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- Dengue Hemorrhagic Fever
- Motor Neuron Disease
- Foodborne Disease
- Allergic Rhinitis in Children

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Editorial

Dear Doctor,

Welcome to this edition of "the *SQUARE*"!

We hope all of you are in good health!

In this issue we have published a special feature on “Dengue Hemorrhagic Fever (DHF)”, a potentially deadly complication with symptoms similar to those of dengue fever. In addition, we have a comprehensive article on “Motor Neuron Disease” a devastating, incurable neurodegenerative disease of the motor neurons that primarily affects people in their 60s or 70s.

We also bring you all the details on “Foodborne Disease”, the infections or irritations of the gastrointestinal (GI) tract caused by food or beverages that contain harmful bacteria, parasites, viruses, or chemicals. Moreover, we have focused on “Allergic Rhinitis in Children” an important and common condition that causes major morbidity in children and is a risk factor for the development of asthma.

Every effort has been made to make this issue interesting and informative. We believe that you will enjoy this issue as well.

On behalf of the management of *SQUARE*, we wish you all a very blissful, healthy and successful life!

Thank You!

Omar Akramur Rab

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Dengue Hemorrhagic Fever

Dengue is a mosquito-borne disease caused by any one of four closely related dengue viruses (DENV-1, -2, -3 and -4). Infection with one serotype of DENV provides immunity to that serotype for life, but provides no long-term immunity to other serotypes. Thus, a person can be infected as many as four times, once with each serotype. Dengue viruses are transmitted from person to person by Aedes mosquitoes (most often *Aedes aegypti*) in the domestic environment. The incidence of dengue has increased dramatically in recent decades, with estimates of 40%-50% of the world's population at risk for the disease in tropical, subtropical and most recently, more temperate areas.

Classic dengue fever or "break bone fever," is characterized by acute onset of high fever 3-14 days after the bite of an infected mosquito. Symptoms include frontal headache, retro-orbital pain, myalgias, arthralgias, hemorrhagic manifestations, rash, and low white blood cell count. The patient also may complain of anorexia and nausea. Acute symptoms, when present, usually last about 1 week, but weakness, malaise and anorexia may persist for several weeks. A high proportion of dengue infections produce no symptoms or minimal symptoms, especially in children and those with no previous history of having a dengue infection.

Some patients with dengue fever go on to develop dengue hemorrhagic fever (DHF), a severe and sometimes fatal form of the disease. Around the time the fever begins to subside (usually 3-7 days after symptom onset), the patient may develop warning signs of severe disease. Warning signs include severe abdominal pain, persistent vomiting, marked change in temperature (from fever to hypothermia), hemorrhagic manifestations or change in mental status (irritability, confusion or obtundation).

The patient also may have early signs of shock, including restlessness, cold clammy skin, rapid weak pulse, and narrowing of the pulse pressure. Patients with dengue fever should be told to return to the hospital if they develop any of these signs.

DHF is currently defined by the following four World Health Organization (WHO) criteria:

- Fever or recent history of fever lasting 2-7 days.
- Any hemorrhagic manifestation.
- Thrombocytopenia (platelet count of <100,000/ mm^3).
- Evidence of increased vascular permeability.

The most common hemorrhagic manifestations are mild and include a positive tourniquet test, skin hemorrhages (petechiae, hematomas), epistaxis, gingival bleeding and microscopic hematuria. More serious types of hemorrhage include vaginal bleeding, hematemesis, melena and intracranial bleeding.

Evidence of plasma leakage due to increased vascular permeability consists of at least one of the following:

- An elevated hematocrit ≥20% above the population mean hematocrit for age and sex.
- A decline in hematocrit after volume-replacement treatment of ≥20% of the baseline hematocrit.
- Presence of pleural effusion or ascites detected by radiography or other imaging method.
- Hypoproteinemia or hypoalbuminemia as determined by laboratory test.

WHO is currently reevaluating the clinical case definition for dengue fever and DHF. Studies from different countries have reported life-threatening complications from dengue in the absence of one or more of the current criteria for DHF. Despite the name, the critical feature that distinguishes DHF from dengue fever is not hemorrhaging, but rather plasma leakage resulting from increased vascular permeability.

Epidemiology

Today, DHF is regarded internationally as the most significant mosquito-borne viral disease. It is endemic to more than 100 countries worldwide, especially tropical and sub-tropical regions. In the United States, the DHF causing Aedes aegypti species can be found seasonally in Louisiana, southern Florida, New Mexico, Arizona, Texas, Georgia, Alabama, Mississippi, North and South Carolina, Kentucky, Oklahoma and Tennessee. In the last 50 years, the incidence of dengue has increased 30-fold.

According to the World Health Organization (WHO), it is estimated that 50 to 100 million infections of

dengue fever occur yearly. Of these cases 500,000 progress to DHF resulting in 22,000 deaths, mostly of children. Based on official data submitted to the WHO, cases of dengue across the Americas, South-East Asia and Western Pacific surpassed 1.2 million in 2008 and over 3 million in 2013. In 2013, 2.35 million cases of dengue were reported in the Americas alone, of which 37, 687 cases were of DHF. After the first known epidemic of DHF in 1953 to 1954 in the Philippines, the disease continued to spread throughout Southeast Asia. During the 1950s, 1960s and most of the 1970s, dengue epidemic was rare in most of South and Central America because the primary mosquito vector, *Aedes aegypti*, was eradicated. However, once the extermination was stopped, *Aedes aegypti* began to reinvade those regions. In 1997, *Aedes aegypti* and the dengue virus gained a worldwide distribution again.

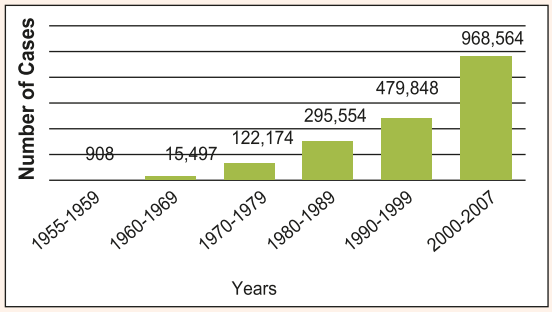

Figure 1: Average number of DF/DHF reported to WHO in the years provided.

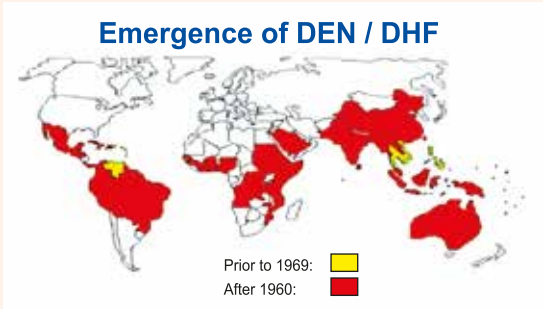

Figure 2: Distribution of dengue/dengue hemorrhagic fever in the Americas, Caribbean, Africa and the Middle East Source: http://www.who.int/csr/disease/dengue/dehngueemergence.jpg

According to WHO, there are now over 2.5 billion people who live in areas where dengue/DHF is endemic and are at risk for being infected with the dengue virus. Several factors contribute to the viral transmission by *Aedes aegypti*; including temperature, rainfall, rural-urban migration, population growth, stored water and increased solid waste, which provides larval habitats for the vector. There can also be outbreaks of travel associated DHF. Dengue fever is the most widespread arbovirus infection worldwide.

Pathogenesis

Two main pathophysiological changes occur in DHF/Dengue Shock Syndrome (DSS). One is an increased vascular permeability that gives rise to loss of plasma from the vascular compartment. This results in hemoconcentration, low pulse pressure and other signs of shock, if plasma loss becomes critical. The second change is a disorder in hemostasis involving vascular changes, thrombocytopenia and coagulopathy. A constant finding in DHF/DSS is activation of the complement system, with profound depression of C3 and C5 levels. The mediators that increase vascular permeability and the precise mechanism(s) of the bleeding phenomena seen in dengue infections have not yet been identified; consequently, further studies are needed. Immune complexes have been described in DHF but their role is not yet clear. Platelet defects may be both qualitative and quantitative, i.e. some circulating platelets during the acute phase of DHF may be exhausted (incapable of normal function). Therefore, even a patient with a platelet count greater than 100000 per mm3 may still have a prolonged bleeding time. A mechanism that may contribute to the development of DHF/DSS is enhancement of virus replication in macrophages by heterotypic antibodies.

