DOWNLOAD PDF TARANTA RHEUