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Several Parasitic Diseases Occasionally Affect the Heart in Childhood

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ABSTRACT
Background/aim: Several parasitic diseases occasionally affect the heart, causing myocarditis, cardiomyopathy and pericarditis. Heart defects usually develop while a baby is still in the uterus. About a month after conception, the heart begins to develop. Pediatric myocarditis is an inflammatory condition of the heart muscle in infants and toddlers, it is uncommon and infrequent in young children due to parasitic diseases. It is more likely to occur in older children and adults. A tendency to be more extreme in newborns and young infants than in children over age two. Symtoms start gentle at first and difficult to recognize, yet show up abruptly in babies and newborn children. Pediatric myocarditis can be difficult to recognize and diganose in light of the fact that the signs and indications frequently impersonate those of other heart and lung infections. A quick pulse , rapid heart beat and unusual aberrant heart sounds with a stethoscope. A physical examination may distinguish liquid in the lungs , neoplasm and swelling in the legs of older children . There might be signs and indications of infection incidence , including fever and rashes. Babies have the most noteworthy hazard for genuine ailment and inconveniences. Newborns have the highest risk for serious disease and complications (including death). In rare cases, harm to the heart muscle is so serious and severe that a heart transplant is needed. Cardiomyopathy is a myocardium's disease mainly associate with heart impairment and dysfunction. Parasites damage to the heart mostly affect either the aortic valve or the mitral valve. Damage of the heart valves causes skipped postulates , irregular pulses, angina, breath shortness, upper back and neck pain. A chest x-ray show enlargement of the heart
Conclusion: The protozoan parasite, Entamoebahistolytica rarely causes a pericarditis while Toxoplasma gondii may cause myocarditis, usually in immunosuppressed hosts. American and African trypanosomiasis causes myocarditis while chagas'disease causes cardiomyopathy in child up to 2 years, but may advance to serious heart failure condition that requires a cardiologist urgent consultation
Keywords: Parasitic diseases, myocarditis, pericarditis, pancarditis, heart disease, heart failure

I. INTRODUCTION

Parasitic infection diseases may from time to time affect the cardiovascular system, albeit seldom in childhood. Several parasitic diseases occasionally affect the heart, causing myocarditis, cardiomyopathy and pericarditis as follows[1]

(Table1)

Myocarditis	*Trypanosoma cruzi* *Trypanosoma brucei* *Toxocara canis, Toxocara cati* *Toxoplasma gondii* *Trichinella spiralis* *Entamoeba histolytica* *Echinococcus granulosus*	American trypanosomiasis [2] African trypanosomiasis [2] Visceral larva migrans [10] Toxoplasmosis [3] Trichinellosis [4] Amoebiasis [5] Echinococcosis [8]
Cardiomyopathy	*Trypanosoma cruzi* *Toxoplasma gondii*	Chages' disease [6] Toxoplasmosis [7]
Pericarditis	*Entamoeba histolytica* *Trypanosoma brucei* *Trypanosoma cruzi* *Toxoplasma gondii* *Echinococcus granulosus*	Amoebiasis [5] African trypanosomiasis [2] Chages' disease [9] Toxoplasmosis [7] Echinococcosis [8]

Parasitic infections previously seen only in developing tropical settings can be presently diagnosed worldwide due to travel and population migration.

Some parasites may specifically, straightforwardly or indirectly influence and affect different anatomical structures of the heart, with severe infections known as myocarditis, pericarditis, pancarditis or pulmonary hypertension[11] The protozoan parasite, *Entamoeba histolytica* rarely causes a pericarditis while *Toxoplasma gondii* may cause myocarditis, usually in immunocompromisedhosts .[12] parasites cause psoriasis, heart problems, muscle weakness, constipation, diarrhea, floaters and swimming in the eyes, depression, allergies and excessive appetite.

The human heart parasites and parasites in the blood are the most serious parasitic infections which are those involving the blood vascular and lymphatic system due to Heart failure . Heart dysfunction may result from stiffen of heart's walls due to infiltrations and infections. For example, in amyloidosis, amyloid, and abnormal protein not usually or normally present in the body. If amyloid infiltrates by particular parasites in muscle of the heart's walls, they stiffen, and heart impairment occurs.