In secondary infections with a virus of a different serotype from that causing the primary infection, cross-reactive antibodies that fail to neutralize virus may increase the number of infected monocytes as dengue virus-antibody complexes are taken into these cells. This in turn may result in the activation of cross-reactive CD41 and CD81 cytotoxic lymphocytes. The rapid release of cytokines caused by the activation of T cells and by the lysis of infected monocytes mediated by cytotoxic lymphocytes may result in the plasma leakage and hemorrhage that occur in DHF.

Etiology

Dengue infection is caused by dengue virus (DENV), which is a single-stranded RNA virus with an icosahedral nucleocapsid and covered by a lipid envelope. The virus is in the family Flaviviridae, genus Flavivirus, and the type-specific virus is yellow fever. The dengue virus has 4 related but antigenically distinct serotypes: DENV-1, DENV-2, DENV-3, and DENV-4. Genetic studies of sylvatic strains suggest that the 4 serotypes evolved from a common ancestor in primate populations approximately 1000 years ago and that all 4 separately emerged into a human urban transmission cycle 500 years ago in either Asia or Africa. Albert Sabin speciated these viruses in 1944. Each serotype is known to have several different genotypes. Viral genotype and serotype and the sequence of infection with different serotypes, appear to affect disease severity. Living in endemic areas of the tropics where the vector mosquito thrives is an important risk factor for infection. Poorly planned urbanization combined with explosive global population growth brings the mosquito and the human host into close proximity. Increased air travel easily transports infectious diseases between populations.

Characteristics of dengue hemorrhagic fever outbreaks

Although the early outbreaks of DHF seem to have appeared suddenly in the Philippines and in Thailand, retrospective studies indicate that they were probably preceded by a decade or so in which cases occurred but were not recognized. In Thailand, outbreaks first occurred in Bangkok in a pattern with a 2-year cycle, then subsequently in irregular cycles as the disease spread throughout the country. DHF then became endemic in many large cities of Thailand, eventually spreading to smaller towns and villages during periods of epidemic transmission. A similar pattern was observed in Indonesia, Myanmar and Vietnam.

During the 40 years' experience with dengue in the Western Pacific and South-East Asia Regions, two important epidemiological patterns have been recognized. First, DHF /DSS has appeared most frequently in areas where multiple dengue serotypes are endemic. The usual pattern is that of sporadic cases or small outbreaks in urban areas that steadily increase in size until there is an explosive outbreak that brings the disease to the attention of public health authorities. The disease then usually establishes a pattern of epidemic activity every 2-5 years. In addition, DHF/DSS is typically confined to children, with a modal age at hospitalization of 4-6 years. A second pattern is observed in areas of low endemicity. Multiple dengue serotypes may be transmitted at relatively low rates of infection (below 5% of the population per year). In these areas, previously uninfected adults are susceptible to dengue infection and children and young adults, with a modal age of 6-8 years, are also vulnerable.

A cyclical pattern of increased transmission coinciding with the rainy season has been observed in some countries. The interactions between temperature and rainfall are important determinants of dengue transmission, as cooler temperatures affect adult mosquito survival, thus influencing transmission rates. Furthermore, rainfall and temperature may affect patterns of mosquito feeding and reproduction and hence the population density of vector mosquitoes.

Although DHF may affect persons of all ages in dengue endemic areas, most DHF cases occur in children less than 15 years of age. Since 1964, the trend in Bangkok has been towards progressively lower attack rates (constant hospital admission rates despite an increasing population), with the modal age of hospitalized children being 6-7 years throughout Thailand. Surveillance data from some areas have suggested a slight excess of infected girls over boys, while other areas have shown an almost even distribution.

A retrospective evaluation of the impact of DHF during an outbreak in Bangkok/Thon Buri in May-November 1962 indicated that in a population of 870000 children under 15 years of age, an estimated 150000-200000 minor febrile illnesses were caused by dengue and occasionally by chikungunya viruses; 4187 patients were hospitalized with DHF and 4000 additional patients were treated in private clinics or at home. Moreover, shock occurred in about one-third of the hospitalized DHF patients.

In the more recent large epidemic in Thailand in 1987, the attack rate of DHF/DSS was 320 cases per 100000 population for all ages. In southern Vietnam between 1975 and 1992, the attack rate of DHF/DSS ranged from 30 to 380 per 100000 population, with mortality rates from 0.39 to 6.42 per 100000 population, while the incidence of DHF in Indonesia for 1991 and 1992 was 11.56 and 9.45 per 100000, respectively.

Clinical diagnosis

Typical cases of DHF are characterized by four major clinical manifestations: high fever, haemorrhagic phenomena and often, hepatomegaly and circulatory failure. Moderate to marked thrombocytopenia with concurrent haemoconcentration is a distinctive clinical laboratory finding of DHF. The major pathophysiological change that determines the severity of disease in DHF-and differentiates it from DF-is the leakage of plasma, as manifested by an elevated haematocrit1 (i.e. haemoconcentration), a serous effusion or hypoproteinaemia.

Children with DHF commonly present with a sudden rise in temperature accompanied by facial flush and other non-specific constitutional symptoms resembling DF, such as anorexia, vomiting, headache and muscle or bone and joint pain. Some patients complain of sore throat and an infected pharynx is frequently evident on examination, but rhinitis and cough are infrequent. Mild conjunctival infection may be observed (see Table). Epigastric discomfort, tenderness at the right costal margin and generalized abdominal pain are common. The temperature is usually high (.39°C) and remains so for 2-7 days. Occasionally, temperature may be as high as 40-41°C; febrile convulsions may occur, particularly in infants.

The most common haemorrhagic phenomenon is a positive tourniquet test, easy bruising and bleeding at venepuncture sites. Present in most cases are discrete fine petechiae scattered on the extremities, axillae, face and soft palate, which are usually seen during the early febrile phase. Epistaxis and gingival bleeding occur infrequently; mild gastrointestinal haemorrhage may be observed during the febrile period.

Table : Non-specific constitutional symptoms observed in haemorrhagic fever patients with dengue and chikungunya virus infection[a]

Criteria	DHF(%)	Chikungunya Fever (%)
Injected pharynx	96.8	90.3
Vomiting	57.9	59.4
Constipation	53.5	40.0
Abdominal pain	50.0	31.6
Headache	44.6	68.4
Generalized lymphadenopathy	40.5	30.8
Conjunctival injection	32.8[b]	55.6[b]
Cough	21.5	23.3
Rhinitis	12.8	6.5
Maculopapular rash	12.1[b]	59.4[b]
Myalgia/arthralgia	12.0[b]	40.0[b]
Enanthema	8.3	11.1
Abnormal reflex	6.7	0.0
Diarrhoea	6.4	15.6
Palpable spleen	6.3[c]	3.1[c]
Coma	3.0	0.0

a Modified from Nimmannitya Set al. American Journal of tropical Medicine and hygiene, 1969, 18: 954-971.
b Statistically significant difference.
c Infants under 6 months.

The liver is usually palpable early in the febrile phase and varies in size from just palpable to 2-4cm below the costal margin. Although liver size is not correlated with disease severity, an enlarged liver is observed more frequently in shock than in non-shock cases. The liver is tender, but jaundice is not usually observed. Splenomegaly is rarely observed in infants; however, the spleen may be prominent on X-ray examination.

The critical stage of the disease course is reached at the end of the febrile phase. After 2-7 days of fever, a rapid fall in temperature is often accompanied by signs of circulatory disturbance of varying severity. The patient may sweat, be restless, have cool extremities and show some changes in pulse rate and blood pressure. In less severe cases, these changes are minimal and transient, reflecting a mild degree of plasma leakage. Many patients recover spontaneously or after a short period of fluid and electrolyte therapy. In more severe cases, when plasma loss is critical, shock ensues and can progress rapidly to profound shock and death if not properly treated.

The severity of the disease can be modified by early diagnosis and replacement of plasma loss. Thrombocytopenia and haemoconcentration are usually detectable before the subsidence of fever and the onset of shock.

