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EXOTIC VIRAL INFECTIONS

The word exotic derived from the latin *exoticus*, means foreign, not indigenous but from outside. Viral infections which are not prevalent or are new for a particular area or country or in other words the infections newly identified or previously unknown are known as exotic viral infections. Exotic also suggests rarity, but infections may be rare or unknown in one region, yet quite common elsewhere in the world.

In India, though some viral infections like Japanese encephalitis, Dengue & Dengue Haemorrhagic fever, West Nile fever, Kyasanur Forest Disease, Poliomyelitis, Rabies etc. are prevalent while other infections like Yellow Fever, Crimean Congo Haemorrhagic Fever (CCHF), Hantaan virus fever, Ebola virus fever, Lassa fever are exotic. Some of these exotic infections are constant threat to our country as the vectors and/or reservoirs for transmission of these pathogens and the favourable environmental conditions for their culmination exist here for example prevalence of mosquitoes (*Aedes aegypti*)- the vector for yellow fever, tick vector for CCHF, rodent reservoir for transmission of Hantaan virus. Fortunately the pathogens for these infections have not entered the boundry of our country.

In recent years undiagnosed encephalitis outbreaks simulating Nipah virus encephalitis have been reported in India. It is, therefore, of utmost importance that a constant vigil of these exotic viral infections be maintained so that in the event of their emergence, immediate containment measures can be taken. Some of these exotic infections which may pose threat to our country are dealt herewith for creating awareness among health professionals about their epidemiology, clinical features, diagnosis, treatment, prevention and control.

YELLOW FEVER

Yellow fever is a viral disease that is endemic

in tropical regions of America and Africa and is transmitted by mosquitoes from monkey to man, subsequently from man to man. This disease has not yet been reported from Asia. The 'yellow' as the name suggests, indicates, jaundice that affects some patients.

Causative agent

The disease is caused by yellow fever virus, which belongs to the genus flavivirus and family flaviviridae.

Epidemiology

Geographical distribution: The virus is constantly present with low levels of infection (i.e. endemic) in some tropical areas of Africa and the Americas. This viral presence can lead to regular epidemics. Thirty-three countries with a combined population of 508 million, are at risk in Africa. These lie within a band from 15°N to 10°S of the equator. In America, yellow fever is endemic in nine South American countries and several Caribbean islands. Bolivia, Brazil, Colombia, Ecuador and Peru are considered at greater risk.

There are 200,000 estimated cases of yellow fever per year. However, due to under reporting, only a small percentage of these cases are identified. Although yellow fever has never been reported from Asia, this region is at risk because the appropriate primates and mosquitoes responsible for transmission of this virus are present here.

Transmission: Human and monkeys are the principal hosts of this infection. The virus is carried from one animal to other (horizontal transmission) through mosquito bites (the vector). Several different species of the *Aedes* and *Haemogogus* (S. America only) mosquitoes transmit the yellow fever virus.

Symptoms

The incubation period is from three to six days. The disease occurs in two phases. The first acute phase is normally characterized by fever, muscle pain, headache, shivers, loss of appetite, nausea and vomiting. After 3 to 4 days most patients improve and their symptoms disappear.

However, 15% enter a “toxic phase” within 24 hours. Fever reappears and several body systems are affected. The patient rapidly develops jaundice and complains of abdominal pain with vomiting. Bleeding can occur from mouth, nose, eyes and/or stomach. Kidney function deteriorates and leads to complete renal failure. Half of the patients in the “toxic phase” die within 10-14 days. The remainder recover without significant organ damage.

Laboratory diagnosis

Virus isolation and serology are used in the laboratory diagnosis of yellow fever. For virus isolation, blood must be obtained within the first 3-4 days of infection. Virus can be isolated from necropsy specimen from liver until 10-12 days after onset.

Serological tests: The haemagglutination inhibition (HI) Test, neutralisation test and ELISA are used to detect antibodies against yellow fever virus. The neutralisation test is the most specific. Antibodies against yellow fever infection appear early (during the first week after onset) and last for many years (probably life long).

Treatment

There is no specific treatment for yellow fever. Dehydration and fever can be corrected with oral rehydration salts and antipyretics. Any superimposed bacterial infection should be treated with appropriate antibiotic. Intensive supportive therapy with intravenous fluid, blood, plasma or plasma substitute may improve the outcome of seriously ill patient.

