

EMERGING AND RE-EMERGING PARASITIC DISEASES

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Abstract: Human emerging and reemerging infectious diseases are the diseases that have appeared in a population in the recent past or that had already existed but are rapidly increasing in incidence or changing their geographic range. An increased incidence of zoonotic and vector borne infections has been recognized. The factors responsible for this emergence or reemergence of diseases include overpopulation, disruptions due to military action, mass migrations of populations due to natural or man-made disasters and the migration of populations into large urban centers and inadequate food and water supplies. Socio-economic and environmental factors e.g., greater rates of trade and travel, agricultural changes and increased antibiotic drug use, deforestation and climate change are also considered as important drivers of the emergence of these diseases. A brief review of clinically significant, food and water borne; vector borne, blood borne and zoonotic emerging and reemerging parasitic diseases has been given. For effective control of these diseases, proper surveillance of the factors influencing their emergence, early detection and monitoring of these pathogens are required. Diagnostics and therapeutics based on genomics, proteomics and nanotechnology should be explored for rapid and accurate diagnosis.

INTRODUCTION

The term emerging and re-emerging infectious diseases have been coined to a group of diseases that have appeared in a population in the recent past or that have existed but are rapidly increasing in incidence or changing their geographic range. The parasitic diseases continue to be a significant health problem in both developed and developing countries. WHO estimates that one person in every four harbors parasitic worms¹. The parasitic infections prevalent in the developing tropical countries are spreading to the developed nontropical countries by the tourists, soldier, or immigrants. Parasitic diseases, such as malaria, trypanosomiasis, schistosomiasis and leishmaniasis have re-emerged in recent years. African trypanosomiasis has reached epidemic proportions. During the past decade cutaneous leishmaniasis has also emerged as a challenging infectious disease in the form of new outbreaks in areas not identified previously. Food borne and water borne protozoan diseases such as, *Cryptosporidium parvum*, *Cyclospora*, *Giardia lamblia*, *Entamoeba histolytica*, *Blatocystis hominus* etc., have also increased².

FACTORS RESPONSIBLE FOR EMERGENCE OR REEMERGENCE

Many factors or combinations of factors contributing to disease emergence include ecological changes, such as those due to human activities or to anomalies in climate; demographic changes and behavioral changes, travel and immigration, technology and industry; microbial adaptation and change and breakdown of public health measures³. The emerging infectious diseases are also attributed to the population growth, ageing population, poverty and malnutrition, environmental pollution, deforestation, crowding, inadequate infrastructure, poor sanitation and water supply, global warming, development of antimicrobial/insecticide resistance etc⁴. In addition to this the immunocompromised patients, including patients with AIDS, solid organ transplant recipients, and patients on immunosuppressive therapy for disorders, are at high risk for opportunistic parasites^{2,5}. The emerging and reemerging parasitic diseases can be reviewed under following headings. i) Foodborne and waterborne parasitic diseases ii) Vector borne parasitic diseases iii) Zoonotic parasitic diseases and iv) Transfusion associated parasitic diseases

FOODBORNE AND WATERBORNE PARASITIC DISEASES

A brief account of some of the emerging and reemerging food and water borne parasitic diseases on the basis of their disease potential has been given (Table 1).

Table 1: Emerging and reemerging food and water borne parasitic diseases.

