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A study on Demographic profile of patients with swine flu

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Abstract
The greatest severity is in young children, elderly people, immune-suppressed people, and those with chronic diseases. Most infected people recover within one to two weeks without requiring medical treatment. However, in the very young, the elderly, and those with other serious medical conditions, infection can lead to severe complications of the underlying condition, pneumonia and death. Pandemic (H1N1) 2009 is a new influenza virus that has never circulated among humans before. After outbreaks in North America early in 2009, the virus spread rapidly around the world. The patients with clinical features of Influenza like illness were enrolled. A complete clinical examination was carried out and relevant investigations done and documented in the proforma. Majority of children were aged less than 15 years and males were more affected. Fever and cold were the most common presentation.

Keywords: Swine flu, Influenza like illness, ARDS

Introduction

Influenza or flu is a viral infection that affects mainly the nose, throat, bronchi and, occasionally, lungs and commonly occurs in winter. Infection usually lasts for about a week, and is characterized by sudden onset of high fever, aching muscles, headache and severe malaise, non-productive cough, sore throat and rhinitis. The virus usually appears in epidemic form and transmitted easily from person to person via droplets and small particles produced when infected people cough or sneeze. Influenza tends to spread rapidly in seasonal epidemics. The greatest severity is in young children, elderly people, immune-suppressed people, and those with chronic diseases. Most infected people recover within one to two weeks without requiring medical treatment. However, in the very young, the elderly, and those with other serious medical conditions, infection can lead to severe complications of the underlying condition, pneumonia and death. Pandemic (H1N1) 2009 is a new influenza virus that has never circulated among humans before. After outbreaks in North America early in 2009, the virus spread rapidly around the world. Pandemic influenza is transmitted like seasonal influenza but people have virtually no immunity to it. Mitigating its effects is a public health priority [1] there are three types of flu viruses existing

- Influenza A
- Influenza B
- Influenza C Types A and B viruses cause seasonal epidemics which hit the USA and Europe virtually every winter. The type C influenza virus causes mild respiratory illness and is not responsible for epidemics. There are no B virus subtypes, but there are different influenza B virus strains. Of the three genuses of influenza viruses that cause human flu, two also cause influenza in pigs. With influenza "A" is being widespread in pigs and influenza "C" being rare.

Influenza "C" virus infects both human and pigs, but does not infect birds. Transmission between pigs and human was occurred in the past. For example, influenza "C" caused small outbreaks of a mild form of influenza amongst children in Japan and California. Due to its limited host range and the lack of genetic diversity in influenza "C", this form of influenza does not cause pandemics in human. Influenza A is generally more pathogenic than influenza B. Influenza A is a zoonotic infection, and more than 100 types of influenza A infect most species of birds, pigs, horses, dogs and seals. Indeed, the 1918 pandemic that resulted in millions of human deaths worldwide is believed to have originated from a virulent strain of H1N1 from pigs or birds [2].

Influenza a (Family Orthomyxoviridae, Genus Influenza virus A) is currently the greatest pandemic disease threat to humankind. Its rivals for this title (HIV-1, Ebola, SARS, Pneumonic plague) have higher mortality if untreated, but either lack influenza's rapid inter-personal transmission (HIV-1) or its widespread seasonal distribution (Ebola, SARS, pneumonic plague). Influenza A is unique among the major pandemic threats in that it could potentially infect 30% of the world's population within a matter of months. Even at a conservative overall mortality rate of 2%, it would result in around 135 million deaths worldwide within the first year of a new pandemic outbreak. This is about 4 times the total mortality attributed to HIV-1 in the last 30 years. Influenza a (H1N1) virus is a subtype of influenza virus A and the most common cause of influenza (flu) in humans. Some strains of H1N1 are endemic in humans and cause a small fraction of all influenza-like illness and a large fraction of all seasonal influenza. Swine flu influenza is known to be caused by influenza "A" of subtypes H1N1, H1N2, H3N1, H3N2, and H2N3. In pigs, three subtypes of influenza "A" virus (H1N1, H3N2, and H1N2) are the most common strains worldwide. In the United States, the H1N1 subtype was exclusively prevalent among swine populations before 1998; however, since late August 1998, H3N2 subtypes have been isolated from pigs. As of 2004, H3N2 virus was isolated in US swine and turkey stocks were triple re-assortments, containing genes from human (HA, NA, and PB1), swine (NS, NP, and M), and avian (PB2 and PA)

lineages. Recently, scientists obtained and sequenced the 1918 H1N1 strain from a frozen corpse found in Alaska.[3] The virus was reconstructed at the Centers for Disease Control and Prevention (CDC) laboratory in Atlanta and was found to be highly lethal when tested in mice; the virus was also found to be lethal to chicken embryos. This unique N1 neuraminidase is being studied in order to provide better insight into the N1 found in H5N1, the type responsible for avian influenza (also known as bird flu). The pathologic and imaging findings with 2009 influenza A (H1N1) infection progressed to pneumonia shows diffused alveolar damages, and ARDS. Clinical management was further complicated by pulmonary interstitial emphysema and by subsequent development of pneumomediastinum, pneumothoraces, and subcutaneous emphysema. Whereas most 2009 influenza A (H1N1) infections in healthy individuals are self-limited, it is not entirely clear which pathologic factors caused the progression to fatal disease in this case and in other cases [4].