Surveillance, Prevention and Control

Surveillance: Surveillance should be based on clinical and serological monitoring of the human population and whenever possible should include surveys in mosquitoes and monkeys.

For clinical surveillance in man the broadest possible case definition should be used, i.e., febrile illness with jaundice or more specific case definition which helps to focus on yellow fever

may be substituted; for example, jaundice with albuminuria or jaundice with haemorrhagic manifestations. Although, in all the cases infection with yellow fever virus does not result in jaundice; however, this feature of the disease is useful as a basis for surveillance since the other clinical signs are non-specific. Most countries have a system for reporting cases of hepatitis which can be adapted to provide information about the occurrence of yellow fever. The monthly incidence of cases and deaths from hepatitis should be examined by geographical region or administrative area.

Mosquito surveillance, although often very informative as regards enzootic or epizootic circulation of yellow fever virus among monkeys in the forest, is feasible only on rare occasions. However, surveillance of *Aedes aegypti* must be carried out in communities at risk or in cities in the endemic zone, and control measures must be instituted when indicated by the mosquito density (house index, container index, or Breteau index). The susceptibility of *Aedes aegypti*, and other possible potential vectors, to the principal insecticides that may be required for their control should be determined regularly.

Prevention: The 17D yellow fever vaccine has been used for almost 50 years for the prevention of yellow fever. This is a live attenuated vaccine. The vaccine is administered subcutaneously in 0.5 ml dose to adults and children older than 6 months. Immunity begins to appear on the 7th day. **Under the current IHR, all persons (except infants upto the age of 6 months) coming to India from yellow fever endemic countries who are not able to produce a valid certificate of vaccination against yellow fever are isolated until the certificate becomes valid, or until a period of not more than six days from the date of last possible exposure to infection has elapsed, whichever occurs first. The certificate of vaccination against yellow fever is valid for a period of 10 years beginning 10 days after the date of vaccination or in the case of a revaccination within such period of 10 years from the date of that revaccination.** Countries where *Aedes aegypti* exist may stipulate a quarantine period of 6 days for non-immunized persons coming from endemic countries.

Control: The recognition of an increased incidence of fatal hepatitis (from hospital reports), the diagnosis of even a single case

(by histopathology, serology or virus isolation), and any reports of suspected cases of yellow fever require immediate epidemiological investigation to determine whether the virus is active in human, mosquito or monkey populations. Investigation in human population is based on clinical diagnosis of suspected cases and laboratory confirmation by the isolation of the virus and serological tests to detect the presence of antigen or antibodies. Serological detection of specific IgM antibodies provides the quickest proof of recent infection. Since immunity appears after 7 days following immunization, mosquito control is necessary to limit the transmission of the virus during this period. Therefore, an entomological investigation is needed to determine the vector species involved, its biological characteristics, its particular mode of contact with the human population, its breeding sites, and sensitivity to insecticides. The later must be determined in order to provide effective control.

CRIMEAN CONGO HAEMORRHAGE FEVER

Crimean Congo Haemorrhagic Fever (CCHF) is a viral haemorrhagic fever of the Nairovirus group. Although primarily a disease of animals, sporadic cases and outbreaks of CCHF affecting humans do occur. The disease was first described in the Crimea in 1944 and given the name Crimean Haemorrhagic Fever. In 1969 it was recognised that the pathogen causing Crimean Haemorrhagic Fever was the same as that responsible for an illness in 1956 in the Congo and the linkage of the 2 place names resulted in the current name for the disease.

Etiological agent

The virus which cause CCHF is a Nairovirus, a group of related virus forming one of the five genera in the Bunyaviridae family of viruses.

Epidemiology

Geographical distribution: The geographical distribution of the virus is widespread like that of its tick vector. The disease occurs over a large area of the world. It has been reported from Afghanistan, Bulgaria, China, Hungary, Iraq, Iran, Arab, UAE, South West Russia and Yugoslavia. It is widespread in East and West Africa. During 2001, sporadic cases and

outbreaks have been recorded in Kosovo, Albania, Iran, Pakistan and South Africa.