| Disease /Name and Type of the Parasite | Mode of Infection | Clinical Symptoms | Diagnostic Methods | Treatment |
|---|---|---|--|--|
| Cryptosporidiosis <i>Cryptosporidium parvum</i> (Protozoa) | Food or drink contaminated with feces infected with Sporulated Oocyst | Watery diarrhea, malaise, nausea, fever, crampy abdominal pain, and flu like illness Immunocompromised have more severe diarrhea with cholera like symptoms. | Detection of oocysts in faeces, modified acid-fast stains or auramine-O staining, ELISA for Antigen detection, Real Time PCR (18S rRNA) | Oral spiramycin and Nitazoxanide |
| Microsporidiosis <i>Enterocytozoon bienersi</i> and <i>Ecephalitozoon intestinalis</i> , <i>Enterophthalmozoon</i> spp. <i>Vitroformia corneae</i> , <i>Nosoma</i> spp etc. (Protozoa) | Ingestion or inhalation of spores. | Debilitating chronic diarrhea and malabsorption. In disseminated infection- ocular infections- kerato conjunctivitis, blurred or decreased vision). genitourinary, respiratory, or musculoskeletal infections. | Direct or stained stool smear examination. Stains recommended - Weber's chromotrope or Calcofluor white M2R or Uvitex 2B. Cytology Histology trichrome, Grams, Giemsa, or Brown-Hopps. | Albendazole |
| Cyclosporiasis: <i>Cyclospora cayentanensis</i> (Protozoa) | fecal-oral route i.e by ingestion of Sporulating oocysts | Watery diarrhea, cramps,nausea, bloating, flatulence, weight loss and fatigue | Acid-fast stains Small-bowel biopsies PCR in human feces | Among Immunocompetents - Self limiting infection. In Immunocompromised |
| Blastocystosis: <i>Blastocystis hominis/ Blastocystis</i> spp. (Protozoa) | fecal-oral route Ingestion of Parasite cysts | Intense abdominal disorders together with pain, diarrhea and constipation | Direct microscopy, Giemsa and trichrome stained smears and culture | Metronidazole, Emetine, Trimethoprim-sulfamethoxazole |
| Toxoplasmosis: <i>Toxoplasma gondii</i> (Protozoa) | Ingestion of water and food contaminated with oocysts or tissue cyst in raw or undercooked meat | Immunocompetent patients: Lymphadenopathy, fever, malaise, night sweat, myalgia, or retinochoroiditis, or retinochoroiditis. Immunosuppressed patients: encephalitis, meningoencephalitis, pneumonitis, myocarditis, and chorioretinitis. Congenital: Retinochoroiditis, cerebral calcifications, convulsions, microcephaly, hydrocephalus, mental retardation | Serology - Sabin and Fieldman dye test, IgM/IgG antibody or Antigen detection by ELISA. Histological procedures/ Imaging, PCR for DNA detection in CSF/BAL/Amniotic fluid | Pyrimethamine, Sulphadiazine, Clindamycin, Atovaquone and Azithromycin |
| Cysticercosis: <i>Taenia solium</i> Larva <i>Cyrtosercus cellulosae</i> (Cestode) | Ingestion of food or water contaminated with feces with <i>Taenia solium</i> eggs | Seizures, headaches, focal neurologic symptoms, visual disturbances, and localized skeletal muscle nodules and pain Ophthalmic: proptosis, diplopia, and loss of vision | Direct microscopy, Giemsa and trichrome stained smears and culture | Metronidazole, Emetine, Trimethoprim-sulfamethoxazole |
| Trematodiasis: <i>Clonorchis sinensis</i> , <i>Fasciola</i> spp., <i>Ophisthorchis</i> spp., <i>Paragonimus</i> spp., <i>Echinostoma</i> spp., <i>Fasciolopsis buski</i> | Ingestion of metacercariae By ating raw/ insufficiently cooked freshwater fish, crab, crayfish, snails/ tadpoles/ aquatic plants or drinking contaminated water. | Acute infection: diarrhea, abdominal pain, and urticaria by fever, cough, night sweat, dyspnea and chest pain Chronic Infection: cirrhosis, progressive ascites,catarrhal cholecystitis, hepatomegaly and jaundice | Microscopy: Direct detection of flask shaped operculated eggs in faeces/Sputum. Serological tests: IHA, Latex, agglutination, ELISA and Western blot Radiodiagnosis for pulmonary and cerebral infections. | Praziquantel, Bithionol, Niclofolan, Surgical removal of worms in biliary tract obstruction |

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Cryptosporidiosis:

Cryptosporidiosis is caused by *Cryptosporidium parvum*, one of the most common enteric protozoan parasite. It is prevalent worldwide with a prevalence rate of 0.6% to 20% in western countries and 5 to 10% in Asia and Africa^{6,7}. It is a significant cause of diarrhoeal disease and an ubiquitous contaminant of water, which serves as an excellent vehicle for transmission. It was first diagnosed as a human pathogen in 1976 in two immunocompromised patients with persistent diarrhea. Man acquires infection by ingestion of food or drink contaminated with feces, containing sporulated oocysts of *cryptosporidium*⁸. Common symptoms of disease are watery diarrhea, malaise, nausea, fever, crampy abdominal pain, and flu like illness. Watery diarrhea can be more severe with cholera like symptom among immunocompromised patients⁷. Spiramycin and Nitazoxanide are the drugs of choice⁹.

