



ROLE OF DIAGNOSIS IN PREVENTION AND CONTROL OF COMMUNICABLE DISEASES

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Abstract- Communicable diseases have been said to cause a nuisance in recent times. They are of several origins but pose a threat to the entire public health arena as they are transmissible. It takes us to one of the most prevalent and recent diseases, that is, the novel coronavirus. This paper focuses on discussing several communicable diseases such as covid-19, Zika, Ebola, tuberculosis, Mokeypox, West Nile virus, Measles, MRSA, Hantavirus, Influenza and HIV in terms of their origin, prevention, treatment and most importantly their diagnosis. The purpose of this paper is to highlight how early diagnosis helps in better treatment. It is quite important because an effective tool can result in reduction of suffering from persistent disease. It also helps in preventing transmission by reducing its span and facilitates treatment in the early stages of the treatment when the immune system of the body is not the comprised. Various diagnostic tools are further discussed in the paper such as blood tests, RT-PCR test, image scans, stool sample, biopsies etc, throwing indications at the need of new and efficient as well as cost effective methods to prevent progression of infection.

Keywords - Communicable disease, Infection, Clinical diagnosis, Laboratory findings, Viral infection.

INTRODUCTION

Communicable diseases are also known as infectious diseases, which spread rapidly and cause enormous loss of health, human lives, as well as large costs to society. HIV/AIDS, tuberculosis(TB), fungal infections, measles, clostridioides difficile enterocolitis, severe acute respiratory syndrome(MERS), ebola are some of the examples of communicable diseases. In general, when they spread over a small areas, they cause epidemics and, in extreme cases, they may cause a devastating pandemic such as the covid-19. The best strategy to control the spread of communicable diseases, is to understand the variables or to halt their spread on early diagnosis of diseases.[1]The success rate of specific diagnostic techniques in any population depends on various factors such as type of type of microbial pathogen, technical expertise, availability of resources, disease severity and degree of epidemic diseases in the area.[2]In this review paper we have discussed the role of clinical and laboratory diagnosis to control communicable diseases.

COVID-19

SARS-CoV-2, a novel beta coronavirus, was discovered in Wuhan, Hubei Province, China, in December 2019. The virus has spread quickly throughout numerous countries, prompting the World Health Organization (WHO) to declare a pandemic on March 11, 2020. Because it shares many symptoms with other respiratory viruses, clinical diagnosis of this unusual sickness, known as coronavirus disease-2019 (COVID-19), becomes problematic. We examine the evolution, features, benefits, and limitations of a variety of laboratory methods used to diagnose SARS-CoV-2, as well as the important clinical and image findings of COVID-19 patients.[3]When it comes to diagnosing an active COVID-19 infection, NAAT is the method of choice. The preferred initial diagnostic test is the use of a real-time polymerase chain reaction (RT-PCR) assay to detect SARS-CoV-2 RNA from the upper respiratory tract. SARS-CoV-2 viral proteins are detected in respiratory samples using antigen detection assays. Although most commercially available kits need samples from the nasal cavity or nasopharynx, other samples such as saliva have been investigated. Non-quantitative antibody detection is particularly valuable in epidemiological surveys, which can quantify the attack rate in a given community. In contrast, although not the test of choice for acute infection, semi-quantitative or quantitative assays that can quantify the quantity of antibody production can identify a change in antibody titre can play a role in diagnosing acute infection. Antibody detection assays typically target one of two antigens in SARS-CoV-2: the nucleocapsid (N) or spike (S) protein. The detection technique differs as well. Enzyme-linked immunosorbent assays (ELISA) and chemiluminescence immunoassays (CLIA) are extensively used in laboratory-based assays. Point-of-care testing are simple to use equipment that can be utilised outside of the lab. The majority of commercially available POCTs rely on the detection of SARS-CoV-2 antigen or host antibody.

Antigen-detecting quick diagnostic tests and antibody-detecting rapid diagnostic tests are no longer recommended for patient treatment as of April 2020, according to WHO. [4] COVID-19 is now divided into four stages based on symptom severity: mild, moderate, severe, and critical. Patients with modest symptoms do not have any radiological findings. Fever, respiratory symptoms, and radiographic characteristics are all present in moderate patients. Patients who are considered severe meet one of three criteria: (a) dyspnea with a respiratory rate of more than 30 times per minute, (b) oxygen saturation less than 93 percent in ambient air, and (c) PaO₂/FiO₂ less than 300 mm Hg. Patients who are critically ill must meet one of three criteria: Respiratory failure, septic shock, and multiple organ failure are all possible outcomes.[5] SARS-CoV-2 is a respiratory virus that spreads through contact droplets and fomites from an infected person, who may or may not be symptomatic. The virus causes a sluggish reaction in the lungs during the incubation phase. SARS-CoV-2 mostly infects alveolar epithelial cells and causes respiratory symptoms. The treatment is symptomatic in the lack of any clinically established therapeutic alternatives, and current clinical management involves infection prevention and control measures as well as supportive care. Antiviral medications (e.g., remdesivir, hydroxychloroquine, chloroquine) and supportive therapies (vitamin C, azithromycin, corticosteroids, IL-6 antagonists) are available. The world's top research goal right now is developing an effective COVID-19 vaccine. Regulatory authorities have already given their approval to some vaccinations for the prevention of COVID-19.[6]