Heart Parasitic infections diagnosed in developed countries due to perpetual worldwide travel, blood transfusions, and increasing numbers of immunosuppression states such as organ transplantation, use of immunocompromise agents, or HIV/AIDS [13] Parasitic harm of the heart most often effects either the aortic valve or the mitral valve, which causes skipped beats, irregular beats, rapid beats, angina, breath shortness, chest pains, arm pain, upper back and neck pain.

Cardiovascular disease involves contracted narrowed or blocked blood veins that can promot to a heart attack, chest pain (angina) or stroke.[14] Heart defects usually develop while a baby is still in the uterus , about a month after conception. It's at this point that heart defects can begin to form. Some medical conditions, medications ,parasitic infection (*Toxoplasma gondii*)and genes may contribute to heart defects.

II. MYOCARDITIS

Myocarditis is an inflammation of the muscular walls of the heart. Myocarditis commonly brings about poor heart function. There are causes by infections, prescriptions, chemicals, radiation, and certain inflammatory diseases in many different organs of the body. Myocarditis in most children is induced and triggered by an infection,mostly viral infections that involves the heart. [15]. There are no known risk factors for developing myocarditis. The seriousness and severity of illness is by all accounts subordinate upon many variables, such as , age, sex, and the hereditary make-up of the immune system. Treatment for myocarditis is advancing as the illness procedure is better understood.

Numerous children encounter an entire recuperation, yet some may create genuine heart dysfunction and require unending consideration by a cardiologist. Children aged over 2 years may be less symptomatic than newborns and infants who are usually more severely affected. This is thought to be due to the immaturity of a baby's immune system. Older children may be less symptomatic.

They may gripe of an influenza like sickness comprising of weakness, malaise and fever. Pediatric myocarditis can be difficult to recognize and diagnose in light of the fact that the signs and symptoms frequently copy . The specialist doctor may hear a quick pulse or unusual heart sounds while listening to the child’s chest with a stethoscope. A physical examination may distinguish liquid in the lungs in older kids. Pediatric myocarditis is an inflammed heart muscles in infants and it’s uncommon in young children but more frequent in older children and adults . It has a tendency to be more extreme in newborns, young infants than in children aged over 2. There is no particular specific test for myocarditis detection . It is mostly a clinical diagnosis; therefore the physician must depend on the medical history profile provided by the family and physical examination of the child.

Diagnosis of Myocarditis : chest X-ray, electrocardiogram, echocardiogram, cardiac catheterization. In some rare cases, a person may even develop heart failure[16]. Prognosis: child’s recovery from myocarditis relies on the cause and his or her overall health. Newborns have the highest affinity for serious disease progress and complications advance, that may ultimately leads to death because of myocarditis. In uncommon cases, harm of the heart muscle is severe to the point that a heart transplant is required.

III. CARDIOMYOPATHIES

Cardiomyopathy : the heart muscle becomes enlarged, thick, or rigid. In rare cases, the muscle tissue in the heart is replaced with scar tissue. It affects around 1 in 500 people of all ages and

usually treated successfully so most people lead a long life. In infants and kids under 16 years of age, cardiomyopathy is a main source of heart muscle inproper function and disorders of heart beat rhythm . The reason or cause of cardiomyopathy in children regularly stays obscure. These incorporate familial cardiomyopathies, inborn metabolism, mitochondrial, neuromuscular disorders and malformation syndromes [17] Cardiomyopathy is a myocardium's disease associates with cardiac dysfunction[18].

Pericarditis

The term pericarditis refers to inflammation of the pericardium[19]. The pericardium is the membrane enclosing the heart, consisting of an outer fibrous layer and an inner double layer of serous membrane.

In children, pericarditis is well on the way to happen taking after surgery to repair congenital heart defects and disorders or gained acquired heart disease. Notwithstanding, different causes may incorporate the accompanying: Infection (viral, bacterial, fungal parasitic), chest injury , chest trauma and disorders of the connective tissue issue such as systemic lupus erythematosus (lupus).

Children will most likely be unable to portray that they have "chest pain" or have the capacity to clarify precisely how they feel. The symptoms and signs of pericarditis may resemble other medical conditions or heart problems. Always consult your child's health care provider for a diagnosis. Parasitic causes of pericarditis is a condition in which the sac that covers the heart becomes inflamed, irritated ,swollen and painful caused by a parasite infection.

