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Research Article

Detection of some Microorganisms in Patients with Pericarditis by Immunofluresent Assay in Hilla City / Iraq

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ABSTRACT

In this study, (17) pericardial effusion specimens and blood samples were obtained from hospitalized patients diagnosed with pericardial effusion at Marjan Teaching Hospital, AL-Sader Teaching Hospital and Ibn-AL-Biatar Cardiovascular Center with age range between (2-77) years old from both sexes, 6(35.30%) male and 11(64.70%) of them were female. The period of collection were extended from July (2018) to January (2019). A total of (17) samples from hospitalized patients with pericardial effusion were included in this study, only 9(52.9%) patients with positive bacterial blood culture media, 7(77.8%) from female and 2(22.2%) from male, and 10(58.8%) patients with positive bacterial pericardial effusion, 7(70%) female and 3(30%) male. In the positive culture group, from 10(58.8%) cases, death occurred in 2(20%) patients, and in the negative culture group, from 7(41.2%) cases, death occurred in 2(38.5%) patients. The sera and pericardium effusion of patients in showed anti-IgM to *M. pneumoniae*, *M. pneumophilia*, *L. pneumophilia*, *C. pneumophilia*, RSV, Adenovirus, Influenza A virus, and Influenza B virus antibodies. The results showed 7/17(41.2%) positive cases of pericarditis attributed to *M. pneumoniae*. In (17) patients with acute pericarditis admitted found 2/17(11.8%) positive cases detected by IFA in patients with *L. pneumophilia*. IFA revealed that 1/17(5.9%) of positive cases for the assay positive for *Chlamydophila pneumoniae*. Recent study revealed that the IFA detect 2/17 (11.8%) of cases positive for Adenovirus. Depending on IFA recent study identified 2/17(11.8%) cases with RSV associated with another etiological agents and present a case of Influenza A virus infection 1/17(5.9%). Our results showed a case with pericardial effusion positive for Influenza B virus 1/17(5.9%) associated with presence of RSV and *L. pneumophilia* in patient with intestinal cancer with negative results of bacterial culture media for both pericardial effusion and blood.

Keywords: Pericarditis, Immunofluresent, Bacterial pericarditis.

INTRODUCTION

Pericarditis refers to inflammation of the pericardium, two thin layers of a sac-like tissue that surround the heart, hold it in place and help it work. A small amount of fluid keeps the layers separate so that there is no friction between them[1]. Although historically a disease of children and young adults, this is no longer the case: the median age at the time of diagnosis has increased by nearly (30) years over the past (6) decades. Despite advances in diagnostic and treatment modalities, purulent pericarditis remains a life-threatening illness. Unfortunately, the diagnosis is made postmortem in more than half the cases[2]. Purulent pericarditis is present in a wide variety of pathological conditions with varying etiologies such as immunosuppression and chronic diseases (e.g., alcohol abuse, rheumatoid arthritis), but is commonly secondary to injury, cardiac procedures, or insult to the pericardium[3]. The diagnosis of purulent pericarditis is often delayed due to the absence of typical pericarditis features and the tendency to attribute the nonspecific constitutional symptoms initially to the underlying infection itself[4]. Bacterial pericarditis is a rapidly progressive infection with high mortality. It is rare in the modern antibiotic era and the majority of cases occur in immunocompromised individuals or in individuals with underlying disease of the pericardium. Bacterial pericarditis usually occurs as a secondary infection by contiguous spread from surrounding intrathoracic focus of infection or by hematogenous spread from distant focus of infection. Primary involvement of the pericardium without evidence of underlying infection elsewhere is very rare[5]. The microbiology and epidemiology of purulent pericarditis have changed dramatically in the last thirty years. In the past *Staphylococcus aureus*, *Hemophilus influenzae*, and *Pneumococcus* were the predominant organisms isolated from the pericardium[6]. The pathogenesis of *H. influenzae* infections is not completely understood, although the presence of the capsule in encapsulated type b (Hib), a serotype causing conditions such as epiglottitis, is known to be a major factor in virulence. Their capsule allows them to resist phagocytosis and complement-mediated lysis in the nonimmune host. The unencapsulated strains are usually less invasive. However, produce an inflammatory response in humans, which can lead to many symptoms. Vaccination with Hib conjugate vaccine is effective in preventing Hib infection, but does not prevent infection with NTHi strains[7]. *Mycoplasma*

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pneumonia primarily causes respiratory tract infections in persons aged (5-20) years. Tracheobronchitis and bronchopneumonia are the most commonly recognized clinical symptoms associated with *M. pneumoniae* infection. Complications of this infection are unusual; in particular, cardiac involvement is very rare and is generally accompanied by pneumonia. Non-respiratory illness can therefore involve direct invasion by *M. pneumoniae* or autoimmune mechanisms, as suggested by the frequency of cross-reaction between human antigens and *M. pneumonia*[8]. *Legionella pneumophila* is a thin, aerobic, pleomorphic, flagellated, non-spore-forming, gram-negative bacterium of the genus *Legionella*. *L. pneumophila* is the primary human pathogenic bacterium in this group and is the causative agent of Legionnaires' disease, also known as legionellosis[9]. *Chlamydophila pneumoniae* has been recognized as a common cause of respiratory tract infections affecting all age groups. The organism has been implicated as an infectious trigger for acute exacerbations of COPD[10]. Human adenoviruses (HAdV) account for (7–8%) of viral respiratory illnesses in children less than (5) years. HAdV infections can cause prolonged fever with elevated inflammatory markers4 and may mimic other illnesses that require specific treatment, such as bacterial infections or Kawasaki disease (KD). Over (60) HAdV types have been defined based on genomic sequences and are classified into (7) species (A–G). HAdV-C is known for its ability to establish persistence in lymphoid organs such as tonsils and adenoids[11]. Influenza A virus infection can cause a number of complications in the pulmonary (viral or bacterial pneumonia), neurological (encephalopathy, Guillain Barré Syndrome, etc.), renal, cardiac (myocarditis, pericarditis), and muscular systems[12]. Respiratory syncytial virus is the most common pathogen causing lower respiratory tract infection in infants. Respiratory syncytial virus infection is also associated with a number of extra-pulmonary manifestations, including the cardiac system. Pericardial effusion, however, is a very rare occurrence with respiratory syncytial virus infection[13]. The mortality rate in congenital heart disease infants is reported to be as high as (37%). RSV infection is associated with myocarditis and heart block and has been suggested as a cause of pericardial disease with pericardial effusion and cardiac tamponade. The noninvasive assessment of global cardiac function and pulmonary artery pressure is suitable to elucidate the pathophysiologic mechanisms underlying the cardio-pulmonary interaction in patients with acute RSV infection in young age group[14].

Aim of study

Identification of unculturable bacteria and some viruses by using Indirect Immunoflurecent assay depending on IgM antibodies Kit.

