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Review Article

Selected viral zoonoses in medical practice

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ABSTRACT

Introduction: Viral zoonoses develop only in living organisms, are characterized by a great variability that stems from an adaptation to their hosts and can be a serious problem with respect to medical pathology. This group also embraces prion disease or bovine spongiform encephalopathy (BSE), whose etiological factor is modified prion protein (infectious protein particle) deposited in the nerve cells.
Aim: This work was aimed at presenting basic data concerning zoonoses occurring in Poland, including their etiology and epidemiology, along with the methods applied to break the epidemiological chain.
Materials and methods: The material used in this work consisted of available medical literature, including the latest reports concerning this subject.
Results and discussion: Zoonotic infections are caused by infected, asymptomatic or cured animals. Infections may be transmitted via animal products or slaughter products (animal-derived foods), as well as various elements of that environment contaminated by excrement from sick animals. From an epidemiological perspective, diseases such as anthrax or tick-borne encephalitis which have been prevalent for a long time are still significant. Diseases which have appeared more recently, such as bovine spongiform encephalopathy (BSE), avian influenza (H5N1) and swine influenza (H1N1), have also been discussed. Medical procedures in the event of recognizing such a disease are presented, including the official rules of veterinary actions with respect to infected animals and the food derived from such animals.
Conclusions:

1. Viral zoonoses can be a serious threat to human health due to the significant pace of pathogen proliferation. Despite medical advances, these diseases remain a serious problem for both medical and veterinarian services.
2. Due to globalization, "new zoonoses" constitute a worldwide rather than a local problem. This is clearly testified to by the European Union (EU) Zoonoses Directive issued in 1994 by the Council of the European Union.
3. The application of vaccines does not prevent the development of all zoonoses. Knowledge concerning the transmission modes of these diseases is especially important, as is the cooperation of epidemiologists and specialists in epizootology.

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1. Introduction

Zoonoses are defined as diseases or infections transmitted naturally between animals and humans, whereas zoonotic agents include bacteria, viruses, fungi, and parasites that trigger zoonoses.[5] These diseases develop in specific social and economic conditions favorable to infectious mechanisms, especially in rural environments. The number of pet animals that develop latent zoonoses is increasing. In recent years, new zoonoses have been identified, since new forms develop every year.[23] One of the reasons for this situation is the ever closer contact of humans with an environment which serves as a reservoir for zoonoses and contains their agents and vectors. On the basis of their etiology, zoonoses are divided into infectious and parasitic diseases. Infectious zoonoses are caused by pathogenic agents which have adapted to human beings and to specific animal species. Viruses that develop only in living organisms pose a significant problem for medical pathology. Viruses survive in a natural environment due to their replication in host organisms and are characterized by their great variability stemming from their ability to adapt to their hosts. This is evidenced by such zoonoses as avian influenza (H5N1) and swine influenza (H1N1) both of which have recently become important sources for the worldwide outbreak of diseases. This group also embraces prion diseases or transmissible spongiform encephalopathies (TSE), whose etiological factor is modified prion protein (infectious protein particle). Prion proteins are deposited in the nerve cells as amyloid concretions and lead to spongiform changes. Nearly 90% of prion proteins are found in the brain and spinal cord. They are also found in a distal part of the intestine, retina, bone marrow, the sensory nerve cell bodies of dorsal root and trigeminal ganglia.[24] This disease has caused the slaughter of hundreds of thousands of cattle in Europe, as a result of which the economy was also affected.[14]

2. Aim

This work presents basic data concerning selected viral zoonoses occurring in Poland, including their etiology and epidemiology, along with the methods applied to break the epidemiological chain.

3. Materials and methods

The material used in this work consisted of available medical literature, including the latest reports concerning this subject which can further serve to initiate additional scientific reports.

4. Results and discussion

Important viral zoonoses occurring in Europe include rabies, tick-borne encephalitis, avian influenza, swine influenza, as well as the prion disease – bovine spongiform encephalopathy (BSE).