Vectors: A number of tick genera are capable of becoming infected with CCHF virus, but the most efficient and common vector for CCHF appears to be member of Hyalomma Tick genus. Transovarial and venereal transmission has been demonstrated amongst some vector species, indicating one mechanism which may contribute to maintaining the circulation of virus in nature.

Vertebrate hosts: Wide range of domestic and wild animals are infected by CCHF virus from the bite of infected ticks. It is possible that domestic animals (sheep, goats and cattle) act as amplifying hosts during epizootic season.

Human infection: Human becomes infected with CCHF virus from direct contact with blood or other infected tissues from livestock or may become infected from a tick bite. High risk group comprises of agricultural workers, slaughter house workers, veterinarians, campers and the military.

Incubation period

The length of incubation period for the illness appears to depend on the mode of acquisition of the virus. Following contact with infected blood or tissues, it is 5-6 days (maximum 13 days) and following tick bite it is 1-3 days (maximum 9 days).

Clinical features

Onset of symptoms is sudden with fever, myalgia, neck pain and stiffness, photophobia, nausea and vomiting which may be accompanied by diarrhoea and generalised abdominal pain. Patients may experience sharp mood swing. After two to four days abdominal pain may localise to the right upper quadrant with detectable hepatomegaly. Other clinical signs include tachycardia, lymphadenopathy, petechial rash on skin and mucosal surfaces and haemorrhagic phenomena such as melaena, haematuria, epistaxis. There is usually evidence of hepatitis. The severely ill patients may develop hepatorenal and pulmonary failure after fifth day of illness. The mortality rate from CCHF is approximately 30%. Improvement generally begins on the ninth or tenth day after onset of illness in recovered cases.

Laboratory diagnosis

Laboratory diagnosis of suspected CCHF should be carried out in specially high biosafety level equipped laboratories. Specific IgG and IgM antibodies may be detected in serum by ELISA from about day six of illness. Patient with fatal disease do not usually develop a detectable antibody response. So in these individuals, as well in patients, within the first few days of illness, diagnosis is possible by virus isolation in cell line from blood or tissue samples. Recently PCR test for detecting viral genome, has been successfully applied in diagnosis.

Treatment

General supportive therapy is the mainstay of patient management in CCHF. Intensive monitoring for volume and blood component replacement is required. The antiviral drug ribavirin has shown apparent benefit when used in treatment of established CCHF infection cases.

Prevention and control

Although an inactivated, mouse brain derived vaccine against CCHF has been developed and used in a small scale in Eastern Europe still there is no safe and effective vaccine widely available for human use. Tick control with acaricides is only a realistic option for well managed livestock production facilities. Persons living in endemic areas who work with livestock and/or other animals in the endemic areas should use personal protective measures. Patients with suspected or confirmed CCHF should be treated under isolation using barrier-nursing techniques. Specimens of blood or tissues taken for diagnostic purposes should be collected and handled using universal biosafety precautions.

EBOLA HAEMORRHAGIC FEVER

Ebola Haemorrhagic Fever (EHF) is one of the most virulent viral disease known to human kind, causing death in 50-90% of all clinically ill cases. The disease has its origin in the jungles of Africa and Asia.

Etiological Agent

The disease is caused by Ebola virus, a RNA virus which belongs to family of Filoviridae.

There are four identified subtypes of Ebola virus, three of which cause disease in humans viz. Ebola-Zaire, Ebola-Sudan, and Ebola-Ivory Coast. The fourth Ebola-Reston has caused disease in non-human primates.

Epidemiology

Geographical distribution: Ebola virus was first identified in western equatorial province of Sudan in 1976. Confirmed cases of EHF have been reported in the Democratic Republic of the Congo (Zaire), Gabon, Sudan, the Ivory Coast, Uganda and the Republic of the Congo.

Transmission: The Ebola virus is transmitted by direct contact with blood, secretions, organs or semen of infected persons. Transmission through semen may occur up to seven weeks after clinical recovery. People can also get exposed to Ebola virus through contact with objects, such as needles, that have been contaminated with infected secretions. Transmission of the Ebola virus has occurred by handling ill or dead infected chimpanzees. Health care workers have frequently been infected while attending patients.

Natural reservoir: The natural reservoir of the Ebola virus seems to reside in the rain forests of Africa and Asia which has not yet been identified. Laboratory observations have shown that bats infected experimentally with Ebola virus did not die which raised speculation that these mammals may play a role in maintaining the virus in the tropical forest.