MATERIALS AND METHODS

Patients and study design

A total of (17) pericardial effusion specimens and blood samples were obtained from hospitalized patients diagnosed with pericardial effusion at Marjan Teaching Hospital, AL-Sader Teaching Hospital and Ibn-AL-Biatar Cardiovascular Center with age range between (2-77) years old from both sexes. The period of collection were extended from July (2018) to January (2019). Pericardial effusion and blood specimens are obtained aseptically before the antibiotic therapy from all patients with pericarditis form various Hospital pericardium effusion are collected and processed for bacterial diagnosis by conventional method and by vitek system. A volume of (5) ml of fresh blood was drown from each patient by technique of vein puncture as described by[15]. The blood specimen was divided into two parts: two ml were used for blood bacterial cultivation, and the remainder three ml were used to separate the serum for serological tests.

Ethical Approval

A valid consent was achieved from each patients before their inclusion in the study.

Identification of bacteria

Colonial morphology and microscopic examination

A single colony from each primary positive culture on blood, MacConkey and nutrient agar and identify it depending on its morphological properties (colony shape, size, color, borders, and texture) and exam it by light microscope after being stained with Gram's stain. After examination it, biochemical tests were done on each isolates to complete the final identification according to[16-20].

Pneumoslide IgM

Indirect immunofluorescent assay (IFA) Kit for the simultaneous diagnosis in human serum of IgM antibodies of the main etiological agents of infectious diseases of the respiratory tract: *Legionella pneumophila* serogroup 1, *Mycoplasma pneumonia*, *Coxiella burnetii*, *Chlamydophila pneumonia*, adenovirus, respiratory syncytial virus, influenza A, influenza B and parainfluenza serotypes 1, 2 and 3.

Principle of the test

The IFA method is based upon the reaction of antibodies in the sample, tested with the antigen adsorbed on the slide surface. The specific antibodies present in the sample react with the antigen, and the immunoglobulins not binded to the antigen are removed in the washing step. In the next step, the antigen – antibody complexes react with the fluorescein – labeled anti-human globulin. It can be examined using an immunofluorescence microscope.

Kit contents

vircell pneumoslide: (10) slides of (10) wells with the following antigens:

L. pneumophila serogroup (1) suspended (0.5%) normal chicken yolk sac to improve the antigen adhesion and avoid the bacterial aggregation.

M. pneumonia in McCoy cells.

C. burnetii in phase II suspended in (0.5%) normal chicken yolk sac to improve the antigen adhesion and avoid the bacterial aggregation.

C. pneumonia, elementary bodies.

Adenovirus in HEp-2 cells.

Respiratory syncytial virus in HEp – 2 cells.

Influenza A in LLC – MK2 cells.

Influenza B in LLC-MK2 cells.
Par influenza serotypes 1, 2 and 3 in LLC-MK2 cells.
Cells control.
All the antigens in the slide are obtained in cell culture except *L. pneumophila*. Each viral well contains between (1-15%) of infected cells inactivated with formaldehyde and non-infected cells fixed with acetone.
Vircell PBS: (1) vial of PBS pH (7.2) powder to reconstitute with (11) of distilled water.
Vircell pneumoslde IgM positive: 500 µl of positive control serum, containing sodium azide.
Vircell pneumoslde IgM negative control: 500 µl of negative control serum, containing sodium azide.
Vircell anti-human IgM FITC conjugate: (2) vials with (1.1) ml of fluorescein – labeled anti-human IgM fluorescein conjugate in a phosphate buffer containing Evans blue, sodium azide and a protein stabilizer.
Vircell mounting medium: (3) ml of mounting medium: buffered glycerol, containing sodium azide.
Vircell anti-human IgG globulin (sorbent): (1) vial of (1.65) ml of sorbent (goat anti – human IgG, containing sodium azide).

Assay procedure

Bring all reagents to room temperature before use. Allow the slides to reach room temperature before opening. Prepare a (1\2) dilution of serum samples by adding (25) µl of sample to (25) µl of PBS (2). The control sera (3) and (4) should not be diluted. Treat diluted sera with anti-human IgG sorbent (7) by adding (30) µl of sera to (150) µl of sorbent and thoroughly mix. Control sera (3), (4) must not be sorbent treated. The treated sera should be centrifuged to remove the precipitate, which interfere with the test. Add (15) µl of sorbent – treated serum in every slide well (1). Add (15) µl of non – diluted positive control (3) to each well of a slide and (15) µl of non – diluted negative control (4) to each well of another slide. Place the slide in a humid chamber and incubate at (37C0) for 90 minutes. Rinse slide (1) briefly with a gentle stream of PBS (2) (avoid directing PBS at wells) and immerse in PBS while shaking gently on a shaker, for ten minutes. Dip wash slide briefly in distilled water. Allow the slide (1) to air dry. Add (15) µl of anti – human IgM FITC conjugate solution 5M to each well. (No dilution required). Incubate slide in a humid chamber for 30 minutes at (37C0). Repeat steps 6 and 7. Add a small drop of mounting medium (6) to each well and carefully cover with a coverslip. Read the slide as soon as possible in a fluorescence microscope at 400X magnification. If this is not possible, store in the dark at (2-8C0) up no more than (24) hours, until observation.

RESULTS AND DISCUSSION

A total of (17) pericardial effusion specimens and blood samples were obtained from hospitalized patients diagnosed with pericardial effusion at Marjan Teaching Hospital, AL-Sader Teaching Hospital and Ibn-AL-Biatar Cardiovascular Center with age range between (2-77) years old from both sexes, 6(35.30%) male and 11(64.70%) of them were female. These results shown in Figure (1). The period of collection were extended from July (2018) to January (2019). A total of (17) samples from hospitalized patients with pericardial effusion were included in this study, only 9(52.9%) patients with positive bacterial blood culture, 7(77.8%) from female and 2(22.2%) from male, these results shown in Figure (2a, b), and 10(58.8%) patients with positive bacterial pericardial effusion, 7(70%) female and 3(30%) male. These results were shown in Figure (3a, b). In the positive culture group, from 10(58.8%) cases, death occurred in 2(20%), and in the negative culture group, from 7(41.2%) cases, death occurred in 2(38.5%). The results were shown in Figure (4a, b).