Laboratory diagnosis

Because the signs and symptoms of dengue fever are nonspecific, attempting laboratory confirmation of dengue infection is important. Laboratory criteria for diagnosis include one or more of the following:

- Demonstration of a fourfold or greater change in reciprocal immunoglobulin G (IgG) or immunoglobulin M (IgM) antibody titers to one or more dengue virus antigens in paired serum samples
- Demonstration of dengue virus antigen in autopsy tissue via immunohistochemistry or immunofluorescence or in serum samples via enzyme immunoassay (MAC-ELISA, IgG ELISA, NSI-ELISA, EIA)
- Detection of viral genomic sequences in autopsy tissue, serum or cerebral spinal fluid (CSF) samples via reversetranscriptase polymerase chain reaction (RT-PCR)
- Less commonly, isolation of dengue virus from serum, plasma, leukocytes, or autopsy samples

RT-PCR yields a serotype-specific diagnosis very rapidly. It is most useful early in the course of illness. It is not susceptible to the cross-reactivity with other flaviviruses seen with serologic testing. The following laboratory tests should also be performed:

- Complete blood cell (CBC) count
- Metabolic panel
- Serum protein and albumin levels
- Liver panel
- Coagulation profile and disseminated intravascular coagulation (DIC) panel

Grading severity of dengue haemorrhagic fever

DHF is classified into four grades of severity, where grades III and IV are considered to be DSS. The presence of thrombocytopenia with concurrent haemoconcentration differentiates grades I and II DHF from DF.

Grade I : Fever accompanied by non-specific constitutional symptoms; the only haemorrhagic manifestation is a positive tourniquet test and/or easy bruising.

Grade II: Spontaneous bleeding in addition to the manifestations of Grade I patients, usually in the forms of skin or other haemorrhages.

Grade III: Circulatory failure manifested by a rapid, weak pulse and narrowing of pulse pressure or hypotension, with the presence of cold, clammy skin and restlessness.

Grade IV: Profound shock with undetectable blood pressure or pulse.

Grading the severity of the disease at the time of discharge has been found clinically and epidemiologically useful in DHF epidemics in children in the WHO Regions of the Americas, South-East Asia and the Western Pacific and experience in Cuba, Puerto Rico and Venezuela suggests that grading is also useful for adult cases.

Treatment

Successful management of severe dengue requires careful attention to fluid management and proactive treatment of hemorrhage. Admission to an intensive care unit is indicated for patients with dengue shock syndrome.

Patients may need a central intravenous line for volume replacement and an arterial line for accurate blood pressure monitoring and frequent blood tests. Exercise caution when placing intravascular catheters because of the increased bleeding complications of dengue hemorrhagic fever. Urethral cathete-Intravascular volume deficits should be corrected with isotonic fluids such as Ringer lactate solution. Boluses of 10-20 mL/kg should be given over 20 minutes and may be repeated. If this fails to correct the deficit, the hematocrit value should be determined. If it is rising, limited clinical information suggests that a plasma expander may be administered. Starch, dextran 40, or albumin 5% at a dose of 10-20 mL/kg may be used. One study has suggested that starch may be preferable because of hypersensitivity reactions to dextran.

If the patient does not improve after infusion of a plasma expander, blood loss should be considered. Patients with internal or gastrointestinal bleeding may require transfusion and patients with coagulopathy may require fresh frozen plasma. After patients with dehydration are stabilized, they usually require intravenous fluids for no more than 24-48 hours. Intravenous fluids should be stopped when the hematocrit falls below 40% and adequate intravascular volume is present. At this time, patients reabsorb extravasated fluid and are at risk for volume overload if intravenous fluids are continued. Do not interpret a falling hematocrit value in a clinically improving patient as a sign of internal bleeding. Platelet and fresh frozen plasma transfusions may be required to control severe bleeding. A case report demonstrated good improvement following intravenous anti-D globulin administration in 2 patients. The authors proposed that, as in immune thrombocytopenic purpura from disorders other than dengue, intravenous anti-D produces Fcγ receptor blockade to raise platelet counts.

Patients who are resuscitated from shock rapidly recover. Patients with dengue hemorrhagic fever or dengue shock syndrome may be discharged from the hospital when they meet the following criteria:

- ❏ Afebrile for 24 hours without antipyretics
- ❏ Good appetite, clinically improved condition
- ❏ Adequate urine output
- ❏ Stable hematocrit level
- ❏ At least 48 hours since recovery from shock
- ❏ No respiratory distress
- ❏ Platelet count greater than 50,000 cells/μL

Vaccine development

One vaccine is currently approved for the prevention of dengue infection. Dengvaxia, a live recombinant tetravalent vaccine was registered in several countries in late 2015-2016, with Mexico being the initial country to register the vaccine in December 2015. The vaccine is given in 3 doses at age 0, 6, and 12 months. It underwent testing in more than 30,000 volunteers and was shown to reduce the risk of severe illness and hospitalization by as much as 30% in individuals previously infected with one or more strains. The vaccine proved less effective in persons who were not previously exposed to dengue and in areas with a lower burden of disease. Owing to concern that the vaccine may act like an initial dengue infection in this second group of individuals not previously infected with the virus, with additional exposure to a second serotype placing these individuals at increased risk of severe dengue, the WHO released a position paper in July 2016, stating that countries should consider introduction of vaccine as a part of an comprehensive dengue control strategy only where epidemiologic data indicate a high burden of disease.

Dengue vaccine development was prolonged because immunity to a single dengue strain is the major risk factor for severe dengue; as such, a vaccine must provide high levels of immunity to all 4 dengue strains to be clinically useful. Seroconversion alone does not predict protection. Several other immunogenic tetravalent vaccine candidates have been developed and are undergoing clinical trials.

Prognosis

Dengue fever is typically a self-limiting disease with a mortality rate of less than 1%. When treated, dengue hemorrhagic fever has a mortality rate of 2-5%. When left untreated, dengue hemorrhagic fever has a mortality rate as high as 50%. Survivors usually recover without sequelae and develop immunity to the infecting serotype.

Dengue Hemorrhagic Fever

The fatality rate associated with severe dengue varies by country, from 12-44%. In a 1997 Cuban epidemic, the fatality rate in patients who met criteria for severe dengue was approximately 6%. The mortality rate associated with dengue fever is less than 1%. Data from the 1997 Cuban epidemic suggest that, for every clinically apparent case of dengue fever, 13.9 cases of dengue infection went unrecognized because of absent or minimal symptoms. A 2005 review from Singapore of 14,209 patients found that useful predictors of death included the following :

- Atypical presentations
- Significant comorbid illness
- Abnormal serum markers (including albumin and coagulation studies)
- Secondary bacterial infections

Factors that affect disease severity include the following:

- Patient age
- Pregnancy
- Nutritional status
- Ethnicity
- Sequence of infection with different dengue serotypes
- Virus genotype
- Quality and extent of available medical care

Complications and sequelae of dengue virus infections are rare but may include the following:

- Cardiomyopathy
- Seizures, encephalopathy, and viral encephalitis
- Hepatic injury
- Depression
- Pneumonia
- Iritis
- Orchitis
- Oophoritis

In 20-30% of dengue hemorrhagic fever cases, the patient develops shock, known as the dengue shock syndrome. Worldwide, children younger than 15 years constitute 90% of dengue hemorrhagic fever patients ; however, in the Americas, dengue hemorrhagic fever occurs in both adults and children. Although dengue is an extremely important arboviral illness globally, literature evaluating the economic impact is fairly sparse, with some conflicting findings. A recent expert panel assessment and 2 studies in the Americas recommended additional research to fill important information gaps, including disease outcomes and accurate statistics regarding disease burden, that could better inform future decision making regarding control and prevention.

A 5-year prospective study in Thai children examined the relative economic burden of dengue infection in children on the local population. Most disability-adjusted life years (DALYs) lost to dengue resulted from long-term illness in children who had not been hospitalized. The infecting serotype appeared to be the major determinant of DALYs lost, with DEN-2 and DEN-3 responsible for 59%. The mean cost of illness from dengue was significantly higher than that from other febrile illnesses studied.