Microsporidiasis

Microsporidia are, obligate-intracellular and spore forming protozoa that infect the intestine, liver, kidney, brain, and other tissues. There are 13 species, which have been shown to cause disease in humans⁷. The most common microsporidia are *Enterocytozoon bienersi* and *Encephalitozoon intestinalis*. Microsporidia are distributed world over. Cases have been reported from America, Asia including India, Europe and Africa.

Spores are the infective forms of this parasite. Their size measures 1.5 µm to 4 µm in *Encephalitozoon sps. Vittaforma corneae* and *Nosema sps.* and from 0.8 µm to 1.4 µm in the case of *Enterocytozoon bienersi*. They have increased incidence in patients with acquired immunodeficiency syndrome^{10,11}. Humans get infection by ingestion or inhalation of spores. Intestinal infection cause debilitating chronic diarrhea and malabsorption. Disseminated infection can lead to genitourinary, respiratory, musculoskeletal or ocular infections causing keratoconjunctivitis, blurred or decreased vision.

Cyclosporiasis:

The causative agent is a coccidian protozoan parasite named *Cyclospora cayetanensis*. It was first described as a human pathogen in 1994. Infection occurs via the fecal-oral route by ingestion of contaminated water or produce. Human-to-human infection is made less likely because of the long sporulation time (at least 7 days) after shedding in feces. Most cases occur in tropical and subtropical countries. Cyclosporiasis is endemic in Nepal, Peru and Haiti¹².

The infective forms, are sporulating oocysts containing two sporocysts, each measuring 4 µm in diameter and containing four sporozoites. In the intestine the oocysts releases sporozoites, which invade the enterocytes. The sporozoites develop into unsporulated oocysts, which are excreted in faeces. Oocysts are twice the diameters of *Cryptosporidium* i.e. measuring 8 µm to 10 µm. It is a self-limiting infection in an immunocompetant host This infection is associated with watery diarrhea, cramps, nausea, bloating, flatulence, weight loss and fatigue^{6,13}.

Blastocystosis

It is a zoonotic disease caused by an intestinal protozoan named Blastocystis as *Blastocystis hominis* while *Blastocystis sp* is used for isolates from other animal hosts. A wide range of animals e.g. mammals, birds, reptiles, amphibians, arthropods are known to be reservoir of this infection. Blastocystis sps. Infection is the most common protozoan detected in human faecal samples worldwide¹⁴. It has various morphological forms like vacuolar, granular, amoeboid, and cyst forms. It is transmitted by faecal- oral route. The infective form is the parasite cyst.

The common symptoms are intense abdominal disorders together with pain diarrhea and constipation Infection occurs in both immunocompetent and immunocompromised individuals. Prevalence is much less in developed countries (2-10%) as compared to developing countries (20-50%). Poor hygiene and sanitation facilities are major contributing factors. Population at risk is travelers and immigrants to developing tropical countries, soldiers posted in endemic countries, refugees, school children, food and animal handlers, people who have pets like cats etc are more prone to diarrhea due to Blastocystis. The predisposing conditions are immunosuppressed patients, irritable bowel syndrome patients etc¹⁵. In the changing scenario Blastocystosis should be considered as re-emerging potential zoonosis.

