrome, drug (usually NSAID) induced disorders can also mimic the clinical picture. Alcoholic or other chronic diseases of the liver may also present with co-existing renal failure. The occurrence of non oliguric renal failure should not be missed. These patients might also have bleeding abnormalities due to coagulation factor abnormalities or associated thrombocytopenia or even uremia associated platelet dysfunction.

In leptospirosis the organism gains access to the kidney, it migrates to the interstitium, renal tubules, and tubular lumen causing interstitial nephritis and tubular necrosis. When renal failure develops, it usually is due to tubular damage, but hypovolemia from dehydration and from altered capillary permeability also can contribute to renal failure. Liver involvement is seen as centrilobular necrosis with proliferation of Küpffer cells. Jaundice may occur as a result of hepatocellular dysfunction. The hyper-bilirubinemia in leptospirosis tends to be more severe, as there will be contributions from intra and extra vascular hemolysis, hemorrhages and from the myoglobin released from the inflamed muscles.

Fever with CNS Involvement

The common tropical infectious diseases under this category are Cerebral malaria, Japanese Encephalitis, Typhoid fever and viral meningitides. Pyogenic meningitis and other intracranial infections and occasionally cerebrovascular accidents may be overlooked in some cases. Delirium and seizures might occur in patients with hepatic encephalopathy, alcohol withdrawal or even metabolic encephalopathies (like hyponatremia, hypoglycemia etc.). The common pathophysiologic mechanisms in infections are similar. In JE, neurologic invasion develops, possibly by growth of the virus across vascular endothelial cells, leading to involvement of large areas of the brain, including the thalamus, basal ganglia, brainstem, cerebellum, hippocampus,

and cerebral cortex. Many tropical infections may present with post infective demyelination presenting as weakness or sensory disturbances. (See description under varicella)

Management of Tropical Infectious Diseases in ICU

Detailed presentation and management of all tropical infectious diseases is beyond the purview of this discussion. An attempt is made to bring up common ICU related management in emergency situations.

- (a) *Leptospirosis*: As has been discussed before, the common problems encountered in the ICU are:
- (i) hypotension requiring vasoactive support (with dopamine/dobutamine);
 - (ii) thrombocytopenia- with hemorrhage requiring platelet transfusions [though not needed in all cases- except when there is active bleeding (other than sub conjunctival hemorrhages);
 - (iii) renal failure with oliguria- uremia- hyperkalemia requiring emergency dialysis (hemo, peritoneal or CVVHD);
 - (iv) Hepatic encephalopathy management especially in patients with underlying alcoholic or chronic liver disease;
 - (v) myocarditis requiring continuous cardiac monitoring and management of arrhythmias; and
 - (vi) ARDS (usually with hemorrhagic pneumonitis) requiring urgent and intensive ventilatory support and pulse dose steroid therapy.¹³

Treatment using crystalline penicillin in doses of 1.5MU every six hours may be supplemented by other measures as noted elsewhere depending on the system that is affected. Doxycycline is the recommended substitute in cases of hypersensitivity to penicillins, even though many antibiotics are reported to be useful. Fluid administration must be done cautiously considering the implications on the kidney, heart, lungs and brain. Recently desmopressin has been reported to be effective in controlling massive hemoptysis in leptospirosis.¹⁴

- (b) *Dengue Fever* is usually associated with hypovolemic shock, positive tourniquet test, massive hemorrhages, intracranial bleeds or, occasionally, with multi organ failure. The care will necessitate platelet transfusions restricted to cases with active bleeding, rapidly falling platelet count, particularly in those vulnerable for bleeds. Inotropic and hydration support with intravenous fluids may be necessary where the "capillary leak" has led to fall in blood pressure or a rise in hematocrit.^{15,16} These patients may also need care for the associated intracranial problems.
- (c) *Malaria*: Patients may present with various complications like hypotension, hemorrhages, altered levels of consciousness, renal failure, and even hepatorenal syndrome. Deep coma, seizures, anuria, hypoglycemia, lactic acidosis, thrombocytopenia with platelets <50,000 cmm, bilirubin >3.0 mg/dl, heavy parasitemia >100000/microliter are suggested as bad prognostic features. Metabolic acidosis resulting both from lactic acidosis and acute renal failure is a major cause of death in severe malaria¹⁷. Peritoneal dialysis provides much slower correction of metabolic abnormalities than hemofiltration. Hemodialysis should be similar to hemofiltration in its efficiency. The management must always be prompt with early suspicion and quick installation of parenteral antimalarials like IV quinine, IV artesunate or IM artemether.¹⁸ The possibility of drug resistance and adverse reactions must be major considerations in choosing therapy. Exchange transfusions have been recommended in cases with massive parasitemia (usually in falciparum infections). Continuous Renal replacement therapy has been observed to be associated with good recovery.¹⁹
- (d) *Japanese Encephalitis* and other neurological emergencies must be handled with proper airway maintenance, volume support, anticonvulsants (when indicated) and anti edema measures. No scientific evidence exists for the use of antivirals or steroids in this condition.