A prospective study examined the direct and indirect costs of dengue infection in 1695 pediatric and adult patients in 8 countries. The average illness lasted 11.9 days for ambulatory patients and 11 days for hospitalized patients. Hospitalized students lost 5.6 days of school. Those at work lost 9.9 work days. Overall mean costs were more than double (1394 international dollars [I$]) for hospitalized cases. With an annual average of 594,000 cases the aggregate economic cost was estimated to be at least I$587 million, without factoring in underreporting of disease and dengue surveillance and vector control costs. This represents a significant global economic burden in low-income countries.

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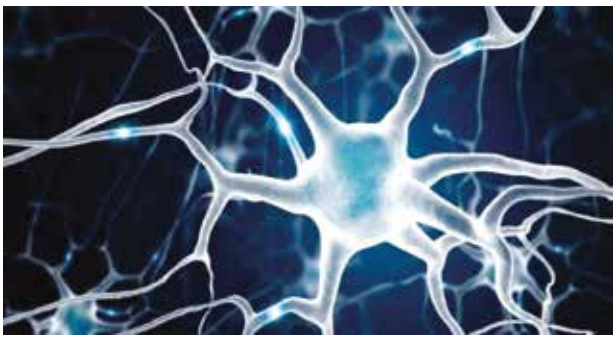
Motor Neuron Disease (MND) is a neurodegenerative disorder that selectively affects motor neurons, the cells that control voluntary muscles of the body. Motor neurons control important muscle activity, such as: gripping, walking, speaking, swallowing, breathing. MND is characterized by loss of upper motor neurons (UMNs, including the Betz cells of the motor cortex) and lower motor neurons (LMNs, anterior horn cells of the spinal cord and brainstem nuclei).



It is a progressive disease, which means symptoms get worse over time. It can affect how one walks, talks, eats, drinks and breathes. In some cases, it can also affect how one thinks and behaves.

A French doctor called Jean-Martin Charcot first described MND in 1874. The renowned English physicist Stephen Hawking lived with ALS (Amyotrophic Lateral Sclerosis, the most common type) for many decades until his death in March 2018. Guitar virtuoso Jason Becker is another example of someone who has been living with ALS for several years.

Stephen Hawking

Types

There are several types of motor neuron disease.

Amyotrophic Lateral Sclerosis (ALS) or Lou Gehrig's disease: It is the most common type, affecting muscles of the arms, legs, mouth and respiratory system. Mean survival time is 3 to 5 years, but some people live 10 years or more beyond diagnosis with supportive care.

Progressive Bulbar Palsy (PBP): It involves the brain stem. People with ALS often have PBP too. The condition causes frequent choking spells, difficulty in speaking, eating, and swallowing.

Progressive Muscular Atrophy (PMA): Slowly but progressively, it causes muscle wasting (especially in the arms, legs, and mouth). It may be a variation of ALS.

Primary Lateral Sclerosis (PLS): It is a rare form of MND that advances more slowly than ALS. It is not fatal, but it can affect the quality of life. In children, it is known as juvenile primary lateral sclerosis.

Spinal Muscular Atrophy (SMA): It is an inherited MND that affects children. There are three types, all caused by an abnormal gene known as SMA1. It tends to affect the trunk, legs and arms. Long-term outlook varies according to type.

The different types of MND share similar symptoms, but they progress at different speeds and vary in severity.

Motor neuron diseases affect either upper motor neurons (UMN) or lower motor neurons (LMN) or both:

Type	UMN Degeneration	LMN Degeneration
Amyotrophic Lateral Sclerosis (ALS)	Yes	Yes
Progressive Muscular Atrophy (PMA)	No	Yes
Primary Lateral Sclerosis (PLS)	Yes	No
Progressive Bulbar Palsy (PBP)	No	Yes, bulbar region
Spinal Muscular Atrophy (SMA)	No	Yes

Risk factors

Here are some of the risk factors associated with MND.

Heredity: Around 1 in every 10 cases of ALS is inherited. SMA is also known to be an inherited condition.

Age: After the age of 40 years, the risk rises significantly. ALS is most likely to appear between the ages of 55 and 75 years.

Sex: Men are more likely to develop an MND.

Some experts have linked military experience to a higher chance of developing the disease. Studies have found that professional footballers are more likely to die from ALS, Alzheimer's disease and other neurodegenerative diseases, compared with other people. This implies a possible link with recurrent head trauma and neurological disease.

Causes

Motor neuron disease occurs when motor neurons in the brain and spinal cord progressively lose their function. It's not clear why this happens. In most cases, a person with motor neuron disease doesn't have a family history of the condition. This is known as Sporadic Motor Neuron Disease. Researchers believe that the cause is probably a series of steps involving a mixture of damaging genetic and environmental factors. As people get older, they may gradually lose the ability to keep this damage under control, triggering irreversible neurodegeneration.

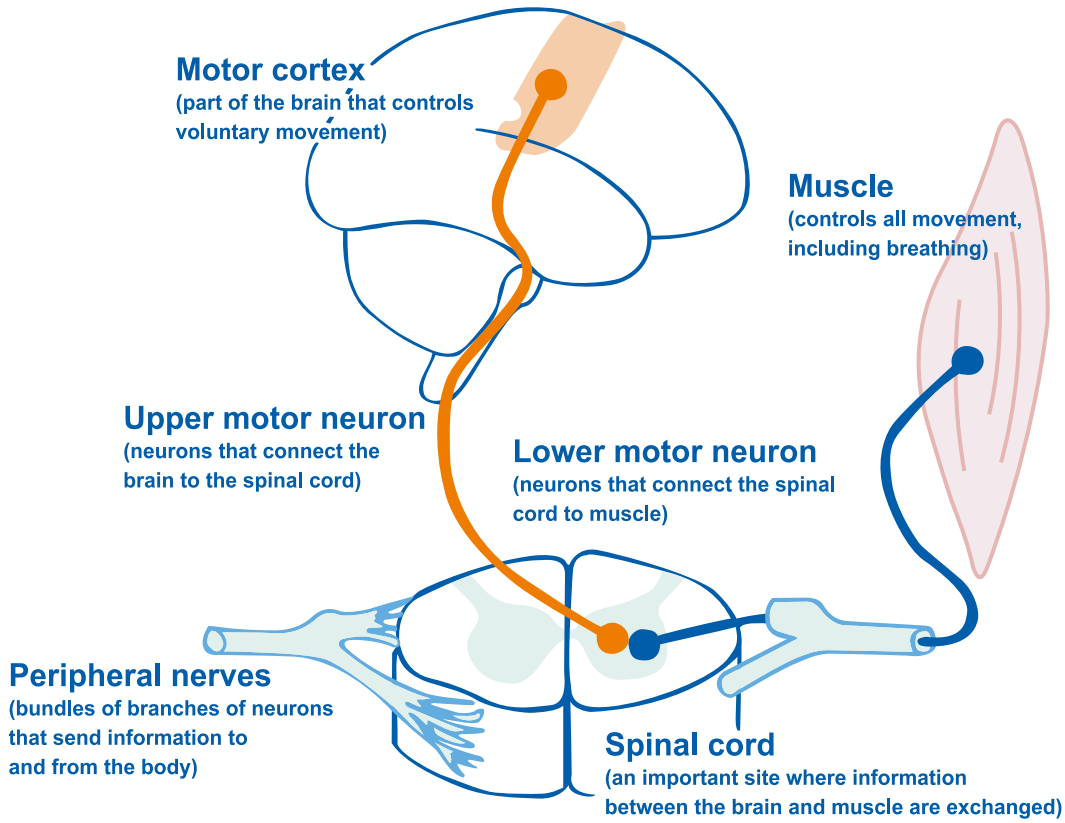
About 5% of people with motor neuron disease have a close family relative with the condition or a related condition known as frontotemporal dementia. This is called familial motor neuron disease which can be hereditary or linked to a problem with genes that cause problems at a younger age.

It's still unclear why the motor neurons begin to lose function. Most experts believe that it's a combination of interrelated factors that ultimately affect either the motor neurons or the nerve cells that support them.

Aggregates and RNA processing

Aggregates are abnormal clumps of protein that develop inside motor neurons. They are found in nearly all cases of motor neuron disease and may disrupt the normal working of the motor neurons or at least be a marker that the cell is under great strain. The most common aggregate found is TDP-43, which is a very important protein involved in the correct processing of the genetic instructions.