Immunoflorucent assay for detection of microorganisms

The sera and pericardium effusion of patients in showed anti-IgM to *M. pneumoniae*, *M. pneumophilia*, *L. pneumophilia*, *C. pneumophilia*, RSV, Adenovirus, Influenza A virus, and Influenza B virus antibodies. Immunofluorescence is the visualization of antigens using antibodies as fluorescent probes. The benefits of immunofluorescence are numerous, and the technique has proven to be a powerful tool for determining the cellular distribution of known antigens in the frozen tissues or in the localization of specific DNA sequences on chromosomes. The method has achieved the status of combining high sensitivity with high resolution in the visualization of antigens and will be a major tool for many years that any pathologist studying cells or molecules cannot afford to ignore. For a methodology article on immunofluorescence labeling of formalin-fixed, paraffin-embedded tissue or microbial antigens[21]. *M. pneumoniae* is a common pathogen that causes upper and lower respiratory tract infections, manifesting as pharyngitis, bronchitis, and atypical pneumonia in children and adolescents. Patients with or without respiratory symptoms can manifest illness involving the skin, central nervous system (CNS), blood, heart, gastrointestinal tract, and joints. Skin lesions include a variety of exanthemas, most notably maculopapular rashes, erythema multiform, and Stevens-Johnson syndrome. Cardiac involvement, such as acute myocarditis, pericarditis, or myo-pericarditis[8]. The results showed 7/17(41.2%) positive cases of pericarditis attributed to M. pneumoniae. These results were shown in Figure (5a, b). In conclusion, our experience suggests that *M. pneumoniae* should be considered as a potential cause in cases of pericarditis associated with upper respiratory symptoms, pneumonia, pleural effusions, arthralgia, and/or a recurrent/refractory clinical course. The results of the present study were in accordance to the outcome of a work done by[22]. who conducted the serological results showed that the serum sample obtained from the patient with pericarditis was positive for IgM antibodies against M. pneumoniae. The cardiac complications of *M. pneumoniae* infections include pericarditis and myocarditis possibly leading to cardiac tamponade arrythmias, have been reported occasionally. In most of these cases, *M. pneumoniae* was diagnosed by serological examination, although diagnosis by culture of the organisms in pericardial fluid specimens has been documented elsewhere[23]. *M. pneumoniae*-associated

carditis is a rare and serious disease, requiring in many cases intensive care during the acute phase, and leading to cardiac sequelae in almost one-half of the patients. Cardiac tamponade by *M. pneumoniae* is a rarely reported complication[24]. *Legionella* infection is a rare cause of acute pericarditis. In (17) patients with acute pericarditis admitted found 2/17(11.8%) positive cases detected by IFA in patients with associated pneumonia and serological signs of *Legionella* infection with *L. pneumophilia*. these results were shown in Figure (6a, b). *Legionella* pericarditis should be suspected in cases of combined pericarditis and pneumonia. Prompt identification of Legionella will permit early appropriate

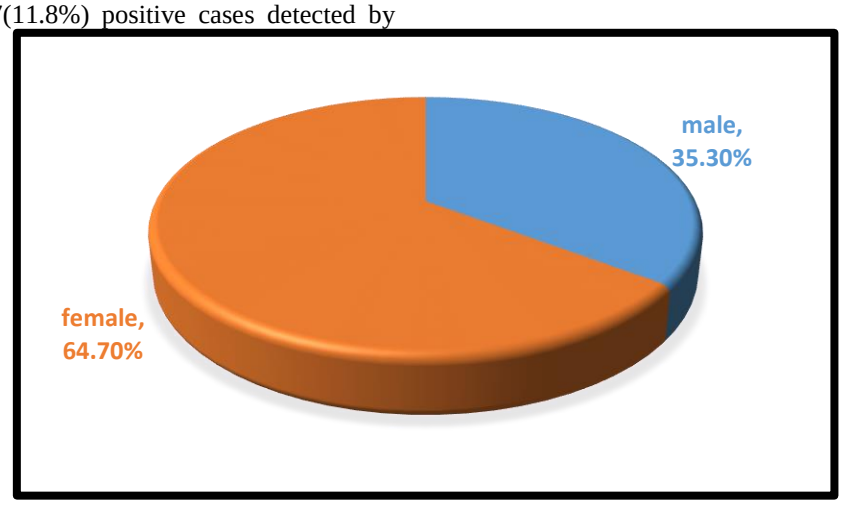

Figure 1: Distribution of patients according to Gender.

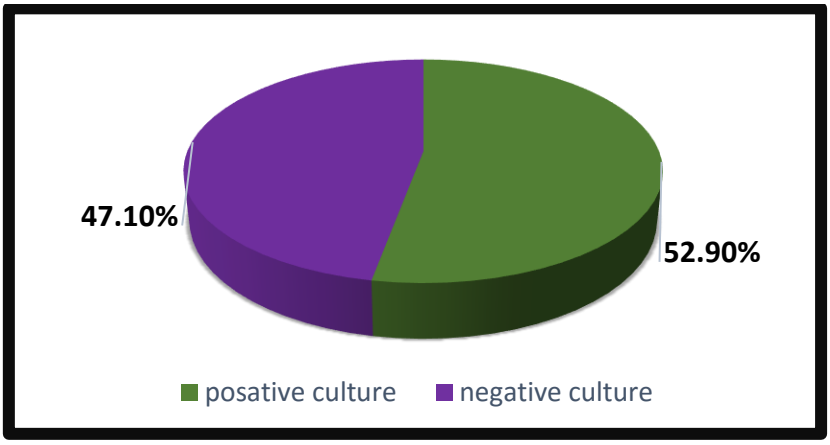

Figure 2a: Distribution of hospitalized patients with pericardial effusion according to bacterial blood culture.

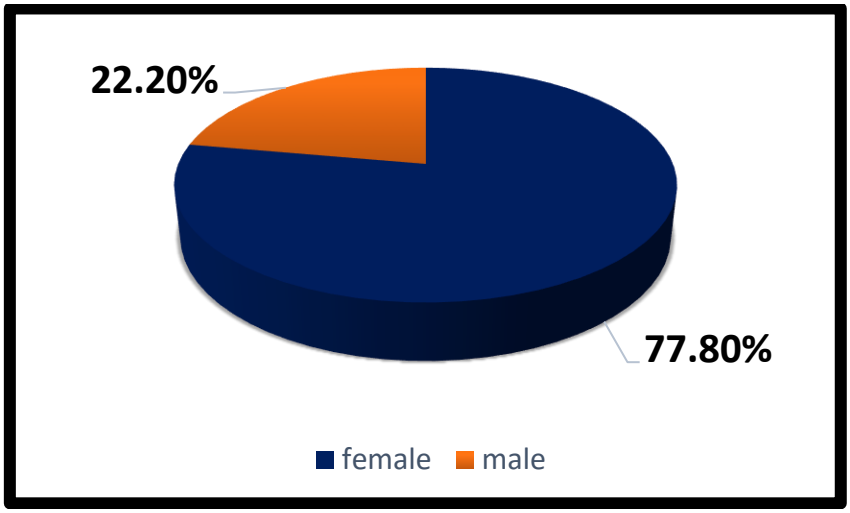

Figure 2b: Distribution of hospitalized patients with pericardial effusion according to gender.

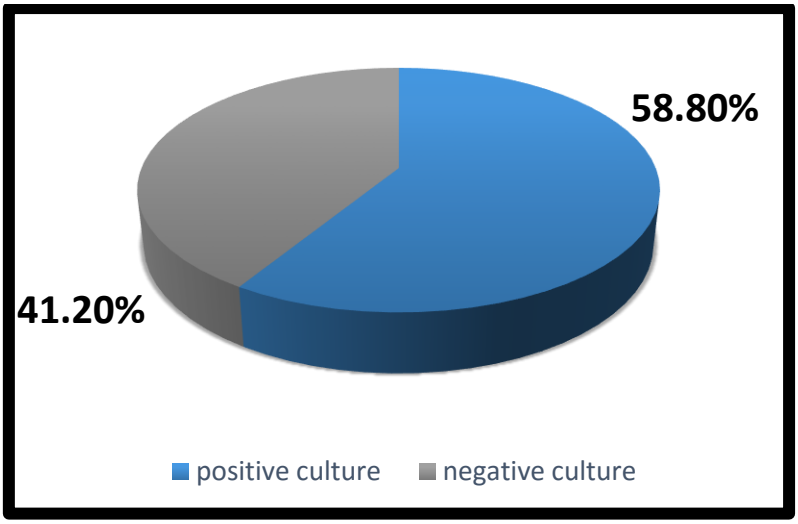

Figure 3a: Distribution of hospitalized patients with bacterial pericardial effusion.