Incubation period

It is 2 to 21 days, usually around 7 days. Incubation period of the disease following needle exposure to Ebola virus is 6 days with range of 1-8 days.

Clinical features

Onset of Ebola virus fever is abrupt with shivering and a rapid rise in temperature accompanied by severe headache, backache, muscle pain and sore throat. This is followed by vomiting, profuse diarrhoea, and erythematous maculopapular rash on trunk of the body. Derrangement of liver and kidney functions takes place and about half of the patients present with bleeding, both internally and externally. Fatality rate is very high (50-90%).

Laboratory diagnosis

As the disease is highly infectious the laboratory diagnosis of the disease should be conducted under maximum biological containment conditions i.e. in BSL-4 lab. The diagnostic tests include (i) virus isolation in cell culture (ii) serological tests to detect specific IgM and IgG antibodies.

Treatment

No specific treatment or vaccine exist for Ebola fever. Severe cases require intensive supportive care, as patients are frequently dehydrated and in need of intravenous fluid.

Prevention and Control

Suspected cases should be isolated from other patients and strict barrier nursing techniques practiced. All hospital personnel should be briefed on the nature of the disease and its routes of transmission. Hospital staff should have individual gowns, gloves and masks. Any person who has had close physical contact with patients should be kept under strict surveillance, for three weeks after their last contact. Casual contacts should be placed on alert and asked to report in the event of any fever. Hospital personnel who come into close contact with patients or contaminated materials without barrier or nursing attire must be considered exposed and put under close supervised surveillance.

LASSA FEVER

Lassa fever is an acute viral illness that occurs in West Africa. The disease was first described in the 1950s, although the virus was not isolated until 1969. The disease was named after the town in Nigeria where the first case originated.

Etiological agent

Lassa fever is caused by Lassa virus which is a single stranded RNA virus and belongs to family Arenaviridae.

Epidemiology

Geographical distribution: Lassa fever is an endemic disease in parts of West Africa. It is recognised in Guinea, Liberia, Sierra Leone and regions of Nigeria.

Transmission: It is transmitted to humans from

wild rodents (*Mastomys natalensis*). In rodents virus is shed throughout the life following infection. Disease transmission is primarily through direct or indirect contact with excreta of infected rodents. Person-to-person and laboratory infections occur especially in hospital environment by direct contact with blood, pharyngeal secretions or urine of a patient, or by sexual contact. The spread may occur during the acute phase of the illness when the virus is present in the throat.

Incubation period

It is 6-21 days. All age groups are susceptible to Lassa infection.

Clinical features

The onset of the disease is gradual with fever, malaise, headache, sore throat, cough, nausea, vomiting, diarrhoea, myalgia, chest and abdominal pain. In severe cases hypotension or shock, pleural effusion, haemorrhage, seizures, encephalopathy and swelling of the face & neck are frequent. Approximately 15% of hospitalized patients die.

Laboratory diagnosis

As in Ebola fever, the laboratory diagnosis of Lassa virus infection should also be conducted under maximum biological containment conditions (BSL-4 lab). It is mostly diagnosed by serological assays like ELISA, which detect specific IgM and IgG antibodies as well Lassa antigen.

Primary isolation of Lassa virus can be successfully performed in the continuous cell line, Vero E6 in a high biosecurity laboratory. The virus can also be detected by RT-PCR.

Treatment

Treatment with the anti-viral drug, ribavirin may be effective if given within first six days of the illness. Ribavirin should be given intravenously for ten days along with other supportive care, which includes maintenance of fluid and electrolyte balance, oxygenation and blood pressure.

Prevention and Control

Isolation of cases: Strict barrier isolation of cases in a hospital room and strict compliance

of biosafety procedures for handling of body fluids and excreta should be maintained.

Disinfection: Patient's excreta, sputum, blood and all objects with which the patient has had contact, including laboratory equipment used to carry out tests on blood, should be disinfected with 0.5% sodium hypochlorite solution or 0.5% phenol with detergent.

Surveillance of contacts: Close surveillance of contacts should be carried out for three weeks after the last exposure. Prophylaxis with ribavirin is recommended for close contacts. No vaccine is currently available.