MEASLES

With an estimated 770,000 fatalities each year, measles is a primary cause of morbidity and mortality worldwide.[7]The WHO recommends that nations implement case-based monitoring with laboratory testing as a substantial confirmatory component when the incidence of measles drops in regions. A serum sample for IgM testing and a urine sample or nasopharyngeal (NP) sample for detection of measles viral genome should be collected from clinically suspicious measles patients upon presentation to the health centre, according to the WHO.[8] Because of the high transmissibility of measles infection and the necessity for prompt and reliable laboratory confirmation of clinically diagnosed measles, an appropriate assay should be able to detect most measles cases early in the course of disease without requiring a second sample. Current methods have centred on serologic testing for the first appearing serum immunoglobulin, IgM, and to a lesser extent, IgG, to address these objectives. ELISA and EIA for measles IgM and IgG, which meet the basic criteria for rapid, accurate, reproducible, and efficient measles diagnosis, come in a variety of formats, are logistically easier to execute than previously utilised laboratory.[9] EIA testing for IgM. The current recommended laboratory assays for the confirmation of clinically confirmed measles are serum-based IgM EIAs [10] EIA IgG. Measles in a qualitative sense IgG EIAs, which are accessible as commercial and "home-made" kits, provide significant advantages over previously utilised tests in

terms of speed, sensitivity, and specificity, as well as ease of use.[11] Measles is a highly contagious respiratory virus infection with maculopapular rash, fever, cough, coryza, and conjunctivitis as common clinical signs. The disease's causal agent, measles virus (MV), is a negative-strand RNA virus belonging to the Morbillivirus genus in the Paramyxoviridae family.[12]

HIV

In 1981, the ACQUIRED IMMUNODEFICIENCY syndrome (AIDS) was first recognised as a clinical entity. [13] Since then, the causative agent - the human immunodeficiency virus - has been identified, and researchers have established that AIDS is only one of the clinical manifestations of this virus infection. Acute immunodeficiency syndrome (AIDS) is the most severe form of a range of clinical diseases caused by a chronic retroviral infection that largely affects the immune system's cell-mediated arm.[14] The early signs and symptoms of HIV infection are diverse, ranging from mild non-specific fatigue and malaise to fever, night sweats, and weight loss. They can be caused by the HIV virus directly or by an opportunistic infection or malignancy. It's helpful to understand the natural history of HIV infection when studying early signs and symptoms, which is represented in the Centers for Disease Control's (CDC) classification of HIV infection's clinical manifestations.[15] Recognizing the infection is the first step in treatment. Early detection of HIV infection should prompt an evaluation of the patient's immunological status, consideration of antiretroviral medication, commencement of prophylaxis against some frequent opportunistic pathogens, and coordination of patients with other health care providers.[16]

The existence of HIV-specific antibodies can be used to detect an infection .[17] Antibodies specific to HIV can be identified in nearly every HIV-positive person. Their existence indicates that you have an active HIV infection that is persistent. The demonstration of infectious virus utilising cell culture or the detection of viral antigen (p24 antigen) or viral nucleic acid (through NAT, nucleic acid testing) can also be used to provide a direct diagnosis of HIV infection. Aside from qualitative examinations, assays for quantitative virus identification have grown increasingly important: the concentration of viral RNA in plasma, or "viral load," has become a critical tool for guiding antiretroviral therapy.[18] A variety of quick HIV tests, often known as rapid/simple (R/S) test instruments, are now available. One of four immunodiagnostic principles underpins these tests: Immunodot (dipstick), immunofiltration, and immune chromatography are examples of particle agglutination techniques.[19] Many viral assays [such as DNA polymerase chain reaction (PCR), p24 antigen testing, and HIV isolation] identify the virus but do not quantify it, hence they are typically utilised in diagnostic situations.[20]

MRSA

Methicillin-resistant MRSA, or methicillin-resistant *Staphylococcus aureus*, has emerged as a source of skin infections and, less typically, invasive infections in otherwise healthy adults and children in the community.[21] SSTIs, pneumonia, osteoarticular infections, toxic shock syndrome . *S. aureus*, caused by the release of bacterial toxins and presenting with clinical features such as fever, rash, and hypotension) and bacteraemia, which can be complicated by endocarditis or severe sepsis HA-MRSA, CA-MRSA, and LA-MRSA strains have different clinical presentations and risk factors for infection.[22] Clinical and screening samples are two types of microbiological specimens from which MRSA can be isolated. Clinical samples (such as purulent discharge, deep tissues, sputum, and blood) are taken from people who have symptoms or signs of infection in order to look for active infection, whereas screening samples (such as nasal, perineal, and throat swabs) are taken to look for asymptomatic colonisation. MRSA can be detected directly from clinical or screening samples or identified from presumptive staphylococcal colonies isolated from clinical samples using a variety of phenotypic and non-phenotypic approaches. For clinical diagnostics, phenotypic approaches are frequently preferred.[23]