Toxoplasmosis

Toxoplasmosis caused by a protozoan parasite, *Toxoplasma gondii* is one of the most common human parasite. Man acquires infection in several ways: by the accidental ingestion of oocysts shed in cat feces, by the ingestion of rare or raw meats, in utero, and by transfusion. In Western Europe, Africa, South and Central America more than 50% of women of childbearing age are seropositive. In France the prevalence of congenital toxoplasmosis is 1 in 1000 live births. Among HIV patients a prevalence rate of 50% to 75% has been reported⁷ Two outbreaks of acute toxoplasmosis involving 8 adult patients in Korea were linked to eating uncooked pork^{16,17}. The organisms are obligate intracellular parasites. The infectious stages of *T. gondii* are tachyzoites, bradyzoites and the oocysts. Tachyzoites are the active, multiplying trophozoites observed during acute stage of infection. Bradyzoite can be found in chronic form. Tachyzoites are the diagnostic forms. They can be found in any organ but are common in skeletal muscles, cardiac muscles and brain⁷. The most severe symptoms are seen in congenital, transplacental infections and infections in the immunocompromised patients. Toxoplasmosis can cause lymphadenopathy, fever, malaise, night sweat, myalgia, or retinochoroiditis. Among immunosuppressed patients it can cause encephalitis, meningoencephalitis, pneumonitis, myocarditis, and chorioretinitis. Retinochoroiditis, cerebral calcifications, convulsions, microcephaly, hydrocephalus, mental retardation are the classic clinical triads of congenital infections. In ophthalmic cysticercosis presents with - proptosis, diplopia, and loss of vision⁷.

Cysticercosis

It is the parasitic infection caused by larval form of cestode parasite *Taenia solium*. It is endemic in Mexico, Central and South America, and parts of Africa, Asia. In India it is more common in northern parts. Neurocysticercosis (NCC) is one of the serious complication of this infection. 26.3% to 53.8% of adult epilepsy cases in the developing world including India and Latin America are due to NCC. It is also becoming more common in the developed world because of increased migration of people with the disease or *T. solium* carriers and frequent travel to the endemic countries¹⁸.

The larval stage of the pork tapeworm, *T. solium*, causes the clinical syndrome of cysticercosis, after ingestion of *T. solium* eggs. Man is the dead-end host. On ingestion the oncospheres are released from the eggs in the human intestine. These larvae invade the intestinal mucosa and are carried to different tissues through circulation, where they develop into cysts. Its clinical effects vary depending on site of larval lodging, larval burden, and host reaction.²⁰ These effects include seizures, headaches, focal neurologic symptoms, visual disturbances, and localized skeletal muscle nodules and pain. Ophthalmic cysticercosis (intraocular) manifests symptoms like proptosis, diplopia, and loss of vision while extraocular cyst resembles slow growing tumour or nodule with focal inflammation¹⁹.

India) and sub- Himalayan parts of north India including Uttar Pradesh, Himachal Pradesh and Jammu and Kashmir²³.

Leishmana parasite is found in two morphologic forms, the amastigotes and the promastigotes. The amastigotes are small, 3 um to 5 um in diameter, ovoid, nonmotile, intracellular forms, whereas the promastigotes are elongated, motile and extracellular stages. The promastigotes are engulfed by reticuloendothelial cells, and the parasite transforms into the intracellular amastigote form, within 12 to 24 hours. The symptoms of leishmaniasis are skin sores which erupt weeks to months after the person affected is bitten by sand flies. Other consequences include fever, hepatosplenomegaly, hypergammaglobulinemia, weight loss and anaemia. The hypopigmentation of skin seen in Indian patients giving the name Kala-Azar (black fever).

Trypanosomiasis:

Human trypanosomiasis is endemic in Africa and South America. In Africa, the disease, known sleeping sickness, is caused by *Trypanosoma brucei gambiense* (chronic form) or *T. b. rhodesiense* (acute form), whereas the American trypanosomiasis, known as Chagas' disease, is caused by *T. cruzi*. Human African trypanosomiasis is transmitted during blood meals by infected saliva of tsetse flies of genus *Glossina*, while the American trypanosomiasis is transmitted by infected feces of reduviid bugs. However, blood transfusions, shared needles, and the congenital route can also transmit these parasites. In 1999, only 37,000 cases of human African trypanosomiasis were reported to WHO. By 2003, 300,000 to 500,000 new cases had occurred. The problem is greater than these figures indicate, because untreated disease results in 100% mortality. Trypanosomiasis are usually confined to animals in other geographic locations. *T. evansi* is normally found only in animals but there are reports of human infection including India^{24,25}. Reports from India and from Malaysia have identified trypanosomes similar to *T. lewisi* in peripheral blood of patients with short febrile episodes. This trypanosome, which was first identified, in 1881, from Punjab, India, in the horse and camel usually causes a disease called surra in bovines and camel. Although cases of human carriers of animal trypanosomiasis were recorded during that century in India, Sri Lanka and Malaysia, these have either never been formally demonstrated or were only very short-lasting infections²⁴.