A dull pain that begins in the front of the thorax under the breastbone and emanates to left side of the neck, upper stomach area, and left shoulder. The pain is less extreme when the patient sits up or inclines forward and intensifies when lying down. The torment may intensify with a full deep breath, similar to pleurisy, which may accompany pericarditis [20].

Symptoms of Pericarditis :chest pain associates with: rapid breathing ,breathlessness , dry cough, fever and chills , Weakness , hemorrhage of mucus membranes of the eyes, the back, the chest and feelings of anxiety. Pericarditis Diagnosed By Blood tests, Chest x-ray to show the traditional "water bottle" shadow around the heart , Echocardiography (echo) and Electrocardiogram (ECG) to discriminate between pericarditis and a heart attack .

Computed tomography scan(CT scan),Heart catheterization for the heart's chambers and valves viewing , Pericardiocentesis for viruses, bacteria, fungus, cancer, and tuberculosis testing. Blood tests such as LDH and CPK to measure cardiac enzymes, complete blood count (CBC) to look for infection [21] .

Spots on some parasites affecting on heart

Chagas disease, also known as American trypanosomiasis, is a tropical parasitic disease caused by the protozoan *Trypanosoma cruzi* [22] The most perceived marker of acute Chagas sickness is called Romaña's sign that includes eyelids swelling near the bit wound or where the bug feces deposition accidently rubbed into the eye.

Rarely, young children, or adults may die from the acute disease due to severe inflammation/infection of the heart muscle (myocarditis) or brain (meningoencephalitis)[23] . intracellular amastigotes destroy the intramural neurons of the autonomic nervous system in the intestine and heart, leading to megaintestine and heart aneurysms, In the event that left untreated, Chagas infection can be lethal and fatal much of the time because of heart muscle damage[24].

Chronic heart disease caused by Chagas disease is now a common reason for heart transplantation surgery. Up to this point, in any case, Chagas sickness was viewed as a contraindication for the procedure, since the heart harm could repeat as the parasite was predicted to grab and seize the open door gave by the immunosuppression that takes after surgery [25].

Toxoplasma gondii is an obligate intracellular, parasitic protozoan that causes the disease toxoplasmosis [26]. For the most part if a women has been infected and invaded before getting to be distinctly pregnant, the unborn child will be secured on the grounds that the mother has developed invulnerability.

On the off chance that a women is pregnant and turns out to be recently infected with Toxoplasma amid or just before pregnancy, she can pass the contamination to her unborn baby (congenital transmission). The harm to the unborn child is regularly more serious the prior in pregnancy the transmission happens. Potential outcomes can be an unnatural birth cycle, a stillborn child, a child born with toxoplasmosis signs (e.g., aberrant abnormal enlargement of the head or smallness of it) Infants infected prior to birth regularly demonstrate no symptoms at birth. However may develop them further down later with potential vision loss, mental disability, and seizures. Pregnant women, newborns, and infants can be treated, although the parasite is not eliminated completely.

The parasites can stay and remain inside tissue cells in a, quiescent, less dynamic inactive stage; their area makes it troublesome for drugs and medications to totally eliminate or eradicate them. Albendazole is contraindicated during pregnancy and children under two years of age [27][28]Corticosteroidscan treat some of the symptoms, such as inflammation.

Echinococcus granulosus, also called the hydatid worm, hyper tape-worm or dog tapeworm, is a cyclophyllidcestode that parasitizes the small intestine of canids as an adult and has important intermediate hosts as humans, causeing cystic echinococcosis, also termed as hydatid disease.

In the human hydatid disease, *E. granulosus, E. multilocularis, E. oligarthrus and E. vogeli* are localized in the hepatocytes (in 75% of cases), the pulmonary cells (in 5–15% of cases) and other body organs (in 10–20% of cases). There are no human immunizations or vaccination trials against any form of echinococcosis. But further researchs and studies are currently running to come up with a possible and potential vaccine against echinococcosis[29].