EBOLA

The nucleoprotein (NP), glycoprotein (GP), polymerase (L), VP24, VP30, VP35, and VP40 viral proteins are encoded by a single-stranded RNA genome. Several approaches for detecting Ebola virus infection and/or sickness have been developed over the last 25 years that can be used in clinical laboratory settings.[24]. There are three types of tests: (i) serologic tests that detect host antibodies generated against the virus, (ii) antigen tests that detect viral proteins, and (iii) molecular tests that detect viral RNA sequences.[25] since the initial epidemic investigations of the Ebola virus in 1976, serologic techniques for the detection of particular antiviral antibodies

in patient serum have been employed to demonstrate current or prior infection with the virus.[26]Viral isolation in cell culture, commonly using Vero E6 African Green monkey kidney cells, is the classic gold standard approach for confirming the presence of Ebola virus. Within 1 to 5 days of inoculation, the virus can be directly detected by electron microscopy or indirectly visualised by immunofluorescence microscopy. While these approaches are reliable for detecting Ebola virus, they need biosafety level 4 (BSL-4) confinement and are normally limited to research and public health laboratories.[27]

The CDC's Ebola virus diagnostic RT-PCR techniques were originally tested on serum samples acquired from critically unwell patients during the 1995 Kikwit epidemic . These assays used PCR to amplify the L, GP, and NP genes, then gel electrophoresis to detect the amplicons based on their size. The use of a chaotropic agent such as guanidine thiocyanate to chemically inactivate infectious virus during the initial steps of RNA extraction was a significant advantage of this technology, allowing subsequent sample processing to be done on the benchtop.[28]Fever, exhaustion, headache, nausea/vomiting, abdominal discomfort, muscle/joint pain, diarrhea, and anorexia/weight loss were all common symptoms among patients with laboratory-confirmed cases (Table). Except for difficulty swallowing, there was no difference in the proportion of individuals reporting signs and symptoms between those who lived and those who died .[29]

ZIKA

Zika fever is a virus that causes an acute febrile sickness that is spread mostly by mosquitos of the genus Aedes. It distinguishes the disease from others caused by flaviviruses, such as chikungunya and dengue fever. Many patients infected with the Zika virus (ZIKV) will experience no or only minor clinical symptoms. The clinical conditions are non-specific and are defined by low-grade inflammation.Fever, erythematous maculopapular rash with pruritus, non-purulent conjunctival hyperemia without pruritus, arthralgia, myalgia, and arthralgia headache. It's a harmless, self-limiting, and short-term condition. Guillain-Barré syndrome (GBS), for example, is a complication. Abortion and foetal abnormalities, such as microcephaly and retinal defects, are possible outcomes. The importance of the laboratory investigation is greater.In situations of suspected ZIKV infection in pregnant women who have developed neurological problems. Conjunctivitis, rash, sore throat, fever, joint discomfort, myalgia, and headache are the most prevalent signs and symptoms. The Abdominal pain, constipation, and diarrhoea are some of the less common symptoms. Diarrhea, disorientation, canker sores, photophobia, nausea, and vomiting are some of the symptoms that people experience. Retro-orbital discomfort and anorexia. In the early stages of the condition, the patient is in a state of confusion. a fever ranging from 38°C to 38.5°C that lasts one or two days after the rash and headache first appeared.[30]

A layer of skin exists. A maculopapular rash is characterised by small, numerous bumps. They are papules that can clump together to produce big red blotches are frequently irritated and relieved in a subtle manner. It primarily has an impact on the palm and sole, as well as the face, neck, torso, and limbs. In two or three days, the rash will have improved, and it vanishes , on average, however it could last up to two years, weeks(16). Hyperemia conjunctivitis is a common symptom of conjunctivitis. There has been edoema but no purulent discharge from the eyes. Patients complain of joint pain and myalgia, as well as mild lowback pain. Hands, wrists, and ankles are the joints that are most impacte ankles and knees. The joint problems last for about a week between three and five days. The illness is harmless, self-limiting, and only lasts a few days. However complications such as Guillain-Barré syndrome (GBS), a type of paralysis, can occur. It's possible that a neurological condition causes progressive and irreversible loss of function. Muscle strength is only transient. It has been widely reported, particularly in the United States. The relationship between microcephaly and Zika fever in Brazil during the pregnancy.