Rodent control: The ideal method of prevention in endemic areas is to prevent contact between rodents and humans. People in endemic areas should restrict entry of rats into their dwelling, isolate food supplies from rodents, eliminate habitats for rats and minimize activities that produce aerosols containing rodent excreta.

RIFT VALLEY FEVER

Rift Valley Fever (RVF) is an acute fever causing viral disease that affects domestic animals (such as cattle, buffalo, sheep, goats and camels) and humans. RVF is most commonly associated with mosquito borne epidemics during years of unusually heavy rainfalls. The disease was first reported among livestock by a veterinary officer in Kenya in the early 1900s.

Causative agent

The disease is caused by the RVF virus, a member of genus *phlebovirus* in the family *Bunyaviridae*.

Epidemiology

Geographical distribution: RVF is generally found in regions of eastern and Southern Africa where sheep and cattle are raised. It is also reported from sub-Saharan Africa and Madagascar. Outside Africa RVF outbreaks have also been reported from Saudi Arabia and Yemen.

Transmission: The virus is transmitted during epizootics and epidemics by mosquitoes; it undergoes a short incubation period in the insect. The virus may also be transmitted to man by contact with aborted fetuses of sheep and cattle or with blood and other tissues of infected

sheep and cattle during slaughter. High risk group include shepherds, veterinarians and butchers.

Vectors: At least 26 species of mosquitoes have been implicated as potential vectors, of these *Culex theileri* and *Aedes caballus* are considered to be major epizootic vectors.

Vertebrate hosts: Sheep and cattle serve as vertebrate amplifying hosts during epizootic

Clinical features

RVF virus can cause several different disease syndromes. People with RVF typically have either no symptoms or a mild illness associated with fever and liver abnormalities. Patients complain of generalized weakness, back-pain, and dizziness. Fever lasts for 2-7 days. However, in some patients the illness can progress to haemorrhagic fever (which can lead to shock and haemorrhage), encephalitis or ocular disease (retinitis with blindness). Blindness is usually not permanent. Case fatality rate of RVF is about 1%.

Laboratory diagnosis

Early and rapid diagnosis can be made by either detecting antigen in serum during first few days of illness (as viraemia ceases after day 4) or by detecting specific antibodies in serum. Antigen detection can be done by agar-gel precipitation test or by ELISA. Antibody detection can be carried out by IgM Capture ELISA.

Treatment

There is no specific treatment for RVF. It is not necessary to isolate the patient. General supportive treatment is indicated including blood, plasma and electrolyte replacement if necessary, as well as good nursing care.

Surveillance, Prevention and Control

Surveillance for Rift Valley Fever involves

- Detection of sero conversion in sheep, cattle or man
- Excess abortion in livestock with demonstration of virus or antigen in the fetus
- Case finding in persons living in or coming from enzootic zones

For prevention of the disease a live attenuated

vaccine and an inactivated tissue culture vaccine is available for veterinary use. An inactivated tissue culture vaccine for human use has also been produced for the immunization of high-risk personnel such as laboratory staff, veterinarians and the military.

Other control measures recommended are:

- Ultra low volume spraying of malathion, which rapidly kills mosquitoes
- Avoidance of slaughter of sick animals

HANTAAN VIRUS FEVER

Hantaan virus fever, also known as Haemorrhagic Fever with renal syndrome is not a new disease as description of this disease is mentioned in Chinese Medical book that was written about 960 A.D. Hantaan virus fever was first described in Tula Region of USSR in the 1930s and viral origin was proven by passage in human volunteers in Soviet Union in 1940.

Etiological agent

Hantaan virus and related viruses are spherical, RNA viruses, 85-110 nm in diameter, belong to the family Bunyaviridae.

Epidemiology

Geographical distribution: Sero epidemiological surveys and documented case reports show that Hantaan virus is widely distributed through out much of the world. There are atleast two forms of disease: mild and severe. Severe form is common in Asian countries like Republic of Korea, Japan, China, Russia whereas in European countries it is the milder form and reported from Scandanavia, Eastern Europe, Belgium, France and Greece.

Transmission: Large quantities of virus are excreted in the saliva, urine and faeces of infected rodents *Apodemus agrarius*. Excretion persists in saliva and faeces for at least 1 month and in the urine for 12 months. Horizontal transmission of the virus among *Apodemus* mice has been demonstrated. There is no evidence of direct man to man transmission of the virus. Transmission of hantaan virus to humans is thought to be predominantly through respiratory tract infection contracted from aerosols of infectious virus in rodent urine, faeces and saliva.