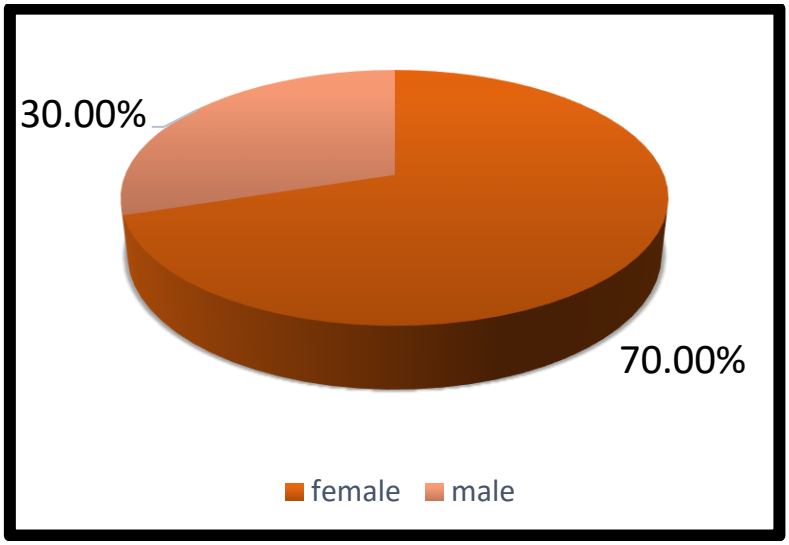

Figure 3b: Distribution of hospitalized patients with bacterial pericardial effusion according to gender.

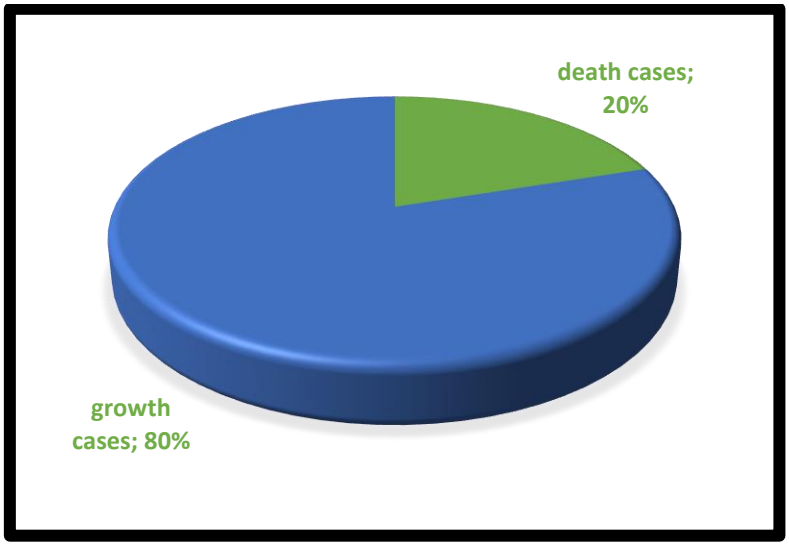

Figure 4a: Death of positive culture bacterial groups.

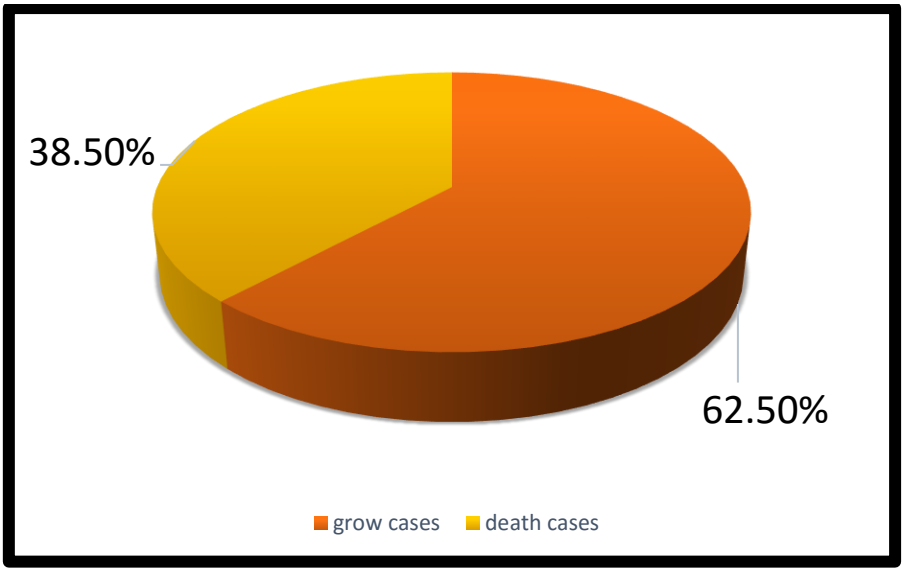

Figure 4b: Death of negative culture bacterial groups.

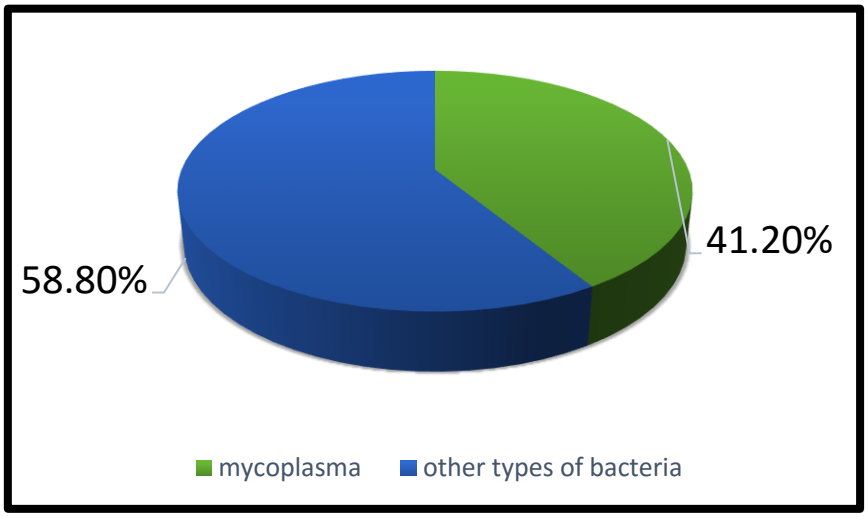

Figure 5a: percentage positive cases of pericarditis attributed to *M. pneumoniae*.

