JUL/AUG 2007

TULIP NEWS

Tulip Group gets busy again to combat another spell of monsoon related diseases...

After the scorching spell of summer, comes the cool monsoon with a long awaited relief. However the cool showers also bring with them a lot of diseases which are peculiar to monsoon. In India, monsoon begins in the first week of June. It can be late or sometimes be earlier. The arrival, period and density of rainfall varies location wise too.

Diseases that spread during monsoon are mainly water-borne, air-borne or vector-borne. Some of the water-borne gastro-intestinal diseases like typhoid, paratyphoid, mosquito-borne disease like malaria, dengue fever and other diseases like Weil’s disease, leptospirosis take a toll on the population during this monsoon.

Infact last year, there was an upsurge of dengue fever. Collection of water in potholes and stagnation provide breeding grounds for mosquitoes and bring about the spread of diseases like malaria and dengue. Contamination of water due to low levels and unhygienic living conditions in the cities are mainly the causes of many monsoon ailments. Typhoid, paratyphoid fever is spread through contaminated water and food. Water gets contaminated with rat or cat urine containing microorganisms, and persons coming in contact with such water becomes victims of diseases like leptospirosis and rat fever. Climatic variations also aggravate the spread of the above diseases.

Like an eagle quick to react, Tulip Group with its array of rapid diagnostic tests is ready to combat the spread of such diseases.

<table>
<thead>
<tr>
<th>Product</th>
<th>Description</th>
<th>Pack</th>
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<tbody>
<tr>
<td>Enterocheck-WB</td>
<td>Rapid immunochromatographic tests for 10 Tests</td>
<td>(Device)</td>
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<td></td>
<td>detection of IgM antibodies to S.typhi in serum/plasma and whole blood</td>
<td>10 Tests</td>
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<td>Dengucheck-WB</td>
<td>Rapid immunochromatographic tests for 10 Tests</td>
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<td>detection of Dengue virus in human Serum, plasma and white blood</td>
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<tr>
<td>Leptocheck-WB</td>
<td>Rapid immunochromatographic tests for 10 Tests</td>
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INFECTIOUS DISEASE RANGE

PARASITOLOGY RANGE

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<tr>
<td>Falcivax</td>
<td>Rapid tests for Malaria Pv/Pf</td>
<td>1/10/25 Tests</td>
</tr>
<tr>
<td>Parascreen</td>
<td>Rapid tests for Malaria Pan/Pf</td>
<td>1/10/25 Tests</td>
</tr>
<tr>
<td>Parabank</td>
<td>Rapid tests for Malaria Pan</td>
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For Details contact:

TULIP XL-FDP

ERLYCONE

ANTU-HUMAN GLOBULIN

TULIP DIAGNOSTICS (P) LTD.

CALCIUM KIT (OCPC METHOD)

TULIP GROUP IS LEADING THE WAY IN DIAGNOSTICS

Dear Customers,

We wholeheartedly owe this great success to you. This would not have been possible but for your tremendous support and patronage during the last 19 years of our service to you. We assure you that Tulip Group of Companies will continue rendering products and services to the best of your expectations for years to come.
DIC is an acquired coagulation disorder characterized by widespread microvascular thrombosis, resulting in coagulopathy and hemorrhage. The pathogenesis of DIC is complex and involves multiple mechanisms that lead to the consumption of coagulation factors and platelets. DIC may occur as a result of various conditions, including severe infections, sepsis, obstetric complications, and trauma.

**Cardinal features**

- **Severe infection and sepsis**, which is the most common cause of DIC, accounting for 95% of cases. About 10-20% of patients with Gram-negative bacteria have evidence of DIC, but DIC is also seen in patients with Gram-positive infections, viral infections, malaria, viral hepatitis, trauma, burns, shock, or severe trauma.

- **Obstetric** DIC is seen in severe pregnancy complications, such as abruptio placentae, placental abruption, and fetal death. DIC may also occur in association with placental separation or placental infarction.

- **Malignancies** can cause DIC by invasion of tissues and release of tissue factor, or by direct tumor-induced activation of the coagulation cascade.

**Pathogenesis**

DIC is characterized by a hypercoagulable state that leads to the formation of microvascular thrombi, resulting in the consumption of coagulation factors and platelets. This process can occur rapidly, leading to a systemic hypocoagulable state. DIC is often associated with other clinical conditions, such as sepsis, obstetric complications, or malignancies.

**Clinical presentation**

- **Severe bleeding** can occur, with bleeding from multiple sites, including the gastrointestinal tract, skin, and mucous membranes.

- **Mortality** is high, with severe DIC having a mortality rate exceeding 75%, usually owing to multiorgan failure.

**Diagnosis**

- **Clinical diagnosis** is based on the presence of clinical features, such as hemorrhage, shock, and multiorgan failure.

- **Laboratory tests** should be performed to confirm the diagnosis, including prothrombin time, partial thromboplastin time, fibrinogen level, and D-dimer measurements.

**Treatment**

- **Therapeutic approach** involves the correction of the underlying disorder and supportive care, including fluid resuscitation, transfusion of platelets and plasma, and the use of anticoagulants.

**Follow-up**

- **Regular monitoring** of laboratory tests and clinical status is essential to assess the response to treatment and to detect complications.

**Conclusion**

DIC is a life-threatening multisystem disorder that requires prompt recognition and treatment. Early identification of the underlying cause is crucial for successful management. The prognosis of DIC is highly variable and depends on the underlying condition and the severity of the clinical presentation.