Humans accidently handling dirts, hair animals and contaminated soil containing eggs become intermediate hosts [30]. The diagnosis can be done by aspiration of a cyst[31]. Picturing the brain with computer tomography (CT) or magnetic resonance imaging (MRI) are also useful diagnostic tool .

Another diagnostic indicator is an elevated number of eosinophils or Eosinophilia in the blood and spinal fluid [31]. Neurocysticercosis treatment may be with praziquantels or albendazoles for prolonged time. For anti-inflammation during treatment, steriods and anti-seizure medications are also required. Surgerical interference is sometimes required to remove the cysts[32].

Both cystic echinococcosis and alveolar echinococcosis are frequently costly and convoluted to treat, sometimes requiring potential surgery and/or lengthy medication treatment.

Trichinosis, trichinellosis or trichiniasis is a roundworm parasitic infection of the genus *Trichinella.* Trichinosis's infection was once extremely common, yet is presently uncommon in the developed world.

During 2008-2010, a yearly average of 20 cases for each year were accounted for in the United States[33]. Several subspecies are highly infectious causing human diseases, but *T. spiralis* is the most pathogenic [34]. Infection may take care without symptoms[34], while intestinal attack can bring diarrhea , vomiting and abdomen discomfort.[34]. One week after infection incidence, the larvae migrates and deposits in the muscular tissues causing edema of the face or around the eyes, conjunctivitis, fever and sever muscle pain and peripheral eosinophilia. Life threatening cases may progress to myocarditis and pneumonitis[34]. Muscular Larval encystment causes pain and weakness, accompanied by gradual symptoms progression[34].

Trichinosis infections have either minor or no symptoms and no complications[35]. There are two primary stages for the parasitic infection: enteral (influencing the intestines) and parenteral (not influencing or affecting the intestines). The indications fluctuate contingent upon the stage, types of Trichinella, measure and amount ingested encysted larvae, age, gender, and host immunity[35].

Primary treatment Early anthelmintics adminstration of mebendazole or albendazole diminishes the probability of larval encystation, especially if taken within three days of infection occurrence. [36].However, multiple cases are diagnosed after this time[36] Mebendazole (200–400 mg three doses each day for three days) or albendazole (400 mg two doses a day for 8–14 days) are given to treat trichinosis[37]. These medications keep recently hatched larvae from growing and developing, yet ought not be given to pregnant ladies or children beyond two years [37]. Secondary treatment prednisone steroids is used to alleviate musclar pain associated with larval migration.

Visceral larva migrans caused by the migratory larvae of certain nematodes in humans, first human death reported in 1952[38]. Nematodes causing such zoonotic infections are *Toxocara canis*[39],*Toxocara cati*[39]and Ascarissuum[40]. Affected organs are the liver, heart (causing myocarditis) and the CNS dysfunction, seizures, and coma. A unique variatiant is the visual ocular larvae where normally *T. canis* larvae go to the eye. visceral larva migrans appear to influence children aged 1–4 while ocular larva migrans are more habitually influences children aged 7-8 years old. In the vicinity of 4.6% and 23% of U.S infected children with dog roundworm infection. Such No. can vary to increase in different countries, such as Colombia where more than 81% of children infection incidence is reported [10].

Entamoeba histolytica is a protozoan intestinal parasite causing amoebiasis. It takes place frequently in the large intestine and causes internal inflammation (histo = tissue, lytic = destroying). worldwide, 50 million individuals are infected.

Higher ration noticed tropical countries of poor sanitation.

In industrialized countries most of the infected patients are immigrants, institutionalized people and those who have recently visited developing countries[41]. Most *Entamoeba histolytica* infections are asymptomatic and trophozoites remain in the intestinal lumen feeding on surrounding nutrients. [42] Minor infections (luminal amoebiasis) can cause symptoms that include: gas (flatulence), intermittent constipation, loose stools, stomach ache, stomach cramping.

Severe infection inflammation causes amoebic dysentery. The parasites penetrate the intestinal wall and travel to organs such as the liver via bloodstream causing extraintestinal amoebiasis. Symptoms of these more severe infections include: anemia, appendicitis (inflammation of the appendix), bloody diarrhea, fatigue, fever, gas (flatulence), genital and skin lesions, intermittent constipation, liver abscesses (can lead to death, if not treated), malnutrition, painful defecation (passage of the stool), peritonitis, pleuropulmonary abscesses, stomach ache, stomach cramping, toxic megacolon (dilated colon), and weight loss.[43] If you have amoebiasis symptoms, you'll be treated with two antibiotics.