In Motor Neuron Disease connections from the brain to the spinal cord (upper motor neurons) and from the spinal cord to the muscle (lower motor neurons) may die. As these neurons die, control of muscles are lost.



Cell transport disruption

All cells contain transport systems that move nutrients and other chemicals into the cell and waste products out of the cell. Research suggests that in case of MND, the transport systems in motor neurons become disrupted. Over time, toxic waste can build up in cells as a natural byproduct of normal cell activity. The body gets rid of the toxic waste by producing antioxidants and packaging the waste into containers called micro-vesicles. In MND, the motor neurons may be deficient in antioxidants. However, There is no evidence that this is due to poor dietary intake.

Glial cells

Glia are cells that surround and support motor neurons and provide them with nutrients. They also help relay information from one nerve cell to another. Some cases of motor neuron disease may be caused by problems with the glial cells.

Glutamate

Nerve cells use special "messenger chemicals" called neurotransmitters to pass information from one cell to another. One of the neurotransmitters is called glutamate. There's evidence that the motor neurons in people with motor neuron disease may have become more sensitive to glutamate, resulting in damage to these cells. However, this isn't linked to dietary intake of glutamate.

Mitochondria

Mitochondria are the "batteries" of cells. They provide the energy that a cell needs to carry out its normal function. The mitochondria in the motor neurons of people with motor neuron disease seem to become abnormal.

Familial Motor Neuron Disease

The fact that motor neuron disease can run in families suggests that single genetic mutation inherited from parents may sometimes have a much larger role in the condition.

A genetic mutation occurs when the instructions carried in cells become scrambled in some way. This results in one or more of the body's processes not working properly. Four major genetic mutations have so far been identified in the 5% of people with a family history of motor neuron disease or the related condition, frontotemporal dementia. The largest group (about one third) have an expanded area of a gene called C9ORF72. Some people with this gene abnormality develop motor neuron disease, some develop frontotemporal dementia and some develop both. Other genes linked to familial motor neuron disease include SOD1, TARDBP and FUS.

If father, mother, sister or brother developed motor neuron disease and were found to have one of these abnormal genes, new member of that family has a 50% chance of carrying the same gene. However, importantly, this doesn't necessarily mean he'll definitely develop motor neuron disease in his lifetime.

If someone has been diagnosed with motor neuron disease but there's no wider family history, the overall risk to his own children is currently thought to be similar to that of the general population. Genetic testing is available to determine whether one has the mutated genes associated with familial motor neuron disease.

Symptoms

MND can be divided into three stages: early, middle and advanced.

Early stage signs and symptoms

Symptoms develop slowly and can be confused with symptoms of some other unrelated neurological conditions.

Early symptoms depend upon which body system is affected first. Typical symptoms begin in one of three areas: the arms and legs, the mouth (bulbar), or the respiratory system. They include:

- A weakening grip, making it hard to pick up and hold things
- Fatigue
- Muscle pains, cramps and twitches
- Slurred and sometimes garbled speech
- Weakness in the arms and legs
- Increased clumsiness and stumbling
- Difficulty swallowing
- Trouble breathing or shortness of breath

Middle stage signs and symptoms

As the condition progresses, symptoms become more severe.

- Muscle pain and weakness increase, and spasms and twinges worsen.

- Limbs become progressively weaker.
- Limb muscles start to shrink.
- Movement in affected limbs becomes more difficult.
- Limb muscles may become abnormally stiff.
- Joint pain grows.
- Eating, drinking, and swallowing become harder.
- Drooling occurs, due to problems controlling saliva.
- Yawning occurs, sometimes in uncontrollable bouts.
- Jaw pain may result from excessive yawning.
- Speech problems worsen, as muscles in the throat and mouth become weaker.

The person may show changes in personality and emotional state, with bouts of uncontrollable crying or laughing. Previously, it was believed MND did not significantly affect brain function or memory but studies have now shown that up to 50 percent of people with ALS have some type of brain function involvement. This includes difficulties with memory, planning, language, behavior and spatial relationships. Up to 15 percent of people with ALS have a form of dementia known as frontotemporal dementia (FTD). Breathing problems may occur as diaphragm, the main breathing muscle deteriorates. There may be a shortage of breath, even when sleeping or resting. Ultimately, breathing assistance will be necessary. Secondary symptoms include insomnia, anxiety and depression.

Advanced stage signs and symptoms

Eventually, the patient will be unable to move, eat, or breathe without assistance. Without supportive care, an individual will pass away. Despite the best of care currently available, complications of the respiratory system are the most common causes of death.

Motor neuron diseases can leave those with the disease severely restricted in mobility.

Diagnosis

In the early stage, MND can be hard to diagnose, because the signs and symptoms are common to other conditions, such as Multiple Sclerosis (MS) or Parkinson's disease.

A primary care physician will normally refer the patient to a neurologist. The neurologist will start with a complete history and physical examination of the neurologic system. Other tests may be helpful.

Blood and urine tests: These analyses can rule out other conditions and detect any rise in Creatine Kinase. This is produced when muscle breaks down, and it is sometimes found in the blood of patients with MND.

MRI: This cannot detect an MND, but it can help rule out other conditions, such as stroke, brain tumor, brain circulation problems, or abnormal brain structure.

Electromyography (EMG) and Nerve Conduction Study (NCS): These are often performed together. An EMG tests the amount of electrical activity within muscles, while NCS tests the speed at which electricity moves through muscles.

Spinal tap or lumbar puncture: This analyzes the cerebrospinal fluid, the fluid that surrounds the brain and spinal cord.

Muscle biopsy: If a physician thinks the patient may have a muscle disease, rather than MND, a muscle biopsy may be performed. After tests, physician will normally monitor the patient for some time before confirming that patient has MND.

Criteria known as **El Escorial Criteria** can help a doctor check for distinctive neurological signs, that may aid in the diagnosis of ALS.

These include:

- Muscle shrinking, weakness or twitching
- Muscle stiffness or abnormal reflexes
- Symptoms spreading into new muscle groups
- Having no other factors that explain the symptoms

Confirming the diagnosis

There are many reasons why there may be delays in diagnosis. It may be that the initial symptoms aren't thought to be serious, or they're not recognized as being related to the nervous system so a neurologist isn't initially consulted.

Sometimes, the diagnosis of motor neuron disease is clear without the need for further tests. However, confirming a diagnosis can sometimes be time-consuming even for an experienced neurologist, who occasionally needs a period of observation to be sure, particularly in cases where the condition progresses slowly. Motor neuron disease can only be diagnosed if the symptoms are clearly getting worse (progressive).

Receiving the diagnosis

Being told someone has motor neuron disease can be emotionally devastating and the news can be difficult to take in at first. Many people diagnosed with the condition go through the classic stages of the grieving process. These are:

Denial- The patient may initially disbelieve the diagnosis and think there's nothing wrong with him or that his doctor has missed another diagnosis.

Anger- Patient may feel angry towards friends, family or medical staff, particularly if he feels that the diagnosis has been unduly delayed.

Bargaining- People with terminal conditions sometimes try to "bargain" with their doctors, asking for any sort of treatment that can prolong their life.

Depression- They may lose interest in life and feel that their situation is hopeless.

Acceptance- They may come to terms with the diagnosis, the feelings of depression pass, and begin to plan for the rest of their life.

If somebody is diagnosed with motor neuron disease, talking to a counsellor or a trained clinical psychologist may help combat feelings of depression and anxiety. It's not unusual to have thoughts of taking the patient's own life, although very few people with motor neuron disease go on to do this. Taking antidepressants or medicines to reduce anxiety may also help as patient moves through the stages of the grieving process.

Treatment

There is no cure for MND, so treatments focus on slowing the progression and maximizing patient independence and comfort. This can include the use of breathing, feeding, mobility and communication appliances and devices. Rehabilitation therapy may include physical, occupational and speech therapy.

Riluzole

Riluzole is the only medication that has shown a survival benefit for people with motor neuron disease. Riluzole is thought to slow down the progressive damage to the motor neuron cells by reducing their sensitivity to the nerve transmitter glutamate. In medical research, Riluzole extended survival by two to three months on average, although this varied from person to person and the condition continued to progress even with Riluzole treatment. Side effects of Riluzole are usually mild and commonly include nausea, tiredness and less commonly, a rapid heartbeat. Very rarely, Riluzole has been known to cause liver damage.