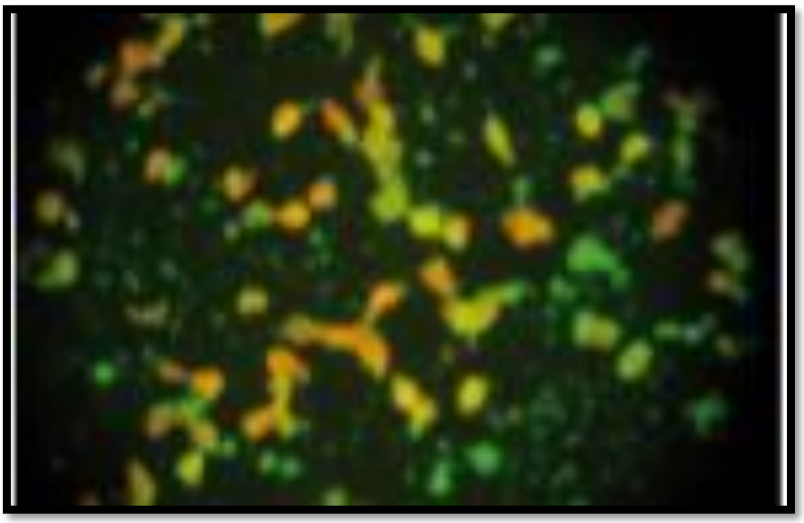

Figure 5b: Detection of *Mycoplasma pneumoniae* by immunoflorucent assay.

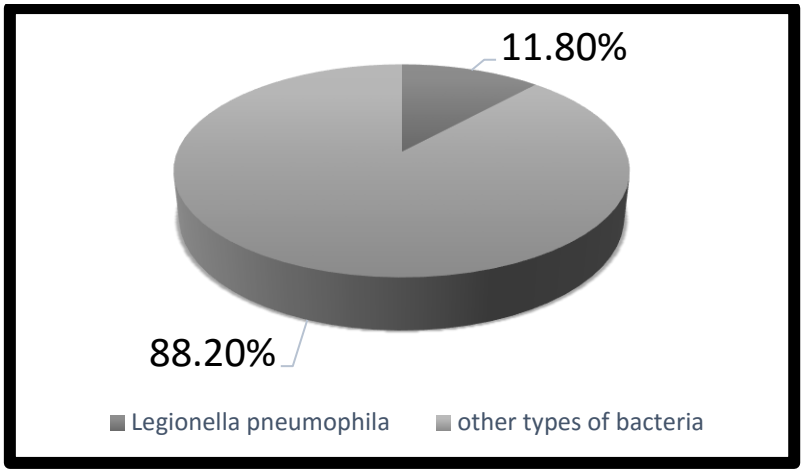

Figure 6a: Percentage positive cases of pericarditis attributed to *Legionella pneumophila*.

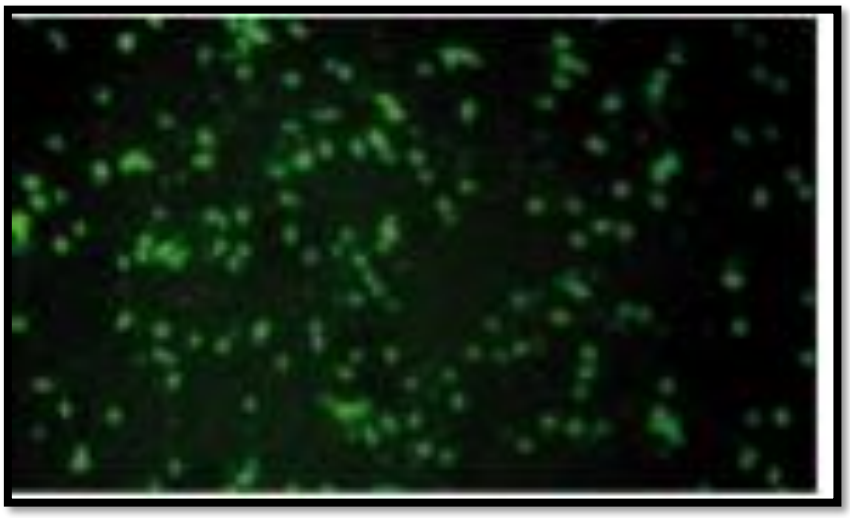

Figure 6b: Detection of *Legionella pneumophila* by immunoflorucent assay.

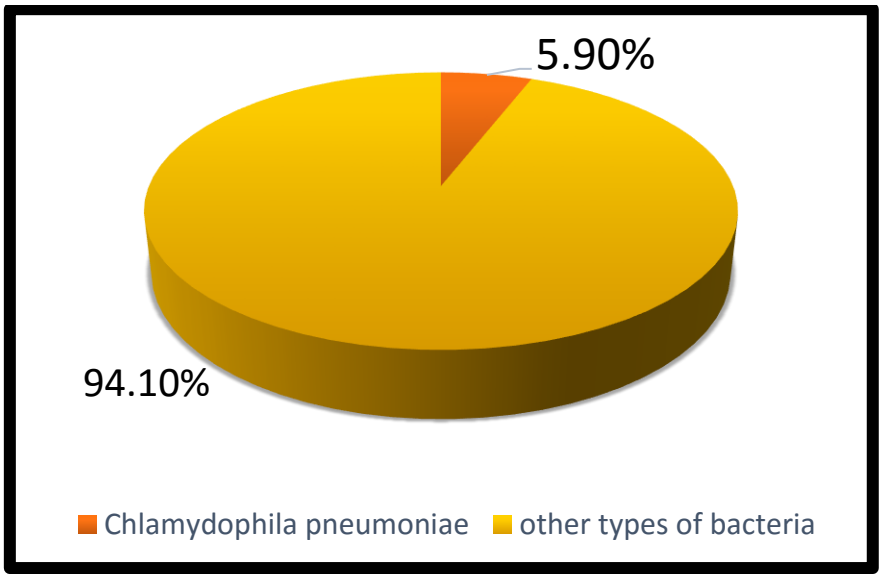

Figure 7a: Percentage positive cases of pericarditis attributed to *Chlamydophila pneumoniae*.

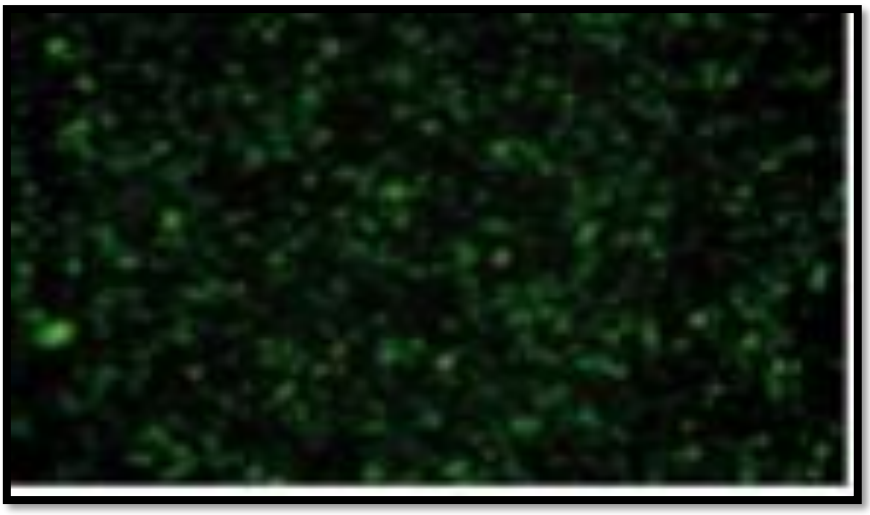

Figure 7b: Detection of *Chlamydophila pneumoniae* by immunoflorucent assay.