The preferred drugs are metronidazole or tinidazole immediately followed with paromomycin, diloxanidefuroate or iodoquinol. Asymptomatic intestinal amoebiasis is treated with paromomycin, diloxanidefuroate or iodoquinol.

Summary

myocarditis, pericarditis and endocarditis heart infections are caused by an irritant invasion, such as a bacterium, virus or chemical, reaches to heart muscle.

The most frequent causes of heart infections include[44]: Bacteria, Viruses, Parasites, Medications that may cause an allergic or toxic reaction, Connective tissue disorders (Lupus), Vasculitis, and Wegener's granulomatosis. Symptoms at first are mild and hard to diagnose. But, in newborns and infants, symptoms may show up all of a sudden.

Such symptoms may include heart failure [45] :Anxiousness, Failure to thrive or poor weight gain, Feeding difficulties, Fever and other symptoms of infection, Listlessness, Low urine output (a sign of decreasing kidney function), Pale, cool hands and feet (a sign of poor circulation), Rapid breathing, and Rapid heart rate Symptoms in children over age 2 may also include: Belly area pain and nausea, Chest pain, Cough, Fatigue, and Swelling (edema) in the legs, feet, and face There may be other indications of infection, including fever and rashes. A chest x-ray can show enlargement (swelling) of the heart (Fig. 2)

Conclusion

The protozoan parasite, Entamoeba histolytica rarely causes a pericarditis while Toxoplasma gondii may cause myocarditis, usually in immunocompromised hosts. American and African trypanosomiasis causes myocarditis while chagas'disease causes cardiomyopathy in child up to 2 years. They need parasitic treatment besides cardiovascular treatment, because may develop serious heart failure need cardiologist.

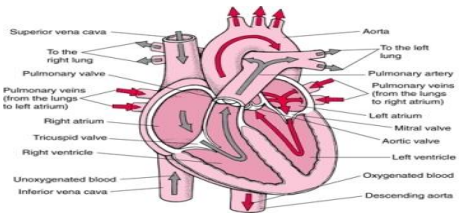

Fig. 1: Normal anatomy of heart

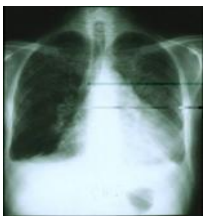

Fig. 2*: enlargement of cardiac silhouette (with pericardial effusion > 250 ml)*
http://cardiopedia.wdfiles.com/local--files/pericardial-effusion/Pericardial%20effusion%20xray.jpg

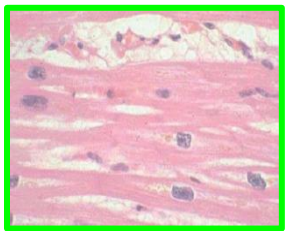

Fig. 3. : Histology of dilated cardiomyopathy. Nonspecific abnormalities, including variations inmyocyte size, myocyte vacuolation, loss of myofibrillar material, and fibrosis, can be seen (hematoxylin-eosin stain, x40). (Courtesy of N.G. Mahon, MD)

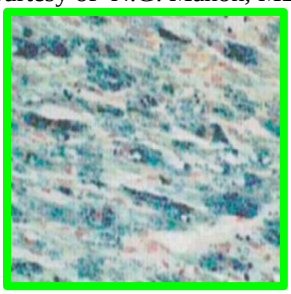

Fig. 4 : Histology of dilated cardiomyopathy. Nonspecific abnormalities, including variations inmyocyte size, myocyte vacuolation, loss of myofibrillar material, and fibrosis, can be seen (hematoxylin-eosin stain, x40). (Courtesy of N.G. Mahon, MD)

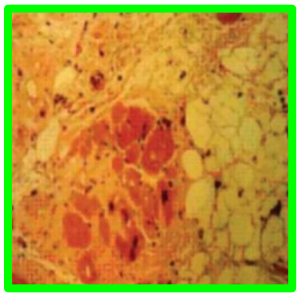

Fig. 5 : Histologic specimen of arrhythmogenic right ventricular cardiomyooooooooopathy. Marked fatty infiltration can be seen (Masson's trichrome stain, x 40). (Courtesy of N.G. Mahon, MD)