Treating symptoms

A range of treatments can relieve many of the symptoms of motor neuron disease and improve the patient's quality of life.

Muscle cramps and stiffness

Muscle cramps and stiffness can be treated with physical therapy and medications, such as botulinum toxin (BTA) injections. BTA blocks the signals from the brain to the stiff muscles for about 3 months.

Baclofen, a muscle relaxer, may reduce muscle stiffness. A small pump is surgically implanted outside the body and connected to the space around the spinal cord. A regular dose of Baclofen is delivered into the nervous system. Baclofen blocks some of the nerve signals that cause spasticity. It may help with extreme yawning.

Treatment for drooling

Scopolamine, a drug for motion sickness, may help control symptoms of drooling. It is worn as a patch behind the ear.

Uncontrolled laughter or crying

Antidepressants like serotonin reuptake inhibitors (SSRIs), may help with episodes of uncontrollable laughter or crying, known as emotional lability.

Speech, occupational and physical therapy

Patients with speech and communication difficulties may learn some useful techniques with a qualified speech and language therapist. As the disease advances, patients often need some communication aids. Physical and occupational therapy can help maintain mobility and function, and reduce stress.

Swallowing difficulties

Not everyone with motor neuron disease will have significant swallowing problems (dysphagia). For those having dysphagia, it can prevent normal eating and drinking. If food goes down the wrong way into the lungs, it can cause chest infections. Weight loss due to poor nutrition can also accelerate motor neuron disease.

One widely used treatment for dysphagia is a thin feeding tube known as a percutaneous endoscopic gastrostomy (PEG) tube. The tube is surgically implanted into stomach through a small cut on the surface of the stomach. It shouldn't restrict someone's daily activities and he can continue to bathe and swim normally if he wishes. It's advisable to have the tube inserted before the breathing muscles are significantly weakened, even if it isn't used for feeding until sometime later.

Pain relief

Motor neuron disease isn't usually a painful condition. If someone experiences pain, it's often aching joints caused by muscle weakness or a change in posture. The type of painkiller recommended will depend on how severe the pain is. Mild to moderate pain can often be controlled using non-steroidal anti-inflammatory drugs (NSAIDs) such as Ibuprofen. More severe pain is very rare, but it can be treated using an opiate-based painkiller such as Morphine. In some cases, Gabapentin is used. It was originally designed to treat epilepsy but it's also useful for treating pain.

Breathing problems

Respiratory muscles usually weaken gradually, but a sudden deterioration is possible.

Mechanical ventilation can help with breathing. A machine takes in air, filters it and pumps it into the lungs often through a tracheostomy. Some people use complementary therapies, including special diets that are high in vitamins. These will not cure MND, but following a healthful diet can improve overall health and wellbeing.

Complementary therapies

Some people with motor neuron disease find complementary therapy helpful. This involves combining conventional treatments with non-medical treatments, such as acupuncture.

Complementary therapies can't slow the progression of motor neuron disease, but they may help reduce stress and make someone's daily life more comfortable.

Stem cell transplant for ALS treatment

Stem cell research and gene therapy have shown promise for treating ALS in the future, but more studies are needed.

Outlook

MND is usually fatal. Depending on the type, most people will not survive longer than 5 years after symptoms appear, but some people live 10 years or longer, depending on the extent and progression of their disease. Living with an MND can be extremely challenging, and diagnosis can seem like a "terrifying possibility." However, with community and other support, the patient's quality of life is often better than expected and the advanced stage "isn't usually distressing." For most people, this comes at home, while sleeping.

Patients may wish to prepare an advanced directive, in which they can state their future wishes about treatment while they are still able to express themselves. Issues may include where they hope to be treated in the later stages, for example, in the hospital, in a hospice center or at home with hospice care and whether they would like to receive mechanical breathing or other assistive care.

As medical research continues, scientists & doctors hope to understand MND fully. They are working towards finding new treatments.

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Foodborne diseases are defined by the World Health Organization as "diseases of infectious or toxic nature caused by or thought to be caused by the consumption of food or water".

Foodborne diseases is the ultimate outcome of failure in the management of good food safety practices. Each year a remarkable number of people are suffered from foodborne diseases. Foodborne disease is not only due to the presence of pathogenic microorganisms but also the presence of toxic and hazardous chemical present in the food. Lack of personal hygiene and sanitation is mainly responsible for foodborne diseases caused by the contamination of microorganisms.

Most foodborne diseases are acute, meaning they happen suddenly and last a short time, and most people recover on their own without treatment. Rarely, foodborne diseases may lead to more serious complications.

Epidemiology:

Foodborne diseases are an important public health problem worldwide and have an important impact on travel, trade, and development. The World Health Organization (WHO) estimated that 31 foodborne hazards caused 600 million foodborne diseases and 420,000 deaths worldwide in 2010. Many of these foodborne diseases and deaths were caused by diarrheal diseases, a leading cause of global deaths in young children. U.S. Centers for Disease Control and Prevention (CDC) estimates that one in six people contract a foodborne diseases each year in the United States and of these.

Causes:

The majority of foodborne diseases is caused by harmful bacteria and viruses. Some parasites and chemicals also cause foodborne diseases.

Bacteria

Bacteria are tiny organisms that can cause infections of the GI tract. Not all bacteria are harmful to humans. Some harmful bacteria may already be present in foods when they are purchased. Raw foods including meat, poultry, fish and shellfish, eggs, unpasteurized milk and dairy products and fresh produce often contain bacteria that cause foodborne diseases. Bacteria can contaminate food-making it harmful to eat at any time during growth, harvesting or slaughter, processing, storage and shipping.

Foods may also be contaminated with bacteria during food preparation in a restaurant or home kitchen. If food preparers do not thoroughly wash their hands, kitchen utensils, cutting boards and other kitchen surfaces that come into contact with raw foods, cross-contamination the spread of bacteria from contaminated food to uncontaminated food may occur.

Many types of bacteria cause foodborne diseases. Examples include-

- Salmonella, a bacterium found in many foods, including raw and undercooked meat, poultry, dairy products, on egg shells and inside eggs.
- Campylobacter jejuni (C. jejuni), found in raw or undercooked chicken and unpasteurized milk.
- Shigella, a bacterium spread from person to person. These bacteria are present in the stools of people who are infected. If people who are infected do not wash their hands thoroughly after using the bathroom, they can contaminate food that they handle or prepare.
- Escherichia coli (E. coli), which includes several different strains, only a few of which cause illness in humans. E. coli O157:H7 is the strain that causes the most severe illness.
- Listeria monocytogenes (L. monocytogenes), which has been found in raw and undercooked meats, unpasteurized milk, soft cheeses.
- Vibrio, a bacterium that may contaminate fish or shellfish.
- Clostridium botulinum (C. botulinum), a bacterium that may contaminate improperly canned foods and smoked and salted fish.

Viruses

Viruses cause infections that can lead to sickness. People can pass viruses to each other. Viruses are present in the stool or vomit of people who are infected. People who are infected with a virus may contaminate food and drinks, especially if they do not wash their hands thoroughly after using the bathroom.

Common sources of foodborne viruses include-

- Food prepared by a person infected with a virus
- Shellfish from contaminated water

- ❏ Produce irrigated with contaminated water

Common foodborne viruses include-

- ❏ Norovirus, which causes inflammation of the stomach and intestines
- ❏ Hepatitis A, which causes inflammation of the liver

Parasites

Cryptosporidium parvum and Giardia intestinalis are parasites that are spread through water contaminated with the stools of people or animals that are infected. Foods that come into contact with contaminated water during growth or preparation can become contaminated with these parasites. Food preparers who are infected with these parasites can also contaminate foods if they do not thoroughly wash their hands after using the bathroom and before handling food.

Trichinella spiralis is a type of roundworm parasite. People may be infected with this parasite by consuming raw or undercooked pork or wild game.

Chemicals

Harmful chemicals that cause illness may contaminate foods such as

- ❏ Fish or shellfish, which may feed on algae that produce toxins, leading to high concentrations of toxins in their bodies. Some types of fish, including tuna and mahi mahi, may be contaminated with bacteria that produce toxins if the fish are not properly refrigerated before they are cooked or served.
- ❏ Certain types of wild mushrooms.
- ❏ Unwashed fruits and vegetables that contain high concentrations of pesticides.