The more likely you are to acquire this deformity. When a maternal infection arises during the first trimester of pregnancy. [31] Ordering laboratory tests that use technology to determine high sensitivity, specificity, and predictive value testing is now essential. It assists us in making decisions. Diagnostic choices The importance of the clinical laboratory cannot be overstated. Primarily for the purpose of diagnosing asymptomatic diseases patients, such as those suffering from neoplasia (cervical cancer) and viral infections, such as human immunodeficiency virus (HIV) infection (HIV). ZIKV infection can be diagnosed using laboratory tests in the blood, urine, sperm, amniotic fluid, and cerebrospinal fluid. The ZIKV was found in a urine sample 15 days after infection using the same RT-PCR approach. Different techniques, such as ELISA, indirect immunofluorescence, and fast immune chromatography, could be used to look for circulating antibodies. Acute infection is defined by

the presence of antibodies to immunoglobulin class M (IgM). In endemic places, it must be done around the third day of illness and can be discovered between the second and 12th weeks after the claimed exposure. The infection can be ruled out if the test results are negative. Immunoglobulin class G (IgG), which is present in both the convalescent and healing stages, can also be quantified.[32]

TUBERCULOSIS

Mycobacterium tuberculosis (*M. tuberculosis*) is a major pathogen that causes tuberculosis. More people die from tuberculosis than any other diseases. Every second, an infection occurs, as a result of which a new TB infection develops a tenth of one percent of the world's population annually.[33] Determining whether a patient has immunologic evidence of tuberculosis infection, or "germs in the body," aids in the diagnosis of tuberculosis disease, particularly when the organism cannot be detected directly. The tuberculin skin test (TST) and the IFN-release assays are two procedures that can be used to identify if someone is infected with *M. tuberculosis*. [34] The first nucleic acid-based amplification test (NAAT) to be certified by the FDA for the detection and identification of *M. tuberculosis* from direct specimens was the amplified mycobacterium direct test (AMTD) (Ho-logic, San Diego, CA) in 1995. To identify the organism, this assay uses transcription-mediated amplification of a region of the 16S rRNA gene exclusive to the *M. tuberculosis* complex. Resistance to at least isoniazid and rifampin, two of the four first-line antituberculosis medications that form the backbone of any antituberculosis regimen, is described as multidrug-resistant tuberculosis (MDR-TB).[35]

INFLUENZA

Depending on the conditions, making a clinical diagnosis of influenza might be challenging or simple. When the epidemiological setting is suitable, the patient is a teenager or an older child, and the symptoms are typical, it's simple. It's difficult, on the other hand, when it doesn't match any of these premises. Because symptomatology is connected to age, typical clinical signs such as rhinitis, fever with or without chills, cough, headache, joint and muscular pain, and malaise are only referred to children aged 3–4 years old. The patient frequently expresses his desire to "feel sick," but his overall state is not alarming. In most situations, however, a generic diagnosis of "flu" or "flu-like illness," regardless of whether the causal agent is one of the flu viruses or another respiratory virus, is sufficient.[36] In routine clinical practice, determining the presence of influenza is quite challenging. Clinical characteristics were discovered in less than 40% of children with established influenza, and only 32% of children clinically diagnosed with (suspected) influenza had the diagnosis confirmed by laboratory testing. 4 The clinical diagnosis of influenza had a higher sensitivity and positive predictive value during peak influenza activity and in children aged 7–13 years, but was statistically lower during the early and late phases of the pandemic and in children under 3 years old.[37] Patient treatment should be guided by tests that produce data quickly and can alter clinical management. The sensitivity and specificity of the test employed, as well as information on influenza circulation in the community, should all be considered when determining the likelihood of influenza infection based on the patient's signs and symptoms. Cell culture in the traditional sense. It takes 2 to 14 days to identify a virus (median: 3–5 days) and is less sensitive than the best polymerase chain reaction test. Rapid cultures have largely supplanted traditional cell culture in many laboratories due to its simplicity and speed. The sensitivity is adequate, and it is comparable to that of traditional cell cultures.[38]

HANTAVIRUS

In recent decades, hantavirus has gotten a lot of attention as an emerging pathogenic virus. It causes two different diseases in humans: Human pulmonary syndrome (HPS) and Hemorrhagic fever with renal syndrome (HFRS).[39] Humans are not a natural host for hantaviruses, thus infection happens by mistake when virus-containing, aerosolized rodent excretions such as urine, feces, or saliva are inhaled. People who live or work in close proximity to infected rodents are at a higher risk of infection, and studies reveal that these persons have higher percentages of seropositive individuals than control subjects.[40] HFRS and HPS are clinical syndromes that share some similarities. The hantavirus serotype Puumala produces NE, a milder subtype of HFRS, in Europe. Viremia develops after an initial infection of alveolar macrophages, and life-threatening acute-phase symptoms are predominantly caused by infection of vascular endothelial cells in the lungs and kidneys, which results in a loss of barrier function and a substantial rise in endothelial permeability. The symptoms of NE include a high fever, headache, backache, and abdominal pain. In the early stages of the disease, transient thrombocytopenia is common. After 3 or 4 days, conjunctival haemorrhages, palatine petechiae, and a truncal petechial rash may appear.