4.1. *Rabies*

Rabies is an acute viral disease that develops in homeothermic animals and humans. It is caused by a neurotropic virus belonging to the family *Rhabdoviridae*. The reservoirs for this virus are host animals, mainly foxes, badgers and bats. They transmit this disease to domestic animals: dogs, cats and cattle. Rodents, such as squirrels, rats, mice, and hamsters can also be rabies carriers; however, people have not been reported to have been infected by these animals. In Poland, approximately 2000 cases of rabies in animals are reported annually.[16] The highest incidence of rabies is detected in cats – 6.8% and dogs – 4.8%, and among wild animals in red foxes – 67.4%, raccoons – 6.5%, roe deer – 3.0%, martens – 2.4%, and badgers – 0.8%. According to the World Health Organization (WHO), 86–90% of rabies cases concerning humans are caused by virus transmission via dogs.[27]

In the natural environment, infection is nearly always caused by a bite, followed by the close contact of skin, muscles and mucous membrane with infected saliva. During the incubation period lasting from several weeks to months the virus replicates in myocytes, and then spreads 50–100 mm daily transferring to the spinal cord and brain. There it replicates, forming the so-called Negri bodies in the neurons, and then it penetrates into the salivary glands, taste buds, cornea, skin, and other organs via peripheral nerves. The virus is released from nerve endings 2–5 days prior to the appearance of clinical symptoms; however, it has been detected in the saliva of infected animals 14 days before the onset of the disease.[5,14,27] Factors such as young age, immune deficiency, corticosteroid treatment, bite severity, approximation of the wound with respect to the central nervous system (CNS), and high infectious dosage contribute to the shortened incubation period.[19]

In humans, the rabies incubation period ranges from 5 days to more than a year (2 months on average). Following the incubation period, hyperesthesia and pain at the wound site appear. Later, all other symptoms develop, such as psychomotor agitation, auditory and visual hallucinations, dysphagia with hydrophobia and sialosis. The last stage of the disease involves coma and paralysis, and death occurs after 1–10 days.[17] In liquid saliva the virus survives for up to 24 h, and in dry saliva for up to 14 h. It is resistant to putrefaction – its virulence in the medulla of a buried corpse was detected even after 14–20 days. At a temperature of 52°C the rabies virus dies within 30 min, and at a temperature of 80°C within 2 min. It is very susceptible to pH changes: in pH 3.0–3.5 environment it dies within 30 min, and in a pH 6.0 condition it loses approximately 90% of its virulence within 2 h.[17]

The last case of rabies in Poland was reported in 2002.[9] Individuals infected with the rabies virus are administered with a vaccine or serum. Infected animals are not treated because rabies is a disease controlled ex officio. It is compulsory to slaughter animals with symptoms of rabies. Carcasses and any other parts of such animals are considered unfit for consumption.[18,20] In Poland, the vaccination of dogs and cats against rabies is imposed by law. Foxes are vaccinated in selected regions by means of bait.

4.2. *Tick-borne encephalitis*

Tick-borne encephalitis is an egzogenic infection triggered by a neurotropic virus of a large family of arboviruses.

Numerous birds, mammals and reptiles found in the natural environment serve as the reservoir for tick-borne encephalitis virus (TBEV); among small mammals, the most important reservoir of the virus is the forest mouse. Domestic animals can also be hosts of the virus, the goat being the most important one.[19,23] TBEV can be easily inactivated by drying, pasteurizing and use of available disinfectants. However, it can survive for months in butter, cheese and a humid environment. The virus vectors are various species of ticks; in Poland it is mainly the common tick.