After ingestion by the fly, the trypomastigote forms transform to procyclic trypomastigotes. In the salivary glands, they become epimastigotes. The epimastigotes mature into metacyclic trypomastigotes, which are the infective form for humans. The metacyclic trypomastigotes are injected into the skin when the tsetse fly takes a blood meal. The parasites move to the lymphatics and eventually reach the blood. They can reproduce in various tissues and fluids, including cerebrospinal fluid, blood, and lymph. The African trypanosomes have evolved a defense mechanism in which they change their surface coats every 1 to 2 weeks, thereby evading the host antibody response⁶. The concentration of parasites in the blood is highest during febrile episodes. Clinical symptoms in case of African trypanosomiasis are, Chancre, intermittent fever, malaise, myalgia, tachycardia, lymphadenopathy, severe headache, Sleep/speech disorders, seizures, muscle rigidity meningoencephalitis, stupor, coma. The American trypanosomiasis causes Chagoma, malaise, fever, Romana's sign, megaesophagus, megacolon, myocarditis, and meningoencephalitis. For both forms of disease, the presence of trypomastigotes in blood, lymph, tissue aspirates, or CSF is diagnostic^{6,7}.

Table 3: Emerging and reemerging zoonotic parasitic diseases.

| Disease/Name/Type of Parasite | Mode of Infection | Clinical Symptoms | Diagnostic Methods | Treatment |
|---|--|---|--|--|
| Babesiosis: <i>B. microti, B. divergens</i> | Ixodid tick | Cell lysis, Anemia, hyperbilirubinuria, hemoglobinuria | Thick or thin blood smears "Maltese cross formations" on the blood film. Serologic method: IFA. Animal inoculation and PCR | Combination of Clindamycin and oral quinolone |
| Echinococcosis: <i>Echinococcus granulosus, E. multilocularis</i> <i>E. vogeli</i> <i>E. oligarthrus</i> (Cestode) | Faeco-oral route by Ingestion of eggs in contaminated food or vegetables or water. | Pain in the upper abdominal region, hepatomegaly, cholestasis, biliary cirrhosis, portal hypertension, ascites fever, anaemia, weight loss, and pleural pain. Chronic cough, expectoration, dyspnea, hemoptysis, pleuritis Neurological symptoms | Imaging techniques, ultrasonography, CT Scan, X-ray Immunodiagnostic tests: ELISA Aspirated cyst fluid microscopy for hooklets and scolices | Puncture aspiration injection respiration (PAIR) and chemotherapy with Ibandazole or Mebendazole |
| Trichinellosis <i>Trichinella trichinella</i> <i>T. nelsoni</i> (Nematode) | Ingestion of larvae encysted raw or undercooked meat | Occasionally - Cardio-neurological syndrome, nephritis, glomerulonephritis, and pneumonitis. | History of eating raw or undercooked pork Hyper eosinophilia i.e. >4000/granulocytes/mm ³ Serological assays: Latex agglutination, bentonite flocculation test, ELISA | Thiabendazole or Mebendazole Along with Corticosteroids |
| Angiostrongyliasis <i>Angiostrongylus cantonensis</i> (Nematode) | Ingestion of raw or undercooked snails or slugs, paratenic hosts such as prawns, fresh water shrimp or contaminated vegetables/water | Eosinophilic meningo-encephalitis Severe headache, neck rigidity, vomiting, altered sensorium and with or without low grade fever | CSF pleocytosis Eosinophilia (10%-50%) Serological tests: IFA, IFA and ELISA | Self limiting |
| Gnathostomiasis: <i>Gnathostoma spinigerum</i> (Nematode) | Eating uncooked meat of fish, shrimp, crab, crayfish, frog, or chicken infected with the third stage larva | Cause visceral larva migrans. Migrating larvae provoke Inflammatory reaction mainly in the upper extremities, shoulder and neck | Demonstration of larvae in the surgical specimens ELISA- L3 IgG1 antibody for screening and L3 IgG2 antibody for confirmation. | Surgical removal of the lesions |
| Baylisascariasis: <i>Baylisascaris procyonis</i> <i>B. transfuga</i> (Cestode) | Faeco-oral route by ingestion of eggs in faeces of infected raccoons | Nausea, tiredness, liver enlargement, loss of coordination, lack of attention to people and surroundings, loss of muscle control, coma, blindness etc. | Demonstration of anti- <i>B. procyonis</i> antibodies in serum and CSF | Prognosis of <i>NLM</i> is grave Anthelmintics Not effective against larvae in human/intermediate host, but can eradicate adult worms from raccoons. |