The incubation period for HFRS is seven to thirty-six days. Only 10 to 15% of cases progress to a severe stage, with fatality rates ranging from 6 to 15%. Capillaries and venules are involved throughout the body in HFRS. It causes a variety of hemorrhagic symptoms and circulatory problems. Acute vision impairment, acute myopia, CNS problems with convulsions, myocarditis, and severe gastrointestinal haemorrhages are all extrarenal symptoms.

Thyroid, liver, and pancreas may also be affected. HFRS also shows lung involvement, however to a lesser level than HPS. HPS is characterised by flu-like symptoms such as high fever, myalgia, and headache when it first appears. Within 2 to 15 days, the individuals develop acute noncardiac pulmonary edoema and hypotension.[41] For the laboratory diagnosis of an acute hantavirus infection, an ELISA-based detection of NP-specific IgM antibodies is commonly used. Between 8 and 25 days following the commencement of the disease, the highest titers are visible. It's worth noting that PUUV NP-specific ELISA cross-reacts with HTNV NP for the differential diagnosis of Puumala and Hantaan virus infections, whereas HTNV NP-specific ELISA exhibits virtually no cross-reaction with PUUV NP. Immunochromatographic assays and reverse transcriptase-PCR have also become more popular in recent years, but they have not yet gained widespread acceptance as conventional clinical laboratory procedures.[42]

WEST NILE VIRUS

The West Nile virus (WNV) was originally discovered from an infected woman's blood in the West Nile area of what is now Uganda in 1937. In addition to people, the virus was found in birds and animals during further tests in Egypt. WNV initially surfaced in the United States in August 1999 and has since spread across the country.[43] The most common way for humans to contract WNV is by a bite from an infected culicine mosquito. When mosquitoes feed on diseased birds with high amounts of WNV in their blood, they become infected. When infected mosquitoes feed on humans or other animals, they can transmit WNV. People, horses, and the majority of other mammals do not develop high-level viremias and are hence considered "dead-end" hosts. When working with live WNV, laboratory staff should take steps to avoid contact with potentially contaminated tissue and bodily fluids and utilise biosafety level 3 containment. If unintended WNV exposure occurs, the affected region should be thoroughly cleansed with soap and water, and a baseline serum sample taken and kept. Medical evaluation, interaction with public health experts, and the collection of further serum samples for testing are all advised if the worker becomes ill during the next two weeks. In humans, the viral incubation time ranges from 2 to 15 days after WNV infection (usual period, 2-6 days).

Clinically, the majority of human infections are undetectable. According to seroepidemiological studies, 1 in 5 infected people will have a febrile illness, which includes fever, headache, backache, myalgia, and anorexia, and lasts 3 to 6 days. About half of the patients develop a roseolar or maculopapular rash that affects the face and torso and can persist up to a week. Lymphocytic lymphadenopathy is very prevalent. Myocarditis, pancreatitis, and fulminant hepatitis are some major non-neurologic consequences that can develop in people. One in every 150 infected people will have a serious central nervous system (CNS) infection. MRI is more accurate than computed tomography in detecting CNS inflammation. In individuals with WNV encephalitis, computed tomographic scans frequently reveal normal results or preexisting lesions, as well as long-term alterations.[44] Virus or virus-specific antibodies are used to confirm infection. Serum or CSF can be used for serologic testing. On or after the eighth day of illness, IgM specific for West Nile virus can be found in serum or CSF using an IgM capture ELISA in at least 90% of patients. PCR can be used to identify viral RNA in clinical specimens. The reverse-transcriptase-PCR (TaqMan) in CSF and serum had a sensitivity of 57 percent and 14 percent, respectively, in a study of individuals with serologically confirmed acute WNVME.[45]

MONKEYPOX

Camelopox, cowpox, vaccinia, and variola viruses are all Orthopoxviruses, and monkeypox virus is one of them. The virus is the most common Orthopoxvirus to impact human populations after smallpox was eradicated in 1980, according to the World Health Organization. In resource-poor endemic locations where monkeypox is found, clinical detection, diagnosis, and prevention are major issues. Human monkeypox was not recognised as a unique infection in people until 1970, during smallpox eradication efforts in the Democratic Republic of the Congo, when the virus was isolated from a patient with probable smallpox infection (DRC).[46] A widespread headache and weariness accompany the early febrile prodrome. Many individuals have maxillary, cervical, or inguinal lymphadenopathy (1–4 cm in diameter) prior to and concurrent with rash development. Lymphoma nodes

