Scientific Sessions 2015
Faculty of Medical Sciences
University of Sri Jayewardenepura

02nd April 2015

6972

Detailed Programme

8.00 am Registration

8.30 am Inauguration

8.35 am **Plenary Lecture**

Open surgery – A thing of the past
Prof. Aloka Pathirana
(Citation read by Prof. S. Yasawardene)

9.00 am **Symposium I - Challenges in Infectious Diseases**

Chairpersons: Prof. Antoinette Perera, Prof. Neluka Fernando

1. Challenges in malaria elimination and prevention of re-introduction
Prof. Rajitha Wickremasinghe

2. Antibiotic resistance – New challenges
Dr. Geethika Patabendige

3. Managing dengue in paediatric patients - The pitfalls and challenges
Prof. Dulanie Gunasekera

10.10 am Tea and viewing of poster presentations

10.30 am **Oral presentations - Session I (OP 1 – OP 6)**

Chairpersons: Prof. Sagarika Ekanayake, Prof. Renu Wickremasinghe

OP1 **Sphingosine 1-Phosphate in acute dengue infection**
Gomes PLR[1], Fernando S[1], Fernando RH[1], Wickramasinghe N[1], Shyamali NLA[1], Ogg GS[2], Malavige GN[1]
[1]Faculty of Medical Sciences, University of Sri Jayewardenepura,
[2]Weatherall Institute of Molecular Medicine, Oxford

OP2 **Development and validation of an in house multiplex real time PCR for quantification of all four serotypes of dengue virus**
Gomes PLR[1], Kamaladasa A[1], Ogg GS[2], Malavige GN[1]
[1]Faculty of Medical Sciences, University of Sri Jayewardenepura,
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Abstract

Managing DHF –The pitfalls & challenges

Dengue Haemorrhagic Fever (DHF) afflicts healthy children, has an unpredictable course and is plagued by challenges in diagnosis and management. The first challenge is to differentiate dengue from other viral fevers since both may be clinically identical. However, the widespread availability of Dengue NS1 antigen and IgG /IgM antibody tests have overcome this problem.

The next problem is to differentiate dengue fever(DF) from DHF. The objective clinical signs of DHF are those of circulatory insufficiency- increase in heart rate, low volume pulse, low pulse pressure (<20mmHg) and prolonged CRT (>2 secs), accompanied by a 20% rise in haematocrit (HCT). However, a patient's base line HCT is rarely available, and therefore has to be assumed. This could create confusion in the case of anaemic patients, where a "normal" HCT really means a 20% elevation. During the febrile phase, FBC and platelet counts decrease, reaching lowest levels during the critical phase, with platelet counts usually below 100,000/cumm. Neutropenia with a relative lymphocytosis also occur during the critical phase. Platelet count may continue to drop till the convalescent period.

Management of DHF is entirely symptomatic and knowing the clinical sequence is essential; the febrile phase(2-7 days) resolves by crisis. The critical phase (leakage phase) occurs when temperature returns to normal and lasts **only** about 48 hours. Thus monitoring should be intensified when fever resolves.

The key to management is meticulous monitoring and adequate fluid replacement. There is no formulae to predict the rate of leakage, but empirically, rapid leakage occurs in the first 12 hours of the critical phase. Rates of leakage may vary, so there is no alternative to continuous monitoring and replacing fluid loss with isotonic crystalloid (normal saline) solution. Crystalloids sometimes continue to leak out of the vascular compartment;then, colloid transfusions (Dextran 40) are indicated. Large volumes of fluid may collect in pleural and peritoneal cavities during leakage.

Another pitfall is concealed internal bleeding(eg. into the gut lumen). During the critical stage, blood loss may not produce a low HCT. Since it is generally high due to leakage, a "normal" HCT in a leaking patient may mean significant blood loss. Hence, a careful evaluation of circulatory signs and rapid replacement of blood is essential in this situation, to avoid shock and metabolic acidosis.

48 hours after the onset of critical phase, leakage stops and fluid re enters the intravascular compartment (convalescent phase). Thus paradoxically, 48 hours after rapid IV fluid replacement, infusions have to be **reduced and stopped**; if not, severe pulmonary oedema ensues. Monitoring should be continued into the convalescent stage to detect and treat early pulmonary oedema. Since reabsorption is also erratic, Furusamide should only be given on a 'PRN' basis.

Most DHF patients with no shock/compensated shock recover completely. Those with non compensated shock have high morbidity and mortality, with a predisposition to fulminant hepatic or renal failure, DIC or dengue encephalopathy. Conversely, prolonged/excessive IV fluid therapy may precipitate pulmonary oedema in a convalescing patient.

The key to management of DHF is meticulous monitoring, and timely, judicious replacement with isotonic fluid. Till dengue vaccines become a reality and vector control is completely effective, following these rules of management will save many lives.