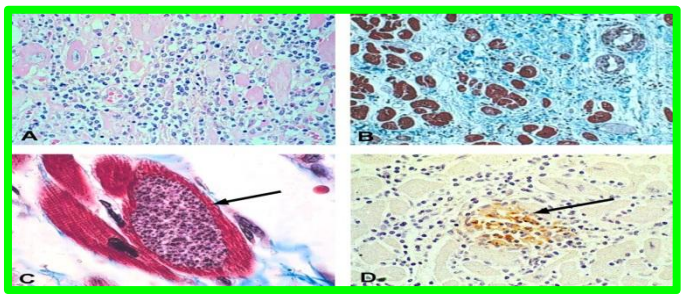

Fig. 6: Microscopic images demonstrating the main histopathological characteristics of chronic chagasic cardiopathy. (A) Severe myocarditis with many lymphocytes aggressing non-infected hypertrophic myocytes (H&E staining, objective magnification: 40 ×). (B) Diffuse myocardial fibrosis, involving each myocardial fiber and focal dense fibrosis associated with thickened arterioles, suggestive of ischemic injury (Masson trichrome staining, objective magnification: 20 ×). (C) A whole myocardial fiber containing a large pseudocyst of *T. cruzi* (arrow), that did not elicit inflammatory reaction (Masson trichrome staining, objective magnification: 100 ×). (D) Macrophages containing antigens of *T. cruzi* (arrow) in the middle of a granulomatous arrangement (immunoperoxidase technique, objective magnification: 40 ×).