ZOONOTIC PARASITIC DISEASES

Most emerging infectious diseases are zoonotic; wildlife constitutes a large and often unknown reservoir. Wildlife can also be a source for reemergence of previously controlled zoonoses (Table 3).

Babesiosis

Babesiosis is an emerging zoonotic, ixodid tick transmitted disease caused by the hematotropic parasites of the genus *Babesia*²⁶. Transmission can also occur by transfusion of infected blood products and transplacentally. Mainly cases of human Babesial infections have been reported from temperate regions, Europe, France and England. However, nowadays cases of Babesiosis have been reported from other parts of the world including Asian countries like India, china and Taiwan. In North America, where most of the clinical cases have occurred the common pathogen is *B. microti* and the disease is transmitted by the bite of *Ixodes scapularis*. In European countries babesiosis is considerably rare but more lethal and common pathogen is *B. divergens*. The spectrum of disease range from mild infection to fulminant malaria like infection leading to death^{26,27}. The infection of erythrocytes results in cell lysis leading to anemia, hyperbilirubinuria, hemoglobinuria and other disease manifestations. The spectrum of disease range from mild infection to fulminant malaria like infection leading to death²⁶.

The life cycle of *Babesia* is similar to that of *Plasmodium*, but with several significant differences. There is no extraerythrocytic stage of the life cycle in the mammalian host. The parasite directly enters the erythrocytes and multiply resulting into rupturing of erythrocytes and asexual reproduction occurs by budding instead of schizogony^{6,7}. Careful examination of multiple blood smears may be necessary, since *Babesia* may infect less than 1% of circulating red blood cells and

thus be easily overlooked.

Echinococcosis

Echinococcosis in humans is a zoonotic infection caused by larval stages of a cestode parasite of genus *Echinococcus*. Man is an accidental host where the parasite reaches its dead end. Cystic echinococcosis (CE) is caused by *E. granulosus*, alveolar echinococcosis (AE) is caused by *E. multilocularis*, and polycystic forms are caused by either *E. vogeli* or *E. oligarthrus*. AE, a life-threatening infection of humans, is caused by proliferative larval stage of the small fox tapeworm, which has a wide distribution in the North America and northern and central Eurasia. Prevalence of human AE has increased from only four countries to seven countries²⁸. *E. granulosus* a dog tapeworm, is known to occur on all the continents. Reports from several countries provide documented evidence for the emergence or reemergence of AE and CE in recent years²⁸. High Prevalence of *Echinococcus* in Wild and Domestic Animals is associated with disease incidence in humans. Pastoralism is the occupation with the highest risk of being infected with the both kinds of echinococcosis due to the proximity of livestock, dogs, and wildlife host species.

Hydatid cyst can cause pain in the upper abdominal region, hepatomegaly, cholestasis, biliary cirrhosis, portal hypertension, ascites fever, anaemia, weight loss, and pleural pain²⁹. Cysts may rupture into the peritoneal cavity, causing anaphylaxis or secondary CE, or into the biliary tree, leading to cholangitis and cholestasis. Abscess formation is possible after bacterial infection of cysts. Chronic cough, expectoration, dyspnea, hemoptysis, pleuritis, and lung abscess are selected symptoms caused by pulmonary cysts, and cysts in the brain can induce neurological disorders.

