DENGUE FEVER. LOCAL EXPERIENCE AT HYATABAD MEDICAL COMPLEX, PESHAWAR

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ABSTRACT

Introduction: Dengue fever is caused by a single stranded RNA virus. Its incidence has increased 30-fold with significant outbreaks occurring in five of six World Health Organization (WHO) regions. At present, dengue is endemic in 112 countries in the world. Sporodic outbreak used to occur in Pakistan but the current monsoon raining has caused flooding resulting in mosquito over breeding & epidemic of dengue fever. The worst affected areas are Sindh Punjab but a few cases has been there in Khyber Pakhtoon Khawa (KPK). We analysed the data of 12 cases of dengue fever at the department of Medicine Hayatabad Medical Complex, Peshawar.

Materials and Methods: It was retrospective analysis of the cases having conformed dengue fever.

Results: Total number of cases were 12, 11 were male & 1 was female. Age range was 14-55 years & mode was 35. 10 cases visited Punjab Province in a week prior to their illness. One was brought from Afghanistan & one from Khyber Pakhthoon Khawa. All had high grade fever, myalgia, headaches but skin rash was absent & there was no bleeding. All were hemodynemically stable i.e. normal pulse, BP & normal hematocrit & non was having an evidence of plasma leak syndrome. All had thrombocytopenia (platelets count less than 150,000), normal bilirubin, elevated alanine amino transferases (ALT more than 2 times above normal) & 10 cases had prolonged prothrombin time (PT > 3 seconds) while 2 had normal PT. Total leuckocyte count was low in 8 cases & normal in 4.

Conclusion and Recommendations: Dengue fever is not very common in KPK & most of the diagnosed cases had acquired the disease in Punjab. Nevertheless we should be vigilant for an impending epidemic which happing just across the Indus River.

INTRODUCTION

During the 19th century, dengue was considered a sporadic disease, causing epidemics at long intervals. However, dramatic changes in this pattern have occurred and currently, dengue ranks as the most important mosquito borne viral disease in the world. In the past 50 years, its incidence has increased 30-fold with significant outbreaks occurring in five of six World Health Organization (WHO) regions. At present, dengue is endemic in 112 countries in the world[1]. Around 2.5 to 3 billion people, living mainly in urban areas of tropical and subtropical regions, are estimated to be at risk of acquiring dengue viral infections[1]. Estimates suggest those annually 100 million cases of dengue fever and half a million cases of dengue haemorrhagic fever (DHF) occur in the world with a case fatality in Asian countries of 0.5%–3.5%[1]. Of those with DHF, 90% are children less than 15 years of age[1].

Although sporadic dengue fever was known for more than 200 years, reasons for the global resurgence of epidemics of dengue fever and DHF are not very clear[2]. Uncontrolled population growth, unplanned and uncontrolled urbanization;, inadequate wastewater management, and lack of effective mosquito control have been implicated in the increased distribution and density of the vector and also the increased spread of the virus. However, microevolution of the dengue virus may have also contributed to the spread of more virulent strains around the world. In fact there is evidence that the more virulent genotypes of the virus are replacing the less virulent genotypes, which may explain the global emergence of dengue infections[2].

Following torrential monsoon rainfall there was flood across the country. It has lead to stagnant water across the country resulting in mosquito over breeding. Currently there is an epidemic of dengue fever in Punjab & Sindh. Khyber Pakhthoon Khawa (KPK) is not much affected by the disease yet but cross country travel has caused a few cases reported in this region. We collected the data of 12 cases of confirmed dengue fever diagnosed at the department of medicine, Hyat abad medical complex Peshawar, in the current epidemic in Pakistan during November 2010.

MATERIAL AND METHODS

Suspected cases of dengue were screened for dengue specific IgM. Retrospective analysis was performed of their case notes who had confirmed dengue.

RESULTS

Out of 12 cases, 11 were male & 1 was a female. Age range was 14-55 years & mode was 35. 10 cases visited Punjab province in a week prior to their illness. One was brought from Afghanistan & one from Khyber Pakhthoon Khawa. All had high grade fever, myalgia, headaches but skin rash was absent & there was no bleeding. All were hemodynemically stable i.e. normal

pulse, BP & normal hematocrit & non was having an evidence of plasma leak syndrome. All had thrombocytopenia (platelets count less than 150,000), normal bilirubin, elevated alanine amino transferases (ALT more than 2 times above normal) & 10 cases had prolonged prothrombin time (PT > 3 seconds) while 2 had normal PT. Total leuckocyte count was low in 8 cases & normal in 4. Only one case had acute renal failure & was dialysis dependent with complete recovery.