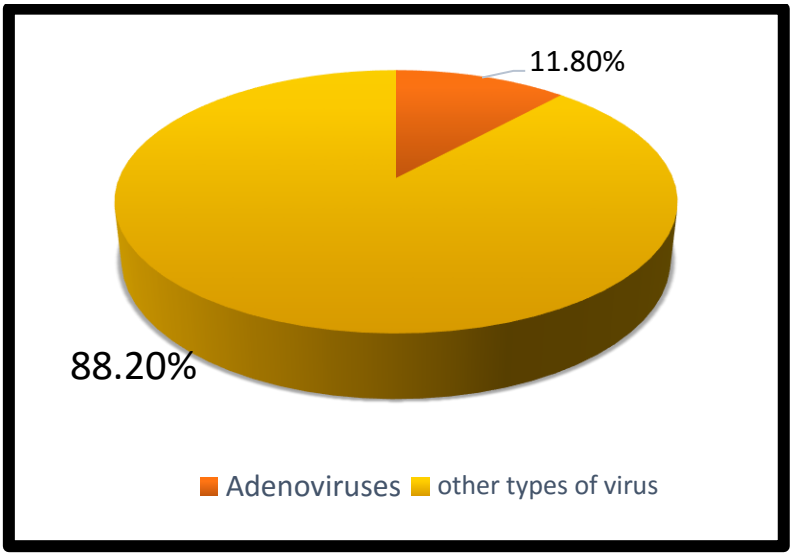

Figure 8a: Percentage positive cases of pericarditis attributed to Adenoviruses.

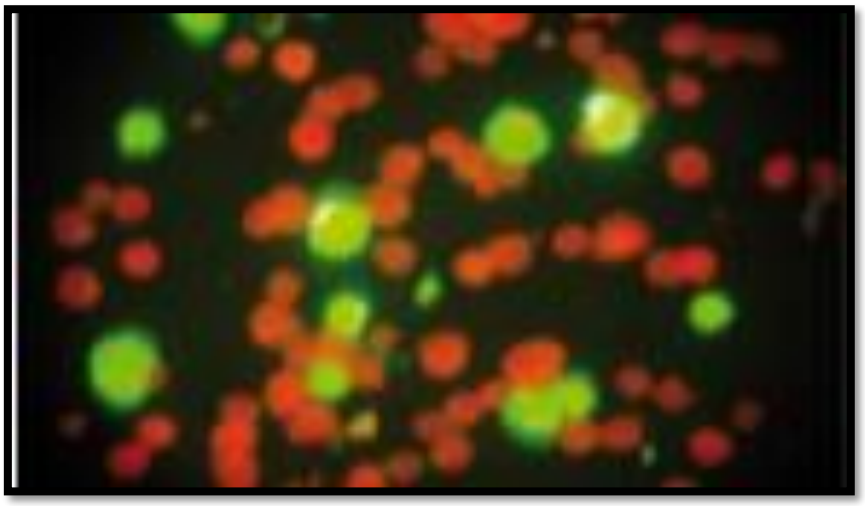

Figure 8b: Detection of Adenoviruses by immunoflorucent assay.

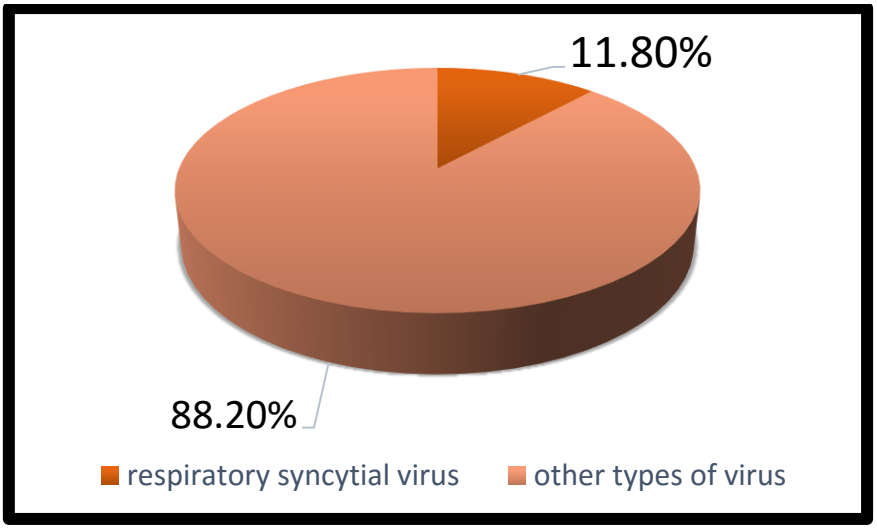

Figure 9a: Percentage positive cases of pericarditis attributed to respiratory syncytial virus.

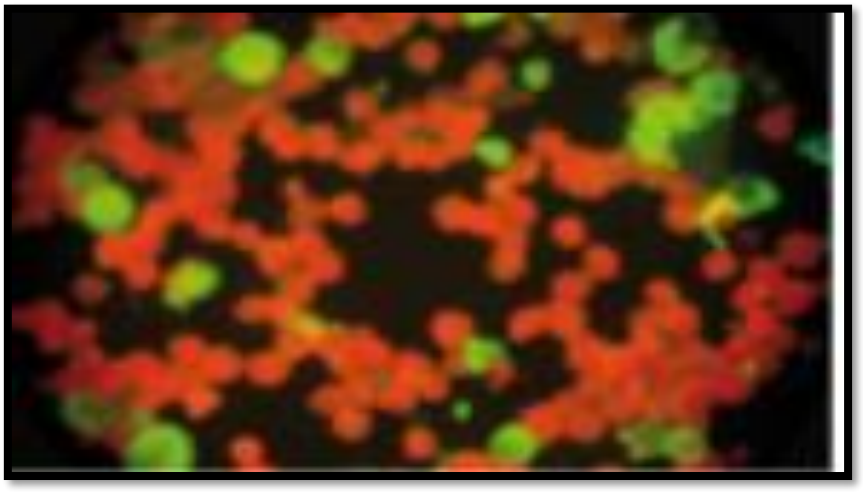

Figure 9b: Detection of respiratory syncytial virus by immunoflorucent assay.