Pathogenesis:

The diseases caused by foodborne pathogens can be classified into three forms: foodborne infection, foodborne intoxication and foodborne toxicoinfection. The principal route of infection for foodborne pathogens is oral and the primary site of action is the intestine. Most foodborne microorganisms causes localized infection and tissue damage but some spread to deeper tissues to induce systemic infection.

Target population:

Anyone can get a foodborne diseases. However, some people are more likely to develop foodborne diseases than others, including-

- ❏ Infants and children
- ❏ Older adults
- ❏ Pregnant women and their fetuses
- ❏ People with weak immune systems

Symptoms:

Symptoms of foodborne diseases depend on the cause. Common Symptoms of many foodborne diseases include-

- ❏ Vomiting
- ❏ Diarrhea or bloody diarrhea
- ❏ Abdominal pain
- ❏ Fever
- ❏ Chills

Symptoms can range from mild to serious and can last from a few hours to several days.

C. botulinum and some chemicals affect the nervous system, causing symptoms such as-

- ❏ Headache
- ❏ Tingling or numbness of the skin
- ❏ Blurred vision
- ❏ Weakness
- ❏ Dizziness
- ❏ Paralysis

Treatment:

The only treatment needed for most foodborne diseases is replacing lost fluids and electrolytes to prevent dehydration.

Over-the-counter medications such as loperamide may help stop diarrhea in adults.

If the specific cause of the foodborne diseases is diagnosed, may need medications, such as antibiotics, to treat the illness.

Hospitalization may be required to treat life threatening symptoms and complications.

Complication:

Foodborne diseases may lead to dehydration, hemolytic uremic syndrome (HUS) and other complications. Acute foodborne diseases may also lead to chronic or long lasting health problems.

Some foodborne diseases lead to other serious complications. For example, C. botulinum and certain chemicals in fish and seafood can paralyze the muscles that control breathing. L. monocytogenes can cause spontaneous abortion or stillbirth in pregnant women.

Acute foodborne diseases may lead to chronic disorders, including-

- ❏ Reactive arthritis
- ❏ Irritable bowel syndrome (IBS)
- ❏ Guillain-Barré syndrome

Prevention:

A few simple actions can cut the likelihood of foodborne illness drastically. WHO's Five keys to safer food are as follows-

1. To keep clean
2. To separate raw and cooked food
3. Cooking thoroughly
4. Keeping food at safe temperatures
5. Using safe water and raw materials

Foodborne disease in the context of Bangladesh

The food safety component of WHO program in Bangladesh aims at building capacity of government institution, Institute of Public Health of DGHS to develop appropriate and effective management frameworks that ensure safety of food from production to the point of consumption.

The food safety program also emphasizes for the need for monitoring of food safety and foodborne illness with modern risk based approach and techniques.

The food contamination and food adulteration situation of Bangladesh is a serious public health concern. Unsafe/contaminated food causes many acute and life-long diseases, ranging from diarrheal diseases to various forms of cancer. However, in Bangladesh dependable assessment of the public health impact due to food contamination is not available due to absence of a regular monitoring system. Limited data from the ICDDR,B indicates 501 hospital visits per day for treatment of diarrhea that were attributable to food and water borne causes.

There is also widespread evidence of food adulteration with harmful chemicals. The chronic effect of such events such as cancer, kidney disorders and birth defects is unlikely to be observed in short term, because the manifestation of the disease only occurs after long-term, low-level exposure. Food contamination and consumers exposure to food hazards have major implication on the food security and consumers heath in Bangladesh. Low level of awareness and weakness in existing food laws and regulation are also contributing to aggravating the country's food safety situation.

Protecting public health from such hazards therefore requires a comprehensive risk analysis and risk management approach of food safety from production to consumption. Further, raising awareness, ensuring safe water, sanitation and improved hygiene practice will play a significant role in reducing foodborne illness.

WHO is advocating for strengthening of government capacity to monitor food safety with modern and comprehensive risk management approach for last few years. So far WHO Bangladesh has:

- Supported the government in capacity building of government institutions through developing training manuals
- Supported in conducting a series of training programs for the health professionals;
- Developed an appropriate IEC (International Electro technical Commission) material for raising awareness among food producers, processors handlers and traders at public and private sector.
- Supported in conducting national and divisional level seminars symposium etc.

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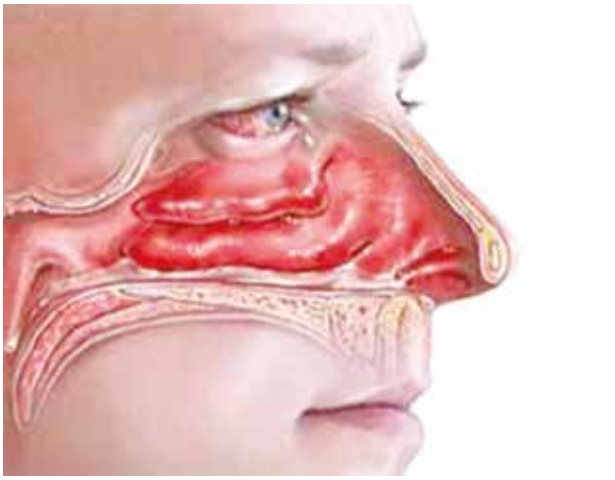
Allergic Rhinitis in Children

Allergic rhinitis or hay fever, is swelling of the inside of child's nose. Rhinitis is a reaction that happens in the eyes, nose and throat when allergens in the air trigger the release of histamine in the body. Histamine causes itching, swelling and fluid to build up in the fragile linings of nasal passages, sinuses and eyelids. Allergens include pollen in weeds, grass



and trees. Indoor dust mites, cockroaches, pet dander or mold are other allergens that can cause allergic rhinitis.

Types of allergic rhinitis

The two categories of allergic rhinitis include:

- **Seasonal -** occurs particularly during pollen seasons. Seasonal allergic rhinitis does not usually develop until after 6 years of age.
- **Perennial -** occurs throughout the year. This type of allergic rhinitis is commonly seen in younger children.

According to severity and duration

Intermittent : < 4 days per week or < 4 weeks.
Persistent : > 4 days per week and > 4 weeks.

Mild

Normal sleep
No impairment of daily activity
Normal work and school
No troublesome symptom

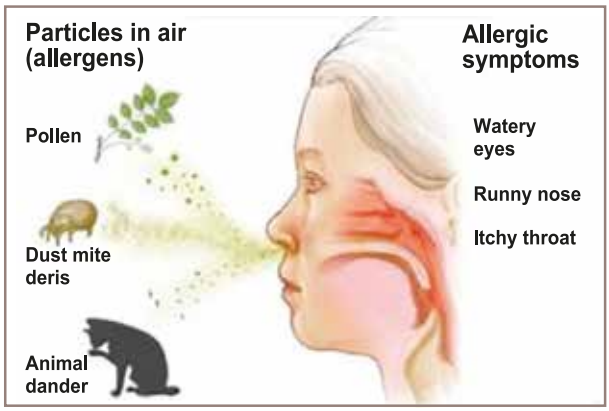
Moderate-severe one or more items

Abnormal sleep
Impairment of daily activity
Abnormal work and school
Trouble-some symptom

Causes

Allergic rhinitis is caused by an allergic reaction to an allergen, such as-

- Pollen from trees, grass or weeds
- Dust mites
- Mold



- Cockroach
- Animal dander
- Tobacco smoke

Oversensitive immune system

When immune system is oversensitive, it will react to allergens by producing antibodies to fight them off. Antibodies are special proteins in the blood that are usually produced to fight viruses and infections.

Allergic reactions don't occur the first time with contact of an allergen. The immune system has to recognize and "memorize" it before producing antibodies to fight it. This process is known as sensitization.

After developing sensitivity to an allergen, it will be detected by antibodies called immunoglobulin E (IgE) whenever it comes into contact with inside of nose and throat.

These antibodies cause cells to release a number of chemicals, including histamine, which can cause the inside layer of nose to become inflamed and produce excess mucus. This is what causes the typical symptoms of sneezing and a blocked or runny nose.

Common allergens

Allergic rhinitis is triggered by breathing in tiny particles of allergens. The most common airborne allergens that cause rhinitis are described below.