Methodology

Source of data

This was a prospective study, which included patients with influenza like illness, who got admitted to attached hospitals of. Medical college

Method of collection of Data

The patients with clinical features of Influenza like illness were enrolled. A complete clinical examination was carried out and relevant investigations done and documented in the proforma.

Inclusion criteria

Proved cases of influenza A H1N1 and negative cases, where no other specific diagnoses were clinched.

Exclusion criteria

All other Influenza like illness patients where specific diagnosis other than influenza A H1N1 was clinched were excluded from the study group.

Investigations

Complete Hemogram

CBC profile of every patient was obtained from Automated Hematology

Results

Table 1: Age & Gender Wise Incidence

Age in years	Male	Female	Total
<15	308	157	465
15-20	86	52	138
20-25	74	44	118
25-30	28	34	62
30-35	18	22	40
35-40	28	15	43
>40	21	12	33
Total	563	336	899

Majority were children <16 years with males Predominantly affected

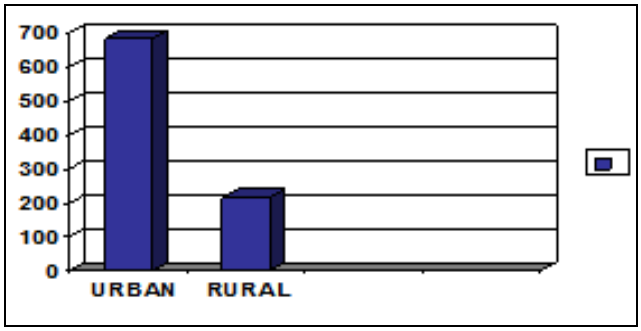

Fig 1: Urban and rural distribution of swine FLU patients

Table 2: Percentage distribution of Symptoms

Symptoms	Symptomatic Patients	Percentage
Fever	832	92.%
Cough	837	93.%
Sore Throat	690	77.%
Nasal Catarrah	459	51.%
Breathlessness	259	29.%
Others	230	26.%

Fever, cough & sore throat were the most Common symptoms at presentation.

Discussion

The known historical roots of the influenza A H1N1 can be traced to 1918, when a virus currently thought to be of avian origin overcame the complex species barrier required to infect humans. Thus began an influenza pandemic that would result in as estimated 50-1200 million deaths, more than any other influenza pandemic in history. Before 1918, influenza in humans was well known, but the disease had never been described in pigs, for pig farmers everything changed after the Cedar Rapids Swine Show, which was held from September 30 to October 5 of that year.

Similarities in the clinical presentations and pathological features of influenza in humans and swine suggested that pandemic human influenza in 1918 was actually adapted to the pig, and the search for the causative agent began. The breakthrough came in 1931 when Robert Shope, a veterinarian, transmitted the infectious agent of swine influenza from sick pigs, by filtering their virus containing secretions, to healthy animals. Viral adaptation to a new host is a complex process, involving adaptation to new cell surface receptors, changes in cell tropisms, innate immunity, and mechanisms of transmission. Influenza a H1N1 virus overcame these barriers in 1918 to emerge from an avian source simultaneously in swine and humans. Analysis of full genome sequences of representative Influenza a H1N1 viruses from 17 countries and five continents that were sampled between 1918 and 2006 shows that all eight segments of virus have had generally congruent patterns of evolution overtime [5].

In 1957, H1N1 virus was replaced by a new strain, designated H2N2, that combined genetic material from its H1N1 predecessor and an avian influenza virus. In January 1976, an outbreak of respiratory disease occurred among soldiers returning to an army base in fort Dix, New Jersy. A

novel virus H1N1/New Jersy/76 was identified as the cause of the epidemic that resulted in serologic evidence of 230 cases and one death. Even though human Influenza a H1N1 virus had not circulated since 1957, the swine Influenza A H1N1 virus that had been identified at fort Dix did not extend outside the base, in November 1977, the H1N1 strain reemerged in the former soviet union, Hong kong and North-eastern China. Since then the H1N1 influenza virus has persistently contributed to seasonal epidemics alongside the often more dominant H3N2 subtype.

This tenacious virus has drawn on a bag of evolutionary tricks to survive in one form or another in both humans and pigs, and to spawn a host of novel progeny viruses with novel gene constellations, through the periodic importation or exportation of viral genes. The 2009, H1N1 pandemic virus represents yet another genetic product in the still growing family tree of the remarkable 1918 virus. This novel Influenza 2009 A (H1N1) virus contains a combination of Swine, Avian and human influenza virus genes [6, 7].