TBEV is found in the saliva of an infected tick. Having been injected into the host, the virus penetrates into its blood. Feeding on blood begins after approximately 12 h as a result of the tick being stuck in the skin; thus the longer the tick remains within the skin, the more serious the risk of infection. A tick can host all morphologic forms of TBEV and can pass the virus on to other generations of ticks. Consequently, a tick also becomes the virus reservoir, ensuring its survival during winter months, when its life cycle is aborted. TBEV not only survives in the host tick organism, but also replicates there.[23] Apart from a typical infection mode via ticks, both in Europe and in Poland, oral transmissions have been recorded by consuming the milk of infected goats.[21]

This disease is most frequent in adults and school-children following holidays. Human-to-human infections have not been observed. Infection is usually asymptomatic, and only approximately 30% of infected individuals develop clinical symptoms. The first stage of the disease involves flu-like symptoms lasting from 1 to 20 days. About 20% of asymptomatic individuals develop CNS disorders – aseptic encephalitis with features characteristic for viral etiology. In the most serious cases cerebral hemiplegia develops as well as transverse myelitis, life-threatening in up to 10% of cases. Permanent neurological consequences are observed in 4–46% of cases.[19] In children the course of the disease is milder, generally with good prognosis and low mortality (1%). Available vaccines are effective as prophylactic measures as well as the administration of specific immunoglobulin.

4.3. *Influenza*

Influenza is the birds' and animals' zoonoses that has probably been known since ancient times. Its morbidity rate can be very high and can result in pandemics. For instance, in Europe between 1918 and 1919, about 30 million people became sick. The reason for this pandemic was unknown. Only in 1930 was the influenza virus isolated. It belongs to the group of orthomyxoviruses, which was proven during the Asian influenza pandemic in 1957 and the influenza epidemic in Hong Kong in 1968.[19] Three antigen types of influenza viruses are differentiated: A, B and C. Variants B and C are almost exclusively detected in humans and trigger the so-called seasonal (common) influenza, with an incidence of 10 million people worldwide annually and a mortality rate of 0.5 million due to complications.[14] Type A is found in humans, swine, horses, and birds and is characterized by a significant antigen changeability. The virus spreads all over the world affecting the populations of domestic, wild and ornamental birds. Infected, asymptomatic birds are the largest natural reservoir for the type A viruses.[4] Birds are infected by direct physical contact or indirectly via dust, infected equipment and other domestic animals. The incubation period is short, ranging from a few hours to 3 days. Avian influenza viruses in feces are infective for 30–35 days at a temperature of 4°C and about 7 days at a temperature of 20°C, whereas 1 g of infected droppings can infect about 1 million birds. Available disinfectants effectively destroy the virus in the natural environment.[14]

4.4. *Avian influenza*

Avian influenza triggered by the H5N1 virus that was detected in birds and humans during the Asian pandemic and in Hong Kong can be transmitted from birds to humans, hence it has been classified as zoonosis. Direct avian-to-human H5N1 virus transmission is the predominant means for human infection, although the exact mode and sites of virus acquisition are incompletely understood. Handling dead poultry during the week before the onset of illness is the most commonly recognized risk factor. In at least 25% of patients infected with the H5N1 virus, the source of exposure is unknown and an environment-to-human transmission is likely.[22] Symptoms of avian influenza are similar to typical flu-like symptoms: fever, cough, malaise, and muscle aches. Respiratory symptoms frequently dominate. Treatment is analogous to that applied to seasonal influenza.

According to official data, until 2005, 120 cases of avian influenza were reported in humans, including 59 deaths, mainly in Thailand and Vietnam.[6] Generally, according to the WHO, average mortality in individuals with avian influenza reached 60% worldwide.[28] Avian influenza has been also detected in Europe: in the Netherlands, Belgium, Germany, Italy, and Russia. In these countries poultry has been destroyed by the billions. In Poland, avian influenza has been detected mainly at poultry farms, with no cases of disease in humans. It is feared that the avian influenza virus can come into contact with another type of human influenza virus in the infected organism, thus leading to a genetic exchange and consequently to a new pathogenic virus mutation. Phylogenetic data indicate that the so-called "Spanish flu" that killed 30 million people between 1918 and 1919 in Europe was in fact a strain transmitted to humans from swine that had been previously infected with the avian virus. The best method for recognizing type A influenza virus (H5N1) is to detect the virus RNA by means of polymerase chain reaction (PCR) technology.[15] Available quick tests for identifying the virus are characterized by a low clinical sensitivity in detecting type A virus.[13]