DISCUSSION

The first epidemic of dengue hemorrhagic fever (DHF) in South East Asia occurred in 1954 in Manila, Philippines. Following this, epidemics have occurred in nearly all countries in this region, and currently are a major public health problem in seven of them. The incidence of DHF has increased dramatically in recent years with approximately five times more cases reported since 1980 than in the previous 30 years[3]. Although serological surveys conducted in Indonesia showed that DEN-1 and DEN-2 were the prevalent serotypes until the late 1980s, the DEN-3 serotype has been the predominant serotype in the recent outbreaks[4]. In fact, DEN-3 has been associated with severe dengue epidemics and it has been suggested that the DEN-3 virus may have certain characteristics that make it more virulent. Although DEN–4 has been isolated in almost all epidemics, it is primarily detected in secondary dengue infections[4]. Unfortunately due to non availability of the diagnostic facilities we do not the serotype of the current serotype of the virus responsible for the current epidemic.

Although small outbreaks of DHF occurred South Asia between 1964 and 1966, the first major epidemic of DHF occurred in Sri Lanka in 1989[5,6]. Since then regular epidemics have occurred in Sri Lanka, resulting in increasing numbers of cases each year. The DEN-3 subtype III was identified as the cause of the first and subsequent epidemics in Sri Lanka along with the DEN-2 serotype[7]. Dengue infections were first reported in India in 1991 (6291 cases of dengue fever), and the first epidemic of DHF occurred in Delhi in 1996[8]. The epidemiological pattern of DHF in South Asia is now similar to that in the South East Asian region. As yet, no cases of DHF have been reported from Nepal or Bhutan. In addition, the endemicity of dengue infections in these two countries is uncertain[9]. Sporadic outbreaks occurred on & off in Sindh & Punjab but not upto the scale of the current epidemic.

Dengue fever may occur either during primary or secondary infections. The onset is sudden with high fever, severe headache (especially in the retro-orbital area), arthralgia, myalgia, anorexia, abdominal discomfort, and sometimes a macular papular rash. The fever may be biphasic and tends to last for 2–7 days[10]. Flushing, a characteristic feature is commonly observed on the face, neck, and chest. Coryza may also be a prominent symptom especially in infants[10]. Younger childc tend to present with coryza, diarrhea, rash and zure, and less commonly with vomiting, headache, a abdominal pain[11]. Although, haemorrhagic manifes tions are uncommon in dengue fever, petechi pupura, gastrointestinal bleeding, epistaxis, and g gival bleeding have been observed in some indivi als[12]. A positive tourniquet test has been reported many individuals with dengue fever possibly due reduced capillary fragility[13]. Recovery from deng fever is usually uneventful, but may be prolonged e pecially in adults[13]. Most of our cases were havi dengue fever & non had DHF or plasma leaking sy drome. Most of the index cases had history of a trip Punjab except the 2, 1 from Afghanistan & one fro district Bannu of KPK.