Approximately 40 to 80% of patients with primary CE have single-organ involvement and harbor a solitary cyst. The initial phase of the primary infection is always asymptomatic. Clinical signs may occur after incubation period of several months or years. CE occurs in age groups from younger than 1 to over 75 years. The Mortality rates in untreated or inadequately treated AE patients can be very high²⁸.

Trichinellosis

One of the longest-known nematode zoonoses is trichinellosis. In Europe, trichinellosis has been described as an emerging and/or re-emerging disease during the past decades. In 2008, morbidity due to trichinellosis doubled in Russia. According to a report of the European Food Safety Authority, trichinellosis caused 779 human cases in EU Member States during 2007³⁰. Humans typically acquire the infection by eating raw or inadequately cooked meat contaminated with infectious larvae. *Trichinella* spp. are cosmopolitan, while some species are restricted to some countries; eg, *T. nelsoni* is limited to Southeast Africa. The most common sources of human infection are pig meat. The diaphragm, tongue, masseter and intercostal muscles are among those most heavily involved in pigs. Horse, dog and many other animal meats have also transmitted the infection. Man is the accidental host and dead end of parasite. Infection occurs by ingestion of larvae encysted in muscle. The young larvae (measuring about 0.1 mm) migrate into the lymphatics and are carried via the portal system to the peripheral circulation, and reach striated muscle where they penetrate individual muscle cells. In humans, heavy infections may produce serious illness with three clinical phases (intestinal, muscle invasion and convalescent). In more severe cases, difficulties with coordinating movements as well as heart and breathing problems may occur. Cardio-neurological syndrome, nephritis, glomerulonephritis and pneumonitis are the occasional life threatening manifestations. A small proportion of cases die from trichinellosis infection³⁰.

Angiostrongyliasis

Human angiostrongyliasis caused by *Angiostrongylus cantonensis*, a rat lungworm, which causes eosinophilic meningitis in man, has been reported globally. During the past few years, several outbreaks of human angiostrongyliasis have been reported in mainland China, Taiwan, Vietnam, Malaysia, Indonesia, Japan and the USA. Few cases have been reported from Bombay, India also. Additionally, sporadic cases in travellers who have returned from endemic areas have been reported. *A. cantonensis* was first described as a parasite of the Norway rat (*Rattus norvegicus*) and the black rat (*R. rattus*) in Guangzhou (formerly Canton), People's Republic of China, in 1933. During the past decade, the number of cases has sharply increased. A large outbreak occurred in Beijing during 2006³¹.

Rat is the natural host and man is an accidental host. Terrestrial snails and slugs are the intermediate host. Human infections are acquired by ingestion of raw or undercooked snails or slugs, paratenic hosts such as prawns, fresh water shrimp or contaminated vegetables and water that contain the infective larvae of the worm. The larvae are carried to CNS where they undergo one or two additional moults but without any further development. In the central nervous system, the larvae provoke an eosinophilic meningo-encephalitis and foreign body granulomatous reaction around the dead worm. The clinical symptoms are typical of eosinophilic meningo-encephalitis³². Severe headache, neck rigidity, vomiting, altered sensorium and with or without low-grade fever. In cases with a heavy load of parasites the infection can be so severe and lead to permanent damage to the CNS or death.

Gnathostomiasis

Gnathostomiasis a zoonotic infection is caused by a helminth parasite, *Gnathostoma spinigerum*. It is being seen with increasing frequency in countries where it is not endemic and should be regarded as another emerging imported disease. It is acquired by eating uncooked meat of fish, shrimp, crab, crayfish, frog, or chicken infected with the third stage larva. It is a parasite of wild carnivores, cats and dogs. Previously, most of these cases were reported from Southeast Asia, particularly Thailand and Japan, because of the dietary habits, however, in recent years, it is an increasing problem in Central and South America. *G. spinigerum* and other species are also found in India, Pakistan, the Middle East, Mexico, Southeast Asia, Japan, and Australia. In India cases have been reported from different parts. The first case of ocular gnathostomiasis was reported from India in 1945. Cats and dogs, serve as important reservoirs of infection in regions where *Gnathostoma* is endemic^{7,33}.