Treatment is mostly symptomatic and involves intensive care methods in serious cases.[9] Antiviral drugs (oseltamivir) improve prognosis and contribute to a higher survival rate but neither an optimal dose nor a treatment period have been determined thus far.[13] The WHO and CIE experts emphasize that avian influenza can pose a serious threat to human health worldwide. A vaccine against this type of influenza is being developed.[15]

4.5. *Swine influenza*

Swine influenza is a new type of influenza triggered by H1N1 influenza viruses spreading throughout the population of

swine worldwide, except for Australia, and larger parts of Africa and Antarctica.[12] Swine influenza is an infectious disease of the respiratory tract in swine. It generally appears in regular outbreaks. Humans usually do not get infected with swine influenza, yet epidemics triggered by mutated strains of the H1N1 virus can involve humans. Swine influenza spreads similarly to seasonal flu. The virus can also be transmitted via contact with contaminated objects and then through the nose or mouth. Infected humans can spread this infection beginning with the 1st day before the appearance of symptoms up to 7 days following the onset of illness, and in the case of children – up to 10 days and even longer. Symptoms of swine influenza are similar to those of typical seasonal flu: fever, cough, and respiratory problems necessitating hospitalization.[1,2,10] Other symptoms include headaches, sore throat, muscle aches, nausea, vomiting, and diarrhea.[7,12,15] Numerous studies indicate that this disease can be especially serious in elderly patients with additional comorbidities, children with immune deficiency and pregnant women.[25]

The first outbreak of swine influenza virus (H1N1) among humans was reported in March and April of 2009 in Mexico City, thus deriving its name of "Mexican flu." It spread to 29 countries; 4379 cases were reported, with several deaths. Because the virulence index was similar to that of the 1918–1919 influenza (Spanish flu), the WHO deemed the outbreak to be a Public Health Emergency of International Concern and declared a Pandemic Alert on 29 April 2009.[3] It was established that this new virus was a new reassortment of some H1N1 strains, with features of human flu, avian flu and swine flu. It was also determined that there was no natural resistance to this virus type. It was also worrying that the virus was dangerous to healthy young individuals and appeared in many places around the globe. Consequently, the WHO declared swine influenza to be a global pandemic.[28] In Poland, since the first case of influenza caused by the H1N1 virus appeared on 3 November 2009, 180 cases have been reported.[13]

The Canadian Food Inspection Agency depopulated the entire herd of pigs in which the virus causing the outbreak of swine influenza in people was detected. Carcasses were rendered or composted.[7] According to Centers for Disease Control and Prevention (CDC), swine flu is not transmitted via food. It is not possible to get infected with swine flu by consuming pork meat and products. Frying or roasting at a temperature above 70°C kills the virus.[8]

Swine influenza treatment in humans involves symptomatic treatment methods similar to those applied in seasonal flu. At present, CDC recommends the administration of oseltamivir or zenamivir to prevent and treat swine influenza. The highest efficiency is achieved when the drug is administered within 48 h from the onset of illness. It should be noted that swine influenza viruses (H1N1) genetically differ from human H1N1 viruses. Consequently, vaccines against seasonal human flu do not protect against A/H1N1 influenza viruses. In 2009, the WHO specialists and virologists from various countries managed to develop a vaccine against the virus that causes swine flu in humans. This vaccine turned out to be controversial due to cases of serious complications.[16]