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Platelets Thrombocytopenia

Causes of abnormal result:
- Thrombocytopenia
- Leukemia
- Viral infections
- Toxins
- Sequestration or hypersplenism.

Thrombocytopenia is caused by:
- Platelet production
- E.g. idiopathic thrombocytopenic purpura, drug-induced thrombocytopenia.

There is no single diagnostic test for DIC. Diagnosis is strongly suggested by a combination of:
- Clinical and laboratory findings
- Medical history
- Rule out causes of DIC

Clinical findings:
- Symptoms of thrombosis or hemorrhage
- Evidence of ischemia
- Metabolic and respiratory acidosis
- Disseminated intravascular coagulation
- Septic shock

Laboratory findings:
- Coagulation studies
- Platelet count
- Fibrinogen level
- PT, aPTT
- haptoglobin

The 50% of serum calcium that is protein-bound.

Possible secondary hyperparathyroidism. Initially lowered serum calcium results
in bone absorption. Therefore the lower calcium level simulates the parathyroid
release of parathyroid hormone, which activates calcium reabsorption.

Abnormal calcium and abnormal phosphorus indicate impaired calcium absorption due
- Malabsorption
- Cystic fibrosis
- Due to renal failure, laxatives, cytotoxic drugs

- Spurious macrocytosis on automated machines?
- Respiratory alkalosis
- Metabolic alkalosis
- Sarcoidosis due to increased IgG or IgA
- Normal kidney function
- Rickets
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GUIDELINES FOR BLOOD GROUPING AND ANTIBODY TESTING IN PREGNANCY (Continued)

Recommendation 10: All women who have previously had an infant affected by HDN should be referred before 28 weeks to a specialist unit for advice and for assessment of feto-maternal haemoglobinopathy, irrespective of antibody level. (Level III, Grade C)

PATERNSAL TESTING

Where there is a clinically significant antibody capable of causing HDN, particularly anti-D, -c, or anti-K, is present in a maternal sample, determining the corresponding antigen is important. This process is also referred to as the paternally-directed testing. Careful attention to technique is necessary to minimise the variables in the method and titrating the anti-D standard in parallel, the corresponding antigen. Careful attention to technique is necessary to minimise the variables in the method and titrating the anti-D standard in parallel.

The presence of any further antibodies should be established and any clinically significant antibodies should be assessed and the antibody titre assessed.

A PATERNAL TESTING

A clinically significant antibody of high concentration is present, and the father has a history of HDN and the father is heterozygous for the relevant antigen, it can be clinically relevant to determine the genotype of the fetus. Until recently, fetal DNA typing by PCR remains the gold standard for this test. These invasive techniques carry a small risk of miscarriage, chromosomal abnormalities, or stillbirth.

The genotype is now available for the accurate determination of fetal RhD genotype from samples of maternal peripheral blood. The same testing service for fetal c and K types is under development and should be available for these specificities. REPORTS OF LABORATORY INVESTIGATIONS

In addition to blood group and specificity of any red cell antibodies present, reports must include the clinical significance for the woman's antenatal care of the likely significance of the antibody, with respect to both the development of HDN and transfusion problems. Reports should also, wherever relevant, alert the clinician to the need to refer the woman to a specialist unit.

Details of the timing of further samples required should also be given.

Recommendation 11: Women with clinical significant red cell antibodies should be issued with a card giving details of the antibody.

Action at the time of birth

A negative maternal DAT would indicate that the baby is D positive, and the baby may type as antigen negative for fetal D antigen sites. Consequently, up to 3-6% of D positive cord samples may be expected to give a positive DAT result of more than 4mL by acid elution technique, should be referred for a feto-maternal haemoglobinopathy (FMH) result of more than 4mL by acid elution technique, should be referred for a feto-maternal haemoglobinopathy (FMH) result of more than 4mL by acid elution technique.

The FMH result of more than 4mL by acid elution technique, should be referred for a feto-maternal haemoglobinopathy (FMH) result of more than 4mL by acid elution technique should be used to calculate for prophylactic anti-D.

If the FMH result of more than 4mL by acid elution technique, should be referred for a feto-maternalhaemoglobinopathy (FMH) result of more than 4mL by acid elution technique, the volume of fetal cells present, so that additional anti-D immunoglobulin may be given. The volume of fetal cells present is calculated by multiplying the volume of blood collected at birth by the percentage of fetal red cells.

A test should be performed on the maternal blood sample to determine and estimate the volume of fetal cells. If the test is not performed, and the test is not performed, the volume of fetal cells present should be taken as the volume of blood collected at birth.

If the test is not performed, the volume of fetal cells present should be taken as the volume of blood collected at birth. This volume should be used to provide information to determine management of the pregnancy. A medical decision should be made regarding the frequent testing of women with a history of HDN with antibody screening of cord blood and antibody screening of cord blood during pregnancy.

Where an antibody has been detected, testing of both booking and 28-week samples should be performed. Other clinically significant antibodies other than anti-D, -c, or -K, should be assessed, and other antibodies excluded at 'first appointment' and at 28 weeks gestation. (Level III, Grade B)

Recommendation 12: All infants born to women who have clinically significant antibodies should be closely observed for evidence of HDN. A DAT should be performed and if positive, haemoglobin and bilirubin levels should be measured. (Level IV, Grade C)

Pro and Post Delivery Testing of maternal samples

Routine antibody screening of immediate pre and post-delivery samples is not required, as the information does not influence the management of the pregnant woman or her infant. Blood grouping and antibody screening of maternal samples other than confirmatory testing should be undertaken only if pre-transfusion compatibility tests are required.