Reservoir: The reservoir of hantaan virus in the rural endemic areas is *Apodemus agrarius* and in the urban areas *Rattus rattus* and *R.norvegicus*.

Vector: The identification of Hantaan virus as a member of the family Bunyaviridae suggests that it may be an arthropod-transmitted virus, but that role has not yet been confirmed. Until now, Hantaan virus has not been isolated from arthropods.

Group at risk: The disease affects most frequently persons in the age group of 20-25 years. The high-risk group comprises primarily of farmers and soldiers on duty at field stations, health personnel working in laboratories, animal room workers and rodent breeders.

Clinical manifestations

The incubation period of Hantaan virus fever is generally 2-3 weeks but may vary from 4 to 42 days. Its major manifestations are fever, headache, backache, abdominal pain, flushed face, prostration, vomiting, proteinuria and haemorrhagic phenomenon. 30% patients show a mild clinical course without haemorrhagic phenomena or proteinuria, about 50% exhibit a moderate course, while 20% have severe form of disease. Death from shock and renal failure occurs in 10-15% of cases.

Laboratory diagnosis

Diagnosis of Hantaan virus fever can be done by

- Serological test – By demonstrating rise in titre of specific immunofluorescent antibodies (IFA) and neutralizing antibodies against Hantaan virus in paired sera collected at an interval of one week. Besides this, ELISA and immuno-adherence passive haemagglutination (IAPH) tests have also been developed for its diagnosis.
- Virus isolation – Isolation of Hantaan and related viruses is possible by inoculation in *Apodemus* mice or Vero E6 cells with blood and serum taken during early stages of infection.

Treatment

The general supportive management of the patients with the Hantaan virus fever is the only

known treatment.

Prevention and control

The most important point for prevention and control of Hantaan virus fever is to reduce the exposure with virus by avoiding contact with rodents and their droppings, in the endemic area. This can be achieved by restricting entry of rats into the dwelling by keeping houses clean, isolating food supplies from rodents, eliminate habitats for rats and minimize activities that produce aerosol containing rodents excreta. Besides this the high-risk group mentioned above should take special personal protection against rodents and rodents excreta.

NIPAH VIRUS ENCEPHALITIS

This is a newly recognised zoonotic viral disease manifested primarily as encephalitis; it is named after the location in Malaysia where the first human isolate was confirmed in 1999.

Etiological agent

Nipah virus is a RNA virus that belongs to family Paramyxoviridae.

Epidemiology

Occurrence: Nipah virus affects swine in the pig farming provinces of Perak, Negeri Sembilan and Selangor in Malaysia and also is reported from Singapore.

Transmission: The disease is transmitted to humans through close contacts with pigs. There is no evidence of person-to-person transmission.

Reservoir: The natural reservoir of Nipah virus is still under investigation, but preliminary data suggest that bats of the genus *Pteropus* are the reservoirs for Nipah virus in Malaysia.

Incubation period:

It ranges from 4 to 18 days.

Clinical features

Illness with Nipah virus begins within 3-14 days with fever and headache. This is followed by drowsiness and disorientation characterized by mental confusion.

These signs and symptoms can progress to coma within 24-48 hours. During the outbreak of Nipah virus disease in 1998-99, about 40% of patients admitted in the hospital with serious nervous disease died from the illness.

Laboratory diagnosis

Laboratory diagnosis can be made by

- Serological test – detection of specific IgM and IgG antibodies by use of capture ELISA or serum neutralization test.
- Confirmatory diagnosis by virus isolation and/or detection of viral RNA by RT-PCR.

Treatment

The antiviral drug ribavirin has been shown to be effective against this virus *in vitro*. However, clinical usefulness of this drug is uncertain.

Prevention and control

Health education to inform the public about appropriate preventive measures viz. proper precaution by animal handlers such as use of protective clothing, boots, gloves, gowns, goggles and proper washing of hands. Besides this obligatory reporting of cases to health authorities, isolation of infected swine, slaughter and barrier or incineration of infected pigs and restriction of movement of pigs from infected farms to other areas, to be undertaken to control the disease.

...about CDAlert

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