4.6. *Bovine spongiform encephalopathy (BSE)*

BSE, commonly known as mad cow disease, belongs to the group of diseases termed transmissible spongiform encephalopathies (TSE). These diseases, both in humans and animals, are characterized by a degeneration of the CNS tissue, absence of inflammatory and immune reaction of the host, long incubation period, chronic course, and 100% mortality. In 1997, the agent causing the Creutzfeldt–Jakob disease (CJD) in humans was recognized as identical with the agent causing BSE in cattle, and in 2000, BSE was recognized as a zoonosis.[6]

BSE appeared in the UK and was first reported in 1986. In the epizootic peak of 1992–1993, 3000 cases were reported monthly. Until November 2001, a total number of 175 838 cases of confirmed BSE were reported in the UK, 98% of which involved cattle over 3 years old. Until 2001 the largest numbers of BSE cases were reported in the UK – 182 268 and Northern Ireland – 1969. In Poland this disease was extremely rare; until 2004, 15 cases were reported. Epidemiological studies carried out in the UK indicated that the "infections" were caused by meat and bone meals produced while not observing high sterilization standards. A ban on such fodder resulted in an evident decrease of BSE cases in subsequent years. A threat to human health caused by the BSE agent in animal-derived food is associated with its localization in the animal carcass: 64.1% is found in the brain, 25.6% in the spinal cord, 2.6% in trigeminal ganglia, 3.8% in the sensory nerve cell bodies of dorsal root, and 3.3% in a distal part of the small intestine. Prions have not been found in the skeletal muscles and adipose tissue. Prions are resistant to organic solvents and alcohols, detergents, chlorine, formaldehyde, gamma rays, and a temperature of 100°C, and are susceptible to, inter alia, potassium thiosulfate, phenol, and potassium permanganate.[29] BSE prions are resistant to cooking; some strains survive cooking at a temperature of 121°C for 1 h. It is assumed that the only safe gelatin is produced from skin. In order to eliminate infected cattle from the human food chain the European Union Commissioner for Agriculture, while emphasizing that "extraordinary events require extraordinary measures," introduced a ban, valid indefinitely, concerning the feeding of all farm animals on meat and bone meals. The Commissioner also imposed duties to examine all cattle older than 30 months and required the purchasing of cattle older than 30 months from infected farms in order to eliminate the entire herd, unless the animals had been tested for BSE. Additionally, a ban on consuming milk from cows infected with or suspected of having BSE was instituted.[29] In Poland, since 1 September 2001, it has been compulsory to examine animals older than 24 months and intended for consumption as well as all animals from risk groups. Animals with BSE symptoms or changes indicating BSE are regarded as unfit for consumption and are eliminated.[18]

Zoonoses have been known for ages; however, not all of them presently occur with similar intensities. Diseases that have been dangerous to human health for a long time have been observed over the last few years (anthrax, viral meningitis), as well as those that have crossed continental borders due to worldwide globalization and pose new problems to many countries, including the threat of pandemics, i.e. BSE, avian influenza (H1N1), and swine influenza (H5N1). It should be remembered that to control these diseases, apart from devised treatment standards, the cooperation of veterinary services is necessary. An ever better

veterinary supervision facilitates eliminating diseases associated with the consumption of infected meat. Some such diseases can be prevented by means of vaccinations. Constant societal education is also extremely important and required so that any introduced epidemiological regulations and recommendations are consciously observed.

5. Conclusions

1. Viral zoonoses can be a serious threat to human health due to the significant pace of pathogen proliferation. Despite medical advances, these diseases remain a serious problem for both medical and veterinarian services.
2. Due to globalization, "new zoonoses" constitute a worldwide rather than a local problem. This is evidenced by the European Union (EU) Zoonoses Directive issued in 1994 by the Council of the European Union.
3. Vaccinations do not protect against all zoonoses. Knowledge concerning the transmission modes of these diseases is especially significant, as well as is the cooperation of epidemiologists and specialists in epizootology.

Conflict of interest

None declared.

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