REFERENCES

[1]. Gilles H. , reviow article for parasitic tropical fevers, in PMC . images paediatric cardiology. 2000 Oct-Dec 2 (4): 28-39
[2]. Aerts, D., P. Truc, L. Penchenier, Y. Claes, and D. Le Ray. 1992. A kit for in vitro isolation of trypanosomes in the field: first trial with sleeping sickness patients in the Congo Republic. Trans. R. Soc. Trop. Med. Hyg. 86:394-395.
[3]. Adair, O. V., N. Randive, and N. Krasnow. 1989. Isolated toxoplasma myocarditis in acquired immune deficiency syndrome. Am. Heart J. 118:856-857.
[4]. Ancelle, T., A. De Bruyne, D. Poisson, and J. Dupouy-Camet. 2005. Outbreak of trichinellosis due to consumption of bear meat from Canada, France, September 2005. Euro Surveill. 10:E051013.3.
[5]. Abd-Alla, M. D., T. F. Jackson, V. Gathiram, A. M. el-Hawey, and J. I. Ravdin. 1993. Differentiation of pathogenic Entamoeba histolytica infections from nonpathogenic infections by detection of galactose-inhibitable adherence protein antigen in sera and feces. J. Clin. Microbiol. 31:2845-2850.
[6]. Acquatella, H. 2007. Echocardiography in Chagas heart disease. Circulation 115:1124-1131.
[7]. Angel, S. O., M. Matrajt, J. Margarit, M. Nigro, E. Illescas, V. Pszenny, M. R. Amendoeira, E. Guarnera, and J. C. Garberi. 1997. Screening for active toxoplasmosis in patients by DNA hybridization with the ABGTg7 probe in blood samples. J. Clin. Microbiol. 35:591-595.
[8]. Ammann, R. W., and J. Eckert. 1996. Cestodes. Echinococcus. Gastroenterol. Clin. North Am. 25:655-689
[9]. Bern, C., and S. P. Montgomery. 2009. An estimate of the burden of Chagas disease in the United States. Clin. Infect. Dis. 49:e52-e54.
[10]. Sakai, S.; Shida, Y.; Takahashi, N.; Yabuuchi, H.; Soeda, H.; Okafuji, T.; Hatakenaka, M.; Honda, H. (2006). "Pulmonary Lesions Associated with Visceral Larva Migrans Due to Ascaris suum or Toxocara canis: Imaging of Six Cases". American Journal of Roentgenology. 186 (6): 1697–1702. doi:10.2214/AJR.04.1507. PMID 16714661.
[11]. Kobayashi D, Aggarwal S, Kheiwa A, Shah N. Myopericarditis in children: elevated troponin I level does not predict outcome. Pediatr Cardiol. 2012 Oct. 33(7):1040-5.)
[12]. Bastien, P. 2002. Molecular diagnosis of toxoplasmosis. Trans. R. Soc. Trop. Med. Hyg. 96(Suppl. 1):S205-S215.
[13]. Politi Okoshi, M., and M. Rubens Montenegro. 1996. Pathology of the heart in AIDS. Study of 73 consecutive necropsies. Arq. Bras. Cardiol. 66:129-133.
[14]. Wynne J, Braunwald E. The cardiomyopathies and myocarditides. In: Braunwald E, Zipes DP, Libby P, eds. Heart Disease: A Textbook of Cardiovascular Medicine. Philadelphia: Saunders; 2001:1751–1806.
[15]. Kawai C. From myocarditis to cardiomyopathy: mechanisms of inflammation and cell death. Learning from the past for the future. Circulation. 1999;99:1091–1100.
[16]. [a b c d e] Imazio, M; Gaita, F; LeWinter, M (13 October 2015). "Evaluation and Treatment of Pericarditis: A Systematic Review.". JAMA. **314** (14): 1498–506. doi:10.1001/jama.2015.12763. PMID 26461998.
[17]. Dr Guiseppe Limongelli, assistant professor of cardiology, Monaldi Hospital, Naples, Italy).
[18]. Troughton RW, Asher CR, Klein AL (February 2004). "Pericarditis". Lancet. **363** (9410): 717–27. doi:10.1016/S0140-6736(04)15648-1. PMID 15001332.
[19]. Maisch B, Seferović PM, Ristić AD, et al. (April 2004). "Guidelines on the diagnosis and management of pericardial diseases executive summary; The Task force on the diagnosis and management of pericardial diseases of the European society of cardiology". Eur. Heart J. **25** (7): 587–610. doi:10.1016/j.ehj.2004.02.002. PMID 15120056.
[20]. McNamara DM, Starling RC, Dec GW, et al. Intervention in myocarditis and acute cardiomyopathy with immune globulin: results from the randomized placebo controlled IMAC trial. Circulation 1999;100(suppl I):I-21
[21]. Mason JW, O'Connell JB, Herskowitz A, et al. A clinical trial of immunosuppressive therapy for myocarditis. N Engl J Med. 1995;333:269–275.
[22]. [a b c d e f g h i] Louis V Kirchhoff (17 December 2010). "Chagas Disease (American Trypanosomiasis)". eMedicine. Retrieved 12 May 2010
[23]. Bocchi EA, Bellotti G, Mocelin AO (June 1996). "Heart transplantation for chronic

Chagas' heart disease". Ann Thorac Surg. **61** (6): 1727–33. doi:10.1016/0003-4975(96)00141-5. PMID 8651775.

[24]. https://en.wikipedia.org/wiki/Chagas_disease#

[25]. Villamil-Gómez WE, Echeverría LE, Ayala MS, Muñoz L, Mejía L, Eyes-Escalante M, et al. Orally transmitted acute Chagas disease in domestic travelers in Colombia. J Infect Public Health. 2016 May 30. [Medline].

[26]. Robertson LJ, Devleesschauwer B, Alarcón de Noya B, Noya González O, Torgerson PR. Trypanosoma cruzi: Time for International Recognition as a Foodborne Parasite. PLoS Negl Trop Dis. 2016 Jun. 10 (6):e0004656. [Medline].

[27]. Dardé, ML; Ajzenberg, D; Smith, J (2011). "3 – Population structure and epidemiology of Toxoplasma gondii". In Weiss, LM; Kim, K. Toxoplasma Gondii: The Model Apicomplexan. Perspectives and Methods. London: Academic Press/Elsevier. pp. 49–80. doi:10.1016/B978-012369542-0/50005-2. ISBN 978-0-12-369542-0.

[28]. Foulon W, Villena I, Stray-Pedersen B, et al. Treatment of toxoplasmosis during pregnancy: a multicenter study of impact on fetal transmission and children's sequelae at age 1 year. Am J Obstet Gynecol 1999;180:410-5.