The febrile phase begins with sudden onset f ver accompanied by generalized constitutional sym toms and facial flush. The fever is high grade, interm tent, and associated with rigors. Epigastric discomfor myalgia, vomiting, and abdominal pain are commo and patients are usually quite miserable. Sore throat and febrile convulsions may be seen, especially amon young children. Tender hepatomegaly is observed i almost all patients and splenomegaly may be seen i some. A macular papular rash similar to that seen i dengue fever is also seen in many patients[14]. The fe ver lasts for 2–7 days and is followed by a fall in tem perature to normal or subnormal levels. At this point the patient may recover or progress to the phase c plasma leakage. Those who remain ill despite the temperature subsiding are more likely to progress t DHF. Clinical deterioration usually occurs during de fervescence (often between days 3 and 4). Tachycar dia and hypotension characterize the onset of plasm leakage. When plasma leakage is severe patients may develop other signs of circulatory disturbance such as prolonged capillary refill time, narrow pulse pres sures, and shock. Inadequate treatment of such pa tients often leads to profound shock. During the phase of plasma leakage (first 24–48 hours after onset o DHF), pleural effusions and ascites are common. Pleural effusions are usually seen on the right side; a right decubitus chest radiograph is best for detecting small effusions[15]. Abdominal ultrasound scans may demonstrate ascites or a thickened gall bladder wall. Pericardial effusions may also occur. This latter complication is uncommon, but is associated with high morbidity and mortality. In DHF, bleeding may occur from any site and does not correlate with the platelet counts. Haemorrhagic manifestations usually occur once the fever has settled. Minor degrees of bleeding may manifest as gum bleeding and petechiae. The commonest site of haemorrhage is the gastrointestinal tract, which manifests as haematemesis or melaena, followed by epistaxis[15]. Vaginal bleeding is commonly reported in females[15]. None of our patient had bleeding though 8 cases had prolonged PT; plasma leaking syndrome but all had thrombocytopenia & elevated ALT.

Table 1: Characteristics of 12 cases of dengue fever at Hyatabad Medical Complex Peshawar

No.	Age	gender	Fever	headache	Myalgia	Rash	Bleeding	Pulse	BP	Petichie	ALT	Bilib	PT	Hematocrit	Platelets	WC
1	14	M	P	P	P	A	A	N	N	A	R	N	pr	N	Lt	L
2	34	M	P	P	P	A	A	N	N	P	R	N	pr	N	Lt	L
3	35	M	P	P	P	A	A	N	N	P	R	N	pr	N	Lt	L
4	35	M	P	P	P	A	A	N	N	A	R	N	pr	N	Lt	L
5	40	M	P	P	P	A	A	N	N	A	R	N	pr	N	Lt	L
6	44	M	P	P	P	A	A	N	N	A	R	N	pr	N	Lt	L
7	35	M	P	P	P	A	A	N	N	A	R	N	pr	N	Lt	L
8	24	M	P	P	P	A	A	N	N	A	R	N	pr	N	Lt	L
9	34	M	P	P	P	A	A	N	N	A	R	N	pr	N	Lt	N
10	50	M	P	P	P	A	A	N	N	A	R	N	pr	N	Lt	N
11	55	M	P	P	P	A	A	N	N	A	R	N	N	N	Lt	N
12	25	F	P	P	P	A	A	N	N	A	R	N	N	N	Lt	N

M= male, F=Female, P=Present, A=Absent, N=Normal, R=Raised, pr=prolonged by >3seconds, L=Less than 4000, Lt=Less than 150,000

In most cases of dengue fever, platelet counts and serum biochemistry are normal. However, leucopenia, thrombocytopenia and raised liver enzymes may be seen. In contrast, DHF is always accompanied by a platelet count <100,000, haemoconcentration (a rise in the packed cell volume. 20% of basal levels), leucopenia, and raised liver enzymes. Elevation of both alanine and aspartate aminotransferase levels occur with plasma aspartate aminotransferase levels being higher in children who develop DHF than in those with dengue fever[17]. A leucopenia has been suggested to predict the onset of DHF[18]. Initial leucopenia is followed by a relative lymphocytosis (with more than 15% atypical lymphocytes) towards the end of the febrile phase. Abnormal coagulation profiles (prolonged partial thromboplastin time and prothrombin time, raised fibrinogen degradation products), hypoalbuminaemia, and reduced serum complement levels are also seen. These coagulation abnormalities suggest that there is activation of both coagulation and fibrinolysis during acute infection and the degree of activation being greater in severe DHF and dengue shock syndrome[19]. During prolonged shock, metabolic acidosis, hyponatraemia, and increased blood urea are frequently seen. Plasma lipid concentrations (cholesterol, high and low density lipoprotein) are reduced in patients with more severe forms of DHF; the levels are significantly lower in patients with grade III or IV DHF compared with mild DHF or healthy controls[18].

CONCLUSION AND RECOMMENDATIONS

Dengue fever is an epidemic in KPK & most of cases had acquired the disease in Punjab. Most of the cases in Hyatabad Medical Complex had classical dengue fever but nevertheless we should be vigilant as the epidemic is happening just across of Indus River.

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