Influenza virus is an enveloped RNA virus of the orthomyxoviridae family, of which influenza A, B and C viruses constitute three separate genera. It is endowed with an inherent capacity for genetic variation and is based on two important features; the presence of a segmented genome, with eight RNA segments that are genetically independent of each other and a high rate of mutation, especially in the surface hemagglutinin and neuraminidase proteins. Influenza A viruses are further sub divided (Subtyped) on the basis of the surface hemagglutinin and Neuraminidase antigens. Individual strains are designated according to the site of origin, isolate number, year of isolation and subtype. Influenza. A has 16 distinct H subtypes and 9 distinct N subtypes of which only H1, H2, H3, N1 and N2 have been associated with epidemics of disease in humans. Influenza B and C are similarly designated but H and N antigens from these viruses do not receive subtype designations, since intratypic variations in influenza B antigens are less extensive than those in influenza A and may not occur with influenza C virus.

Influenza A and B are major human pathogens, they are morphologically similar. The virions are irregularly shaped spherical particles, measure 80-120 nm in diameter, and have a lipid envelope from surface of which the H and N glycoprotiens project [8].

The two surface antigens of influenza undergo antigenic variation independent of each other, Minor antigenic changes are termed antigenic drift, major antigenic changes called antigenic shift, result in the appearance of a new subtype. The swine origin influenza A (H1N1) 2009 is a reassort ant with at least three parents. Six of the genes are closest in sequence to those of H1N2 ― Tripple – Reassort ant influenza viruses isolated from pigs in North America around 1999-2000.

Its other two genes are from different Eurasian avian like viruses of pigs; the NA gene is closest to H1N1 isolated in Europe in 1991-1995 and the MP gene is closest to H3N2 viruses isolated in Asia in 1999-2000. The virus possesses the Polymerase basic-2 (PB2) and polymerase A (PA)

genes of North American avian virus origin, the polymerase basic-1 (PB1) gene of human H3N2 virus origin, the hemagglutinin (HA), nucleoprotein (NP) and nonstructural (NS) genes of classical swine origin and the neuraminidase (NA) and matrix (M) genes of Eurasian swine virus origin.

The influenza virus is pleomorphic, typically having a spherical or elongated shape. It contains three surface proteins: hemagglutinin (HA), neuraminidase (NA), and the M2 proton on channel. The virus membrane consists of a lipid bilayer derived from the host cell, and a structural M1 matrix protein just beneath the lipid bilayer. Inside the virus lies the ribonucleoprotein (RNP) complex consisting of eight negative sense single stranded RNAs, the polymerase proteins (PB1, PB2, PA), and the nucleoprotein (NP).

Binding of the HA to sialic acid residues on the membrane of the host cell initiates the replication cycle. The virus next undergoes endocytosis and is encapsulated in an endosome. At this point the M2 proton channel facilitates an influx of H+ into the virus. The resultant drop in pH effects both the fusion of the virus to the endosomal wall and the release of viral RNPs into the host cell. Once in the cell, RNPs are transported through the nuclear membrane into the nucleus where they undergo transcription and translation forming new viral proteins. Due to a lack of viral RNA transcription checking mechanism, mutations are relatively frequent. The new viral proteins are transported out of the nucleus and assemble near the cell membrane, and undergo budding. The new virus is now formed but is still attached to the host cell via the binding of the HA to the sialic acid residues. In order for the virus to be liberated the NA must cleave the sialic acid residues. Now free from the host cell the virus is able to freely move throughout the host and infect new cells [9].

The initial event in influenza is infection of the respiratory epithelium with the influenza virus acquired from respiratory secretions of acutely infected individuals. The infection is more efficient by a small particle aerosol (particle diameter <10μm) Initially virus involves the ciliated columnar epithelial cells and later may involve other respiratory tract cells. In infected cells virus replicates within 4 to 6 hrs, after which infectious virus is released to infect adjacent or nearby cells. The cells eventually become necrotic and desquamate; in some areas, previously columnar epithelium is replaced by flattened and metaplastic epithelial cells. The severity of illness is correlated with the quantity of virus shed in secretions thus the degree of viral replication itself may be an important factor in pathogenesis [6].

The 2009 H1N1 virus is well adapted to mammalian hosts and binds to both α 2, 6 linked cellular receptors and α 2, 3 linked receptors. which are present in the conjunctivae, distal airways, and alveolar pneumocytes. The virus shows increased ex vivo replication in human bronchial epithelium at 33°C. In uncomplicated illness, nasopharyngeal viral RNA loads peak on the day of onset of symptoms and decline gradually afterward.

Conclusion

Infection usually lasts for about a week, and is characterized by sudden onset of high fever, aching muscles, headache and severe malaise, non-productive cough, sore throat and rhinitis.

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