that have grown in size are hard, sensitive, and occasionally painful. Smallpox was not associated with lymphadenopathy. The occurrence of lymphadenopathy could indicate that the immune system recognises and responds to the monkeypox virus more effectively than the variola virus, but this idea has to be investigated further.[47] Fever usually goes down the next day or up to three days after the rash appears. The rash usually starts on the face and spreads out in a centrifugal pattern across the body. First macular, then papular, then vesicular, and finally pustular lesions are common.[48] A patient's total number of lesions can range from a few to thousands. Lesions in the oral cavity are common and can make drinking and eating uncomfortable. Digital pictures and the Internet are 21st-century instruments for clinical consultation, given the unique presentation of lesions.[49] Patients' skin was described as bloated, stiff, and painful until crusts emerged. A second febrile phase, which occurs when skin lesions become pustular, has been linked to a worsening of the patient's overall state.[50] diagnostic assays are crucial in determining the presence of an orthopoxvirus infection. When these tests are paired with clinical and epidemiological data, such as a patient's vaccination history, they are most effective. Lesion exudate on a swab or crust specimens are still some of the best and least invasive acute patient specimens due to the limited cold chain and limited resources for sample collection and storage. When stored in a dark, chilly environment, viral DNA present in lesion material is stable for a long time, which is vital to consider when cold chain is not readily available. Traditional tests including virus isolation from a clinical specimen, electron microscopy, and immunohistochemistry are still relevant, but they require significant technical skills and training, as well as specialised equipment. To determine the presence of Orthopoxvirus or monkeypox virus in a lesion sample, real-time polymerase chain reaction (PCR) can be used. These tests are extremely sensitive and capable of detecting viral DNA. Because real-time PCR is currently best performed in a large laboratory, its application as a real-time diagnostic in rural, resource-poor places is limited. Advances in technology may make the use of real-time PCR for diagnostic purposes outside of major laboratories more viable.[51]

CONCLUSION

This paper discusses various communicable disease in terms of their clinical as well as laboratory diagnosis indicating a need of further research in the post covid arena where diagnosis was the only reliable tool to prevent its transmission. Early diagnosis not only prevents the disease from progression but also inhibits its transformation into an epidemic. One such scope of further research can be about the use of risk assessment charts like the one used for cardiovascular and various other diseases. This helps in data mining as well as quantifying several aspects of symptoms through which we can assign scores to different individuals presenting with any of the communicable disease. This can help in risk estimation and patient shortening by identifying the niche where the maximum and quickest care needs to be delivered. Also, in terms of the recent novel coronavirus outbreak, such assessment tools can be helpful for public health as a whole to decide which group must be given clinical care first and who can recover at home with self care techniques without supervision. Hence, it can be said that the only way to prevent progression and transmission of communicable disease is an efficient tool for its clinical as well as laboratory diagnosis.

REFERENCES

- [1]. Van Seventer, J. M., & Hochberg, N. S. (2017). Principles of Infectious Diseases: Transmission, Diagnosis, Prevention, and Control. *International Encyclopedia of Public Health*, 22–39.
- [2]. Srivastava, S., Singh, P. K., Vatsalya, V., & Karch, R. C. (2018). Developments in the Diagnostic Techniques of Infectious Diseases: Rural and Urban Prospective. *Advances in infectious diseases*, 8(3), 121–138.
- [3]. da Silva, S., Silva, C., Guarines, K. M., Mendes, R., Pardee, K., Kohl, A., & Pena, L. (2020). Clinical and Laboratory Diagnosis of SARS-CoV-2, the Virus Causing COVID-19. *ACS infectious diseases*, 6(9), 2319–2336.
- [4]. Lai, C., & Lam, W. (2021). Laboratory testing for the diagnosis of COVID-19. *Biochemical and biophysical research communications*, 538, 226–230.
- [5]. Wang, Y., Wang, Y., Chen, Y., & Qin, Q. (2020). Unique epidemiological and clinical features of the emerging 2019 novel coronavirus pneumonia (COVID-19) implicate special control measures. *Journal of medical virology*, 92(6), 568–576.
- [6]. Rahman, S., Montero, M., Rowe, K., Kirton, R., & Kunik, F., Jr (2021). Epidemiology, pathogenesis, clinical presentations, diagnosis and treatment of COVID-19: a review of current evidence. *Expert review of clinical pharmacology*, 14(5), 601–621.