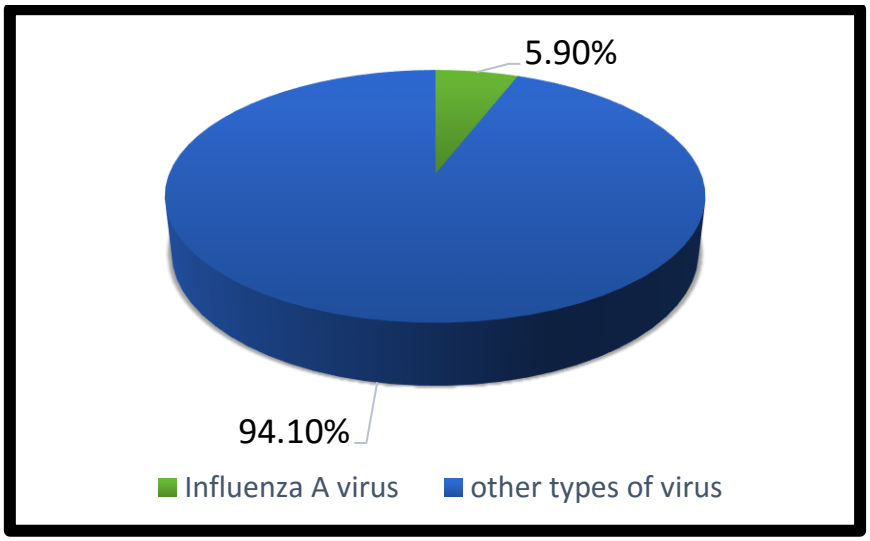

Figure 10a: Percentage positive cases of pericarditis attributed to Influenza A virus.

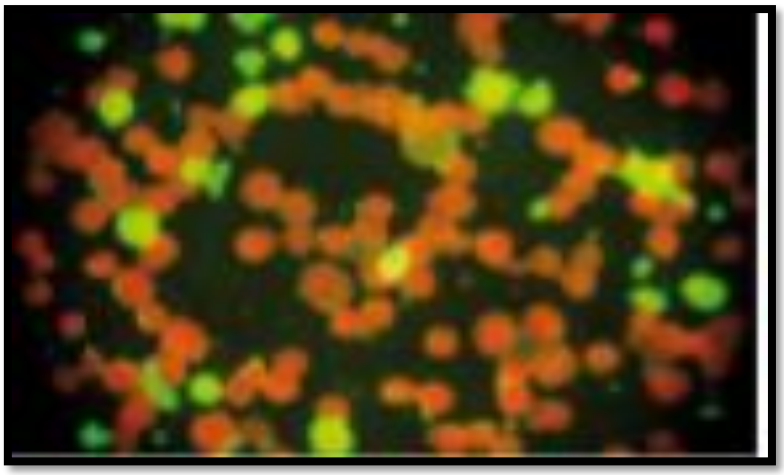

Figure 10b: Detection of Influenza A virus by immunoflorucent assay.

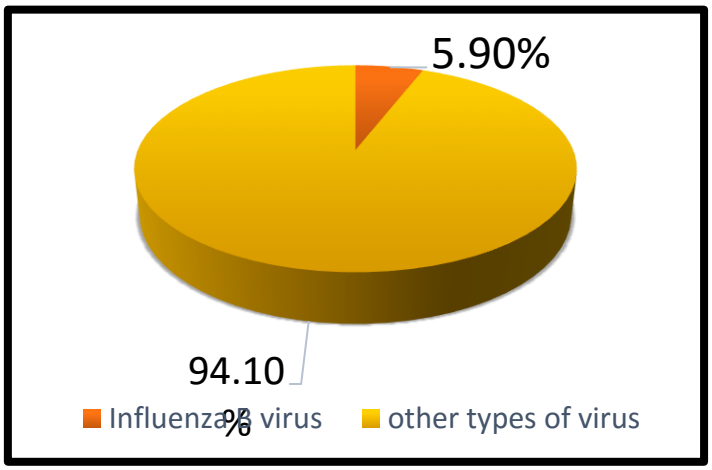

Figure 11a: Percentage positive cases of pericarditis attributed to Influenza B virus.

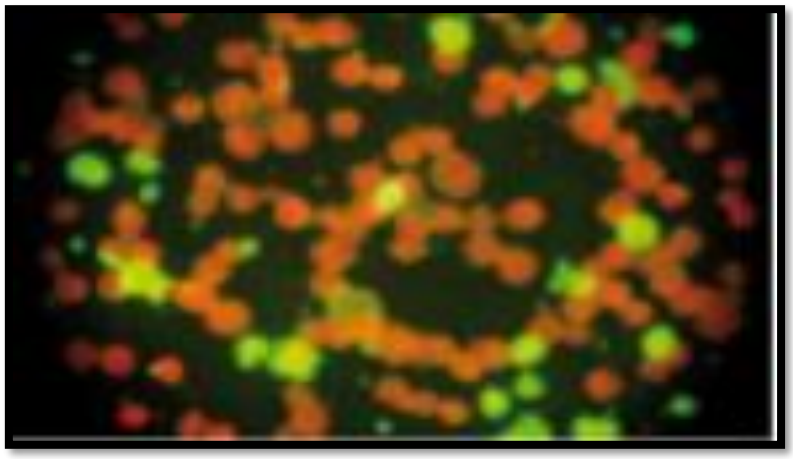

Figure 11b: Detection of Influenza B virus by immunoflorucent assay.

treatment. *Legionella* pericarditis was first described by Harris in (1981)[25]. Since that time, few cases have been reported in the literature[26]. The recent study described a diagnosis of *Legionella* pericarditis based on indirect fluorescent antibody staining of pericardial effusion and patients serum. Present study emphasize the importance of obtaining pericardial effusion specimens for indirect fluorescent antibody analysis, particularly when the diagnosis remains in question. Cases of pericarditis attributed to *L. pneumophilia* associated with pneumoniae in old age patient. The diagnosis of this case depend on indirect fluorescent antibody staining has been successful in making the diagnosis of pericardial fluid in prior reports[27]. *L. pneumophila* was isolated from the pericardial fluid of a no immunosuppressed patient with pulmonary infiltrates, cardiac tamponade, and histologic