[29]. Miro JM, Lopez JC, Podzamczer D, et al. Discontinuation of primary and secondary Toxoplasma gondii prophylaxis is safe in HIV-infected patients after immunological restoration with highly active antiretroviral therapy: results of an open, randomized, multicenter clinical trial. Clin Infect Dis 2006;43:79--89.

[30]. Dang Z, Yagi K, Oku Y, et al. (December 2009). "Evaluation of Echinococcus multilocularis tetraspanins as vaccine candidates against primary alveolar echinococcosis". Vaccine. **27** (52): 7339–45. doi:10.1016/j.vaccine.2009.09.045. PMID 19782112.

[31]. [a b c d e f g h i j] Eckert J, Deplazes P (January 2004). "Biological, epidemiological, and clinical aspects of echinococcosis, a zoonosis of increasing concern". Clin. Microbiol. Rev. **17** (1): 107–35. doi:10.1128/cmr.17.1.107-135.2004. PMC 321468. PMID 14726458.

[32]. "Global Plan to Combat Neglected Tropical Diseases 2008–2015" (PDF). World Health Organization. 2007. Box 1. Selected neglected tropical diseases and zoonoses to be addressed within the Global Plan. p. 2.

[33]. [a b c] Li, Jun; Wu, Chuanchuan; Wang, Hui; Liu, Huanyuan; Vuitton, Dominique A.; Wen, Hao; Zhang, Wenbao (2014). "Boiling sheep liver or lung for 30 minutes is necessary and sufficient to kill Echinococcus granulosus protoscoleces in hydatid cysts". Parasite. 21: 64. doi:10.1051/parasite/2014064. ISSN 1776-1042.

[34]. [a b c d e f g h i j k l m] "CDC - DPDx - Trichinellosis - index". www.cdc.gov. Retrieved 2015-07-19.

[35]. [a b c d e f] Capo, V. & Despommier, D. D. (1996). Clinical Aspects of Infection with Trichinella spp. Clinical Microbiology Reviews, 9, 47–54.

[36]. [a b] John D and William A. Petri. Markell and Voge's Medical Parasitology. 9th ed. Philadelphia: Saunders, 2006

[37]. [a b c d e f g h i j k l m n] Gottstein B; et al. (2009). "Epidemiology, Diagnosis, Treatment, and Control of Trichinellosis". Clinical Microbiology Reviews. **22** (1): 127–145. doi:10.1128/cmr.00026-08.

[38]. "Monograph – Mebendazole". medscape.com. Retrieved 2009-02-24.

[39]. Beaver, P. C.; Snyder, C. H.; Carrera, G. M.; Dent, J. H.; Lafferty, J. W. (1952). "Chronic eosinophilia due to visceral larva migrans; report of three cases". Pediatrics. **9** (1): 7–19. PMID 14911260.

[40]. Gavin, P. J.; Kazacos, K. R.; Shulman, S. T. (2005). "Baylisascariasis". Clinical Microbiology Reviews. **18** (4): 703–18. doi:10.1128/CMR.18.4.703-718.2005. PMC 1265913 PMID 16223954.

[41]. [a b] Beaver, PC (1959). "Visceral and cutaneous larva migrans". Public Health Reports. **74** (4): 328–32. doi:10.2307/4590442. PMC 1929226. PMID 13645880.

[42]. Rashidul Haque,1 Dinesh Mondal,1 Priya Duggal,2 et al. 2006 Entamoeba histolytica Infection in Children and Protection from Subsequent Amebiasis Infect Immun. 74(2): 904–909.

[43]. Blessman, J., I. K. M. Ali, P. A. Ton Nu, et al. 2003. Longitudinal study of intestinal Entamoeba histolytica infections in asymptomatic adult carriers. J. Clin. Microbiol. 41:4745-4750. [PMC free article][PubMed]

[44]. http://www.parasitesinhumans.org/entamoeba-histolytica-amoebiasis.html

[45]. Feldman AM, McNamara D. Myocarditis. N Engl J Med. 2000;19:1388–1398.

[46]. Aretz HT, Billingham ME, Edwards WD, et al. Myocarditis. Am J Cardiovasc Pathol. 1986;1:3–14.

[47].