- [7]. Murray, Christopher & Lopez, Alan & Stein, Claudia. (2001). The Global Burden of Disease 2000 project: Aims, methods and data sources. Global programme on evidence for health policy. 8.
- [8]. Mortality reduction and regional elimination: strategic plan 2001–2005; global measles. 2001:1–31.
- [9]. Bellini, W. J., & Helfand, R. F. (2003). The challenges and strategies for laboratory diagnosis of measles in an international setting. *The Journal of infectious diseases*, 187 Suppl 1, S283–S290.
- [10]. Ratnam, S., Tipples, G., Head, C., Fauvel, M., Fearon, M., & Ward, B. J. (2000). Performance of indirect immunoglobulin M (IgM) serology tests and IgM capture assays for laboratory diagnosis of measles. *Journal of clinical microbiology*, 38(1), 99–104.
- [11]. World Health Organization. Laboratory diagnosis of measles infection and monitoring of measles immunization: memorandum from a WHO meeting. *Bull World Health Organ* 1994; 72:207–11
- [12]. el Mubarak, H. S., Van De Bildt, M. W., Mustafa, O. A., Vos, H. W., Mukhtar, M. M., Groen, J., el Hassan, A. M., Niesters, H. G., Ibrahim, S. A., Zijlstra, E. E., Wild, T. F., Osterhaus, A. D., & De Swart, R. L. (2000). Serological and virological characterization of clinically diagnosed cases of measles in suburban Khartoum. *Journal of clinical microbiology*, 38(3), 987–991.
- [13]. Gottlieb MS, Schroff R, Schanker HM, Weisman JD, Fan PT, Wolf RA, et al. Pneumocystis carinii pneumonia and mucosal candidiasis in previously healthy homosexual men: evidence of a new acquired cellular immunodeficiency. *N Engl J Med* 1981;305:1425-31.
- [14]. Gallo RC, Salahuddin SZ, Popovic M, Shearer GM, Kaplan M, Haynes BF, et al. Frequent detection and isolation of cytopathic retroviruses (HTLV-III) from patients with AIDS and at risk for AIDS. *Science* 1984;224:500-3.
- [15]. Chaisson RE, Volberding PA. Clinical manifestations of HIV infection. In: Mandell GL, Douglas RG Jr, Bennett JE, editors. Principles and practice of infectious diseases. 3rd ed. New York, NY: Churchill Livingstone, 1990:1059-92.
- [16]. Miedzinski L. J. (1992). Early Clinical Signs and Symptoms of HIV Infection: Delaying progression to AIDS. *Canadian family physician Medecin de famille canadien*, 38, 1401–1410.
- [17]. Gürtler L. Difficulties and strategies of HIV diagnosis. *Lancet* 1996;348:176-9.
- [18]. Berger A, Preiser W, Doerr HW. The role of viral load determination for the management of human immunodeficiency virus, hepatitis B virus and hepatitis C virus infection. *J Clin Virol* 2001;20:23-30.
- [19]. Buttò, S., Suligoi, B., Fanales-Belasio, E., & Raimondo, M. (2010). Laboratory diagnostics for HIV infection. *Annali dell'Istituto superiore di sanita*, 46(1), 24–33.
- [20]. Gupta, V., & Gupta, S. (2004). Laboratory markers associated with progression of HIV infection. *Indian journal of medical microbiology*, 22(1), 7–15.
- [21]. strategies for clinical management of MRSA in the community ; summary of an experts' meeting convened by the Centers for Disease Control and Prevention: Gorwitz, Rachel J.; Jernigan, Daniel B.; Jernigan, John A.; 2006.
- [22]. Lowy, F. D. Staphylococcus aureus infections. *N. Engl. J. Med.* 339, 520–532 (1998).
- [23]. Lee, A. S., de Lencastre, H., Garau, J., Kluytmans, J., Malhotra-Kumar, S., Peschel, A., & Harbarth, S. (2018). Methicillin-resistant Staphylococcus aureus. *Nature reviews. Disease primers*, 4, 18033.
- [24]. Strong JE, Grolla A, Jahrling PB, Feldmann H. 2006. Filoviruses and arenaviruses, p 774 –790. In Detrick B, Hamilton RG, Folds JD (ed), Manual of molecular and clinical laboratory immunology, 7th ed. ASM Press, Washington, DC.
- [25]. Towner JS, Rollin PE, Bausch DG, Sanchez A, Crary SM, Vincent M, Lee WF, Spiropoulou CF, Ksiazek TG, Lukwiya M, Kaducu F, Downing R, Nichol S. 2004. Rapid diagnosis of Ebola hemorrhagic fever by reverse transcription-PCR in an outbreak setting and assessment of patient viral load as a predictor of outcome. *J Virol* 78:4330 –4341.