evidence of pericarditis. This is the first reported case in which the association of *L. pneumophila* infection and pericarditis has been proved by IFA of the organism from pericardial fluid and serum. The indirect immunofluorescence assay has been used to demonstrate antibody responses to Legionella pneumophila infections and provide a retrospective serodiagnosis. Validating serological tests for legionellosis has been difficult, however, because of the lack of sufficient numbers of known positive sera from patients with a definite diagnosis of legionellosis. Confirmation by isolation of *L. pneumophila* has not been possible in the majority of cases because of the absence of appropriate media[26]. In this study IFA revealed that 1/17(5.9%) of positive cases for the assay positive for *Chlamydophila pneumoniae* the same patient with *L. pnemophilia*. Similar cases are very rarely described in the literature. The results were shown in Figure (7a, b). *Chlamydiae* are common human pathogens, causing a broad spectrum of infectious diseases. Chlamydial infections involving the heart have been described in numerous reports. These organisms were documented to cause endocarditis, myocarditis and pericarditis. Furthermore, *Chlamydia pneumoniae*, respiratory pathogen, has also been implicated in coronary artery disease. Information on the discovery of *Chlamydia* species is also included and the problem of the species determination of *Chlamydia* in interpretation of the older literature is mentioned[28] referred to case report of pneumonia in a child with hemorrhagic pericardial effusion with a positive result by a new *C. pneumoniae* pericarditis, the detection done by IFA. Recent result also similar to that conducted by[29] who describes the differential diagnostic algorithm used during hospitalization and the corresponding treatments provided for the patient with serious acute *Chlamydia* pericarditis. They discuss an analysis of the possible organic manifestations of acute and chronic *Chlamydia pneumoniae* infections. This bacterium most frequently attacks the respiratory tract but in humans may also cause separate organic complications such as effects on joints, the lungs, the CNS and the heart. The medicine of choice is always long-term antibiotic therapy (macrolides, fluoroquinolones and tetracyclines). *Chlamydia* infection of the cardiovascular system is associated with pericarditis, endocarditis and myocarditis. *Chlamydia* particles can also be observed in damaged heart valves. C. pneumoniae infection increased the risk of a cardiac event three–fold. This risk factor is synergistic with the smoking risk. Immunohistochemistry also demonstrated Chlamydia lipopolysaccharide in samples of aortic aneurysm. Chlamydial inflammation may play a role in the oxidation of low-density lipoprotein in atherosclerotic lesions[30]. An important feature of the adenovirus is that it has a DNA rather than an RNA genome. Portions of this viral DNA persist in host cells after viral replication has stopped either as a circular extra chromosome or by integration into the host DNA[31]. This persistence may be important in the pathogenesis of the known sequelae of adenoviral infection that include Swyer-James syndrome, permanent airways obstruction, bronchiectasis, bronchiolitis obliterans, and steroid-resistant asthma[32]. Adenoviruses are frequent causes of fevers, upper respiratory tract symptoms and conjunctivitis and produce infections that are usually mild and self-limiting. Adenoviral lower respiratory tract infections are infrequent, sporadic and most commonly associated with adenovirus types (3), (5) and (7)[33]. Recent study revealed that the IFA detect 2/17 (11.8%) of cases positive for Adenovirus. The results were shown in Figure (8a, b). Adenoviruses are common human pathogens and have been shown to cause a wide spectrum of disease mainly in children and human immunodeficiency virus-infected individuals. Among the clinical manifestations of invasive adenovirus disease, pericarditis with effusion is a rare complication and the mechanisms by which adenovirus because disease are poorly understood. Adenovirus pericarditis is a commonly reported cause of acute pericarditis[34]. Depending on IFA recent study identified 2/17(11.8%) cases with RSV associated with another etiological agents. The results were shown in Figure (9a, b).[35] describe a case of pericarditis and large pericardial effusion in a 63-year-old patient undergoing autologous hematopoietic stem cell transplant for multiple myeloma. Pericardial tissue biopsy demonstrated fibrinous pericarditis, and immunohistochemistry stains were positive for respiratory syncytial virus. The patient improved with oral ribavirin and intravenous immune globulin infusions. Respiratory syncytial virus is the most common pathogen causing lower respiratory tract infection. Respiratory syncytial virus infection is also associated with a number of extrapulmonary manifestations, including the cardiac system. Pericardial effusion, however, is a very rare occurrence with respiratory syncytial virus infection. Avery young Infant with respiratory syncytial virus bronchiolitis whose clinical course was associated with pericardial effusion, treated conservatively was detected as a rare case[13]. Extrapulmonary manifestations suggest that RSV may infect organs other than the lung. It is unlikely that systemic co-infection with bacterial pathogens is responsible for most extra pulmonary manifestations. However, a direct involvement of RSV is suggested by its isolation from myocardial tissue and the reported occurrence of significant pericardial effusion[36]. Patients infected with RSV may demonstrate clinical manifestations other than lower respiratory tract infection including cardiac involvement. The heart involvement following RSV infection varies from heart block, ventricular arrhythmia or a variable degree of pericardial effusion. RSV infection is associated with myocarditis and heart block and has been also suggested as a cause of pericardial disease with pericardial effusion and cardiac tamponade. The noninvasive assessment of global cardiac function and pulmonary artery pressure is suitable to elucidate the pathophysiologic mechanisms underlying the cardio-pulmonary interaction in patients with acute RSV infection in young age group[37]. Cardiac complications of influenza infections have been described in the literature as early as the 1900s[38]. Recent study present a case of Influenza A virus infection 1/17(5.9%)

leading to pericarditis and subsequent pericardial effusion with cardiac tamponade. The results were shown in Figure (10a, b). Influenza causing a pericardial effusion is rare, and to our knowledge, there was other case report of an adult patient presenting with Influenza A virus cardiac tamponade due to an infection[39]. This case highlights a rare but life threatening complication of Influenza A virus infection[40] describe a case of a young female with no prior cardiovascular history who presents with a pericardial effusion and shock secondary to cardiac tamponade from pericarditis due to influenza A virus. They concluded that the potential severity of the virus infections and the utility of considering cardiac tamponade in patients presenting with influenza symptoms and circulatory collapse. Influenza A virus, is mentioned in textbooks as a cause of myo-pericarditis, pericarditis, and pericardial effusion, few cases have been reported in the medical literature[41]. An adult suspected of acute myocardial infarction was found to have pericardial effusion and perimyocarditis. Rise of influenza A antibody supported the diagnosis of viral cause[41]. Our results showed a case with pericardial effusion positive for Influenza B virus 1/17(5.9%) associated with presence of RSV and *L. pneumophilia* in-patient with intestinal cancer with negative results of bacterial culture media for both pericardial effusion and blood. These results were shown in Figure (11a, b) Influenza is a viral infection caused by Influenza viruses belonging to the Orthomyxoviridae family. They are divided in Influenza A and B virus −responsible for seasonal epidemics with (3–5) million severe cases and about 300,000 deaths per year on the world- and Influenza C virus -causing, in general, mild disease[43]. Specifically, Influenza B virus presents two distinct subtypes -Victoria and Yamagata- circulating in humans, and whose transmission can occur through fine particles (aerosol), droplet nuclei, and contact. Seasonal influenza may present with both asymptomatic and fulminant manifestations, depending on the host and virus characteristics[44]. Upper respiratory tract symptoms (nose, throat, and bronchi) are the most frequent. Pulmonary (primary viral or secondary bacterial pneumonia, exacerbations of underlying chronic lung disease) and non-pulmonary (myositis, rhabdomyolysis, renal failure, myo-pericarditis, exacerbation of coronary artery disease or heart failure, Reye syndrome, encephalomyelitis, transverse myelitis, Guillain-Barrè syndrome, aseptic meningitis, and encephalitis) complications are rare[45].

CONCLUSIONS

In this study, it is concluded that,

There are few data regarding the etiological agents related to pericarditis in Iraq.

Old age patients have the peak incidence of pericarditis and the rate of infection in women was higher than men.

Indirect Immunoflurecent assay is a good tool of identification of different microorganisms by using IgM antibodies.

results were shown in Figure (3.12a, b).

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