Man is an accidental host. Eggs are liberated in the stools of definitive host and are hatched in fresh water as first stage larvae. These larvae are ingested by Cyclops, in which second stage larvae develop. Fresh water fish, frogs, snakes, etc. ingest infected Cyclops, which act as intermediate hosts. Eating the raw or undercooked meat of such intermediate or paratenic hosts can infect man. The third stage-larva cause visceral larva migrans in man. The larva cannot mature into the adult form in humans, the third-stage larva can only wander within the body of the host, where they may remain alive up to 10 years. Clinical symptoms of gnathostomiasis then occur because of the inflammatory reaction provoked by these migrating larvae. These appear commonly in the upper extremities, shoulder, neck and other parts of the body. The immature worms may be found in any part of the body such as mouth, Eye, pharynx, respiratory tract, urinary bladder, intestine or anus^{33,34}.

Baylisascariasis

Baylisascaris procyonis is an intestinal helminthic parasite of raccoons. It causes a very serious zoonotic disease in humans. Small mammals are the intermediate host. *B. transfuga* infects bears. Infection is caused by accidental ingestion of an egg. The worm can provoke retinitis and eosinophilic meningo-encephalitis (visceral larva migrans). Neurological symptoms are prominent. Eosinophilic pseudotumours in the heart have also been described. The prevalence of *B. procyonis* infection is high in wild raccoons in Germany and those kept in zoos or as pets in Japan. Raccoons were formerly introduced into Europe (France, Germany, and The Netherlands), the Soviet Union, and Asia for the commercial fur trade and into Japanese pets³⁵.

Human infections result from ingestion of eggs that are passed in very large numbers (millions of eggs/day) in the feces of infected raccoons. Once ingested, the eggs hatch in the intestinal tract, releasing the immature larvae. However, rather than developing into adult worms as in the raccoon, the larvae begin to migrate extensively throughout the body, causing visceral larva migraines or neural larva migraines (NLM) or ocular larva migraines.

Once inside the body, eggs hatch into larvae and cause disease when they travel through the liver, brain, spinal cord, or other organs. General symptoms of such infection can resemble those of meningoencephalitis³⁵. Symptoms include, nausea, tiredness, liver enlargement, loss of coordination, lack of attention to people and surroundings, loss of muscle control, coma, and blindness. The prognosis for *B. procyonis* NLM is grave.

TRANSFUSION ASSOCIATED PARASITIC DISEASES

Despite of stringent donor eligibility criteria, improved donor screening and introduction of sophisticated technology, the risk of transmission of various emerging and reemerging infections cannot be checked.

Malaria is probably the most commonly recognized parasitic complication of transfusion. Malarial parasites survive for at least a week in components stored at room temperature or at 4°C. Parasitaemia of as low as 10/μL can give rise to transfusion-transmitted malaria. *Babesia* can survive at 4°C in a unit of RBCs for up to 35 days and can also be transmitted through infected packed red cells, frozen-thawed-deglycerolized red blood cells and platelet concentrates. Trypanosomal parasite can remain viable for at least 21 days in the whole blood and RBC units that have been stored at 4°C³⁶.

Toxoplasma can also survive for several weeks in stored whole blood. Although microfilaria can be transmitted when the blood from a microfilaric individual is transfused and, the transfused microfilaria may persist in the recipient's circulation for more than 2 years, but transfused microfilariae do not develop into adult filarial worms.

SUMMARY

These infections will continue to emerge and reemerge leading to unpredictable epidemics and challenges for the medical public health care personnel/ scientists. The specific factors such as ecological, environmental, or demographic will keep precipitating the problem of putting people at increased contact with a previously unfamiliar microbe or its natural host or promote dissemination. These increase in prevalence of these factors together with the ongoing evolution of microbial variants and selection for drug resistance, suggests that

infections will continue to emerge and probably increase and emphasizes the urgent need for effective surveillance and control. Same time there is an urgent need to develop newer advanced surveillance tools, diagnostic tests, vaccines and therapeutics. Diagnostics and therapeutics based on genomics, proteomics and nanotechnology should be explored. Gene and protein based microarrays should be made available for accurate and rapid detection of all types of emerging and reemerging pathogens.

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