- [26]. Johnson KM, Lange JV, Webb PA, Murphy FA. 1977. Isolation and partial characterisation of a new virus causing acute haemorrhagic fever in Zaire. *Lancet* 310:569–571
- [27]. Feldmann H, Sanchez A, Geisbert TW. 2013. Filoviridae: Marburg and Ebola viruses, p 923–962.
- [28]. Broadhurst, M. J., Brooks, T. J., & Pollock, N. R. (2016). Diagnosis of Ebola Virus Disease: Past, Present, and Future. *Clinical microbiology reviews*, 29(4), 773–793.
- [29]. MacNeil, A., Farnon, E. C., Wamala, J., Okware, S., Cannon, D. L., Reed, Z., Towner, J. S., Tappero, J. W., Lutwama, J., Downing, R., Nichol, S. T., Ksiazek, T. G., & Rollin, P. E. (2010). Proportion of deaths and clinical features in Bundibugyo Ebola virus infection, Uganda. *Emerging infectious diseases*, 16(12), 1969–1972.
- [30]. Xavier, Analúcia R. et al. Clinical and laboratory diagnosis of Zika fever: an update. *Jornal Brasileiro de Patologia e Medicina Laboratorial* [online]. 2017, v. 53, n. 4 [Accessed 30 April 2022] , pp. 252-257.
- [31]. Rudolph KE, Lessler J, Moloney RM, Kmush B, Cummings DA. Incubation periods of mosquito-borne viral infections: a systematic review. *Am J Trop Med Hyg*. 2014 May; 90(5): 882-91.
- [32]. Lanciotti RS, Kosoy OL, Laven JJ, et al. Genetic and serologic properties of Zika virus associated with an epidemic, Yap State, Micronesia, 2007. 14(8): 1232-9.
- [33]. Ogbaini-Emovon, E & Ogbaini-Emovon, Dr. (2009). Current Trends In The Laboratory Diagnosis Of Tuberculosis. *Benin Journal of Postgraduate Medicine*. 11.
- [34]. In 1995, the amplified mycobacterium direct test (AMTD) (Ho-logic, San Diego, CA) was the first nucleic acid-based amplification test (NAAT) to be cleared by the FDA for the detection and identification of *M. tuberculosis* from direct specimens. This assay utilizes transcription-mediated amplification of a portion of the 16S rRNA gene specific to the *M. tuberculosis* complex to identify the organism.
- [35]. Dunn, J. J., Starke, J. R., & Revell, P. A. (2016). Laboratory Diagnosis of Mycobacterium tuberculosis Infection and Disease in Children. *Journal of clinical microbiology*, 54(6), 1434–1441.
- [36]. Carlos Rodrigo & Maria Méndez (2012) Clinical and laboratory diagnosis of influenza, *Human Vaccines & Immunotherapeutics*, 8:1, 29-33,
- [37]. Peltola V, Reunanen T, Ziegler T, Silvennoinen H, Heikkinen T. Accuracy of clinical diagnosis of influenza in outpatients children. *Clin Infect Dis* 2005; 41:1198-200.
- [38]. Leonardi GP, Mitrache I, Pigal A, Freedman L. Public hospital-based laboratory experience during an outbreak of pandemic influenza A (H1N1) virus infections. *J Clin Microbiol* 2010; 48:1189-94
- [39]. Muranyi, W., Bahr, U., Zeier, M., & van der Woude, F. J. (2005). Hantavirus infection. *Journal of the American Society of Nephrology : JASN*, 16(12), 3669–3679.
- [40]. Deutz A, Fuchs K, Schuller W, Nowotny N, Auer H, Aspöck H, Stunzner D, Kerbl U, Klement C, Kofer J: Seroepidemiological studies of zoonotic infections in hunters in southeastern Austria—Prevalences, risk factors, and preventive methods. *Berl Munch Tierarztl Wochenschr* 116: 306–311, 2003
- [41]. Beers MH, Berkow R: Infectious diseases; Viral diseases. In: *The Merck Manual of Diagnosis and Therapy*, 17th Ed., Indianapolis, Wiley Publishers, 20
- [42]. Aitichou M, Saleh SS, McElroy AK, Schmaljohn C, Ibrahim MS: Identification of Dobrava, Hantaan, Seoul, and Puumala viruses by one-step real-time RT-PCR. *J Virol Methods* 124: 21–26, 2005

[43]. Smithburn KC, Hughes TP, Burke AW, Paul JH. Neurotropic virus isolated from blood of native of Uganda. *Am J Trop Med.* 1940;20: 471-492.

[44]. Sampathkumar P. (2003). West Nile virus: epidemiology, clinical presentation, diagnosis, and prevention. *Mayo Clinic proceedings*, 78(9), 1137–1144.

[45]. Lanciotti RS, Kerst AJ, Nasci RS, et al. Rapid detection of West Nile virus from human clinical specimens, field-collected mosquitoes, and avian samples by a TaqMan reverse transcriptase-PCR assay. *J Clin Microbiol.* 2000;38:4066-4071

[46]. Ladnyj ID, Ziegler P, Kima E. A human infection caused by monkeypox virus in Basankusu Territory, Democratic Republic of the Congo. *Bull World Health Organ* 1972; 46:593–7.

[47]. Damon IK. Status of human monkeypox: clinical disease, epidemiology and research. *Vaccine* 2011; 29(suppl 4): D54–9.

[48]. Di Giulio DB, Eckburg PB. Human monkeypox: an emerging zoonosis. *Lancet Infect Dis* 2004; 4:15–25.

[49]. Jezek Z, Szczeniowski M, Paluku KM, Mutombo M. Human monkeypox: clinical features of 282 patients. *J Infect Dis* 1987; 156: 293–8.

[50]. Jezek Z, Fenner F. *Human monkeypox*. New York: Karger, 1988.

[51]. McCollum, A. M., & Damon, I. K. (2014). Human monkeypox. *Clinical infectious diseases : an official publication of the Infectious Diseases Society of America*, 58(2), 260–267.