House dust mites

House dust mites are tiny insects that feed on the dead flakes of human skin. They can be found in mattresses, carpets, soft furniture, pillows and beds.

Pollen and spores

Tiny particles of pollen produced by trees and grasses can sometimes cause allergic rhinitis. Most trees pollinate from early to mid-spring, whereas grasses pollinate at the end of spring and beginning of summer.

Rhinitis can also be caused by spores produced by mould and fungi.

Animals

Many people are allergic to animals, such as cats and dogs. The allergic reaction isn't caused by animal fur, but flakes of dead animal skin and their urine and saliva.

Dogs and cats are the most common culprits, although some people are affected by horses, cattle, rabbits and rodents, such as guinea pigs and hamsters.

However, being around dogs from an early age can help protect against allergies and there's some evidence to suggest that this might also be the case with cats.

Work-related allergens

Some people are affected by allergens found in their work environment, such as wood dust, flour dust of latex.

Risk factors

Children with asthma are at a higher risk for rhinitis. Allergic rhinitis is a common problem that may be linked to asthma. However, this link is not fully understood. Experts think that since rhinitis makes it hard to breathe through the nose, it is harder for the nose to work normally. Breathing through the mouth does not warm, filter or humidify the air before it enters the lungs. This can make asthma symptoms worse.

Controlling asthma may help control allergic rhinitis in some children.

Signs and symptoms

- ❏ Sneezing
- ❏ Nasal congestion (child may breathe through his or her mouth at night or snore)
- ❏ Runny nose
- ❏ Itchy nose, eyes, or mouth
- ❏ Red, watery eyes
- ❏ Postnasal drip
- ❏ Cough or frequent throat clearing
- ❏ Feeling tired or lethargic
- ❏ Dark circles under child's eyes

Children with year-round allergic rhinitis may also have these symptoms:

- ❏ Ear infections that keep coming back
- ❏ Snoring
- ❏ Breathing through the mouth
- ❏ Poor performance in school
- ❏ A line or crease across the bridge of the nose from swiping the nose

The symptoms of allergic rhinitis may look like other conditions or medical problems.

Alarming sign:

- ❏ Child's symptoms get worse, even after treatment.
- ❏ If child has a fever.
- ❏ Child has ear or sinus pain, or a headache.
- ❏ If child has yellow, green, brown, or bloody mucus coming from his or her nose.

- ❏ Child's nose is bleeding or child has pain inside his or her nose.
- ❏ If child has trouble sleeping because of his or her symptoms.

Diagnosis

- ❏ Skin testing may show the child is allergic. After pricking or scratching child's skin with tiny amounts of a possible allergen, a bump may appear within a few minutes. It indicates that child is likely allergic to the allergen.
- ❏ A blood test may be done to find out that the child is allergic to.

Management

The best way to manage child's allergic rhinitis is to avoid allergens that can trigger his or her symptoms. Any of the following may help decrease child's symptoms:

- **To rinse child's nose and sinuses** with a salt water solution or use a salt water nasal spray. This will help thin the mucus in child's nose and rinse away pollen and dirt. It will also help reduce swelling so he or she can breathe normally.
- **To reduce exposure to dust mites:** Sheet & Towels should be washed in hot water every week. Blankets need to be washed every 2 to 3 weeks in hot water. Child's pillows and mattresses must be covered by allergen free covers. Limiting the number of stuffed animals and soft toys of child is required. Child's toys should be washed in hot water regularly. Vacuum may be used for indoor cleaning by vacuum cleaner with an air filter. If possible, carpets and curtains should be avoided.
- **To reduce exposure to pollen:** Windows and doors of house and cars should be kept closed. Child must stay inside when air pollution or the pollen count is high. Running air conditioner on recycle and changing air filters frequently is required. Taking shower and washing child's hair before bed every night may be needed to rinse away pollen.
- **To reduce exposure to pet dander:** If possible, keeping cats, dogs, birds or other pets should be avoided. In case of keeping pets at home, they should be out of bedrooms, carpeted rooms and should be bathed regularly.
- **To reduce exposure to mold:** Spending time in basement should be prohibited. Artificial plants instead of live plants can be chosen. Optimum humidity at home is less than 45% should be maintained. Getting rid of ponds or standing water at home or yard is needed.

Treatment

- **Over-the-counter antihistamines -** Antihistamines help to decrease the release of histamine, possibly decreasing the symptoms of itching, sneezing or runny nose. Some examples of antihistamines are diphenhydramine or hydroxyzine. These medications may cause drowsiness.
- **Nonsedating prescription antihistamines -** Nonsedating antihistamines work like antihistamines but without the side effect of drowsiness. Nonsedating antihistamines may include cetirizine, loratadine or fexofenadine.
- **Anti-inflammatory nasal sprays -** Anti-inflammatory nasal sprays help to decrease the swelling in the nose.
- **Corticosteroid nasal sprays -** Corticosteroid nasal sprays also help to decrease the swelling in the nose. Corticosteroid nasal sprays work best when used before the symptoms start, but can also be used during a flare-up.
- **Decongestants -** Decongestants help by making the blood vessels in the nose smaller, thus, decreasing congestion. Decongestants can be purchased either over-the-counter or by prescription.

If child does not respond to avoidance or to the above medications, then may recommend allergy shots or immunotherapy based on the findings. Immunotherapy usually involves a three to five year course of repeated injections of specific allergens to decrease the reaction to these allergens.

Complication

- Asthma exacerbations
- It is also associated with otitis media
- Eustachian tube dysfunction
- Allergic conjunctivitis
- Atopic dermatitis
- Allergic rhinitis may also contribute to learning difficulties, sleep disorders and fatigue
- Hyperlacrimation
- Sinusitis
- Chemosis of conjunctiva
- Nasal polyps
- Rhinorrhoea

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Test Yourself - 47

Correct Answers :

1.	c	2.	d	3.	d	4.	a	5.	a	6.	b

CONGRATULATIONS!

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Test Yourself - 48

1. The followings are true for "Foodborne Disease" except:

a. Hepatitis -A and Norovirus are common foodborne viruses.

b. The diseases caused by foodborne pathogens can be classified into four forms.

c. Infant and children, older people and pregnant women are more likely to develop foodborne disease.

d. Acute foodborne disease may lead to chronic disorders including Irritable Bowel Syndrome, Guillain-Barre Syndrome.

2. All the followings are correct for "Dengue Hemorrhagic Fever (DHF)" accept:

a. The interactions between rainfall and temperature are not important determinants of dengue transmission.

b. A person can be infected as many as four times once with each serotype.

c. According to W.H.O it is estimated that fifty to hundred million infections of dengue fever occur yearly.

d. Typical cases of DHF are characterized by four major clinical manifestations.

3. All the below are true for "Allergic Rhinitis in Children" except:

a. Some medications can cause non-allergic rhinitis.

b. Allergic conjunctivitis, sinusitis, nasal polyps, otitis externa are the complications of allergic rhinitis.

c. Allergic rhinitis is a common problem that may be linked to asthma.

d. Perennial allergic rhinitis is commonly seen in the younger children.

4. All the followings are correct for "Motor Neuron Disease (MND)" except:

a. Amyotrophic Lateral Sclerosis is not a common type of motor neuron disease.

b. In 1874 a French doctor first described motor neuron disease.

c. The different types of MND share similar symptoms but they progress at different speed and vary in severity.

d. Spinal muscular atrophy is an inherited type and affects children.

5. The followings are right for "Dengue Hemorrhagic Fever (DHF)" except:

a. Worldwide children younger than 15 years constitute nine percent of dengue hemorrhagic fever patients.

b. Dengue hemorrhagic fever is classified into four grades of severity.

c. RT-PCR yields a serotype- specific, diagnosis very rapidly

d. The critical stage of the disease course is reached at the beginning of the febrile phase.

6. All the followings are correct for "Motor Neuron Disease (MND)" except:

a. Aggregates are abnormal clumps of protein are found in nearly all motor neuron disease.

b. Complications of the skeletal system are the most common causes of death despite the best of care.

c. Stem cell research and gene therapy have shown promise for treating Amyotrophic Lateral Sclerosis in the future. .

d. Riluzole is thought to slow down the progressive damage to the motor neuron cells.

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