- (e) *Tetanus* may present in our ICUs either following a confirmed diagnosis with a history of contaminated wounds or with generalized tonic-clonic spasms. Maintenance of airway (with tracheostomy, if needed) is the most important step. Parenteral or nasal feeding is very important. In cases refractory to large dose diazepam, complete skeletal muscle relaxation (with curare mimetics or midazolam) with complete assisted ventilation is suggested. These patients will stage a complete recovery if kept alive for three weeks. Specific care of the wounds with local care and antibiotics and early administration of anti-tetanus immunoglobulin are very important.
- (f) *Rabies* may present to the ICU without a history of animal bite as well.^{20,21} Combined occurrence of hydrophobia and aerophobia in a conscious patient must alert any physician to this occurrence. The patients may come with a paralytic illness as well. The dismal outcome of patients with rabies provides little optimism for heroic efforts. Palliative therapy is of paramount importance in this fatal disease. In cases of severe respiratory distress, assisted ventilation with total anesthesia may be considered to reduce the torture they usually suffer from. The dismal outcome of patients with rabies provides little optimism for heroic efforts. The following patient characteristics and resources could be considered favorable in making the decision to embark on an aggressive course of therapy:
1. Administration of proper rabies vaccine before the onset of clinical rabies.
 2. Presentation with a very early stage of disease, including paresthesias or pain at the site of a previous bite exposure, with minimal other neurological symptoms or signs.
 3. Good health and absence of chronic disease.
 4. Relatives who accept the high probability of an unsuccessful outcome and the possibility of disabling neurological deficits in a rabies survivor.
 5. Access to adequate resources and facilities. There may be situations in which an aggressive approach is desirable, in particular during a very early stage of the disease, although the probability of failure is very high. Combination therapy may be superior to therapy with a single agent. Severe edema associated with a risk of brain herniation is rare in patients with rabies, although this is a potential complication of intrathecal therapy with HRIG.²⁰ Combination therapy for a patient with rabies might include administration of rabies vaccine (multiple-site intradermal route), HRIG (intramuscular), ribavirin (intravenous and intraventricular via Ommaya reservoir), IFN- (intravenous and intraventricular via Ommaya reservoir), and ketamine (intravenous infusion). Therapy with corticosteroids should be avoided.
- (g) *Varicella* cases may need to be handled in separate rooms for isolation. Complications in varicella and herpes zoster are limited exclusively to those who refuse to get treated with acyclovir or any other antiviral drugs. The interstitial pneumonia associated with Varicella may require IPPV and large dose steroids. The encephalitis caused by varicella is usually complicated by the associated metabolic problems like hypoglycemia, dehydration, hyponatremia, hypokalemia etc. induced by unscientific dietary restrictions. In all such cases, timely institution of parenteral acyclovir is very useful. Steroids may be indicated in cases with post-infective demyelination following varicella where various neurological problems may occur. IV Gamma Globulin and steroids are good adjuvants to therapy in such cases.
- (h) *HIV disease* is very common now. HIV infected patients may present to the ICU with serious respiratory problems in PCP (with classical mid-zone infiltrates in chest X-ray), extensive pulmonary tuberculosis, extensive mediastinal lymphadenopathy (tuberculous or neoplastic).^{22,23} They may report with severe bleeding at various sites (including GI or CNS) where massive bleeds can occur from diseases like Kaposi's sarcoma or other neoplasms. Various other neoplasms may also weaken the patient. They might develop serious bone marrow suppression from infections, neoplasms or drugs and may require massive transfusions or hematopoietic drugs. Severe metabolic problems like lactic acidosis must be kept in mind in a patient on anti-retroviral therapy. Neurological illnesses are quite common

in advanced HIV disease. This includes strokes, ICSOLs, meningitis and encephalitis by various organisms. The management of these problems are to be done in line with the recommendations in any other patient. It may be remembered that even steroids may have to be used depending on certain indications. Very aggressive antimicrobial therapy may have to be resorted in many of these cases.

CONCLUSION

The intensive care units in developing countries have to be equipped with personnel, facilities and equipments to care for the many common infectious disease problems listed above. In addition rare and exotic infections like SARS, avian influenza, Ebola, Ross river, Chikungunya, Nipah virus disease and many other arboviral / zoonotic diseases might create emergencies in our ICUs. A basic understanding about the pathogenesis and management of complications is necessary for any internist practicing in these areas. The management relies on early suspicion and diagnosis by clinical and microbiologic methods. The institution of specific therapy must be complimented at times by empiric drugs. Along with treatment of the underlying disease, critical care guidelines for management of critical organ involvement must also be followed. Proper critical (intensive) care can bring the life back to many earning members of the society and many families may be put back to the tracks in short time.

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MULTIPLE CHOICE QUESTIONS

1. In a patient presenting with sub conjunctival hemorrhage, jaundice, and oliguria the most important specific test may be:
A. IgM ELISA
B. Periph. Blood Smear
C. QBC
D. ABG
 2. The most important biochemical abnormality in a malaria patient in the ICU:
A. Hyponatremia
B. Lactic acidosis
C. Hypoglycemia
D. Hyperkalemia
 3. The chest X-ray finding suggesting PCP is:
A. Massive pleural effusion
B. Localised pneumothorax
C. Midzone infiltrates
D. Lower lobe cavities
 4. Desmopressin is indicated in the management of Leptospirosis associated with:
A. Renal failure
B. Massive hemoptysis
C. Hypotension
D. ARDS
 5. A paralytic illness with rapid progression is suggestive of which zoonosis:
A. Leptospirosis
B. Dengue fever
C. Rabies
D. Cryptococcal meningitis
 6. ARDS may occur with:
A. Leptospirosis
B. Malaria
C. Hantaa virus disease
D. All of the above
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1. A 2. C 3. C 4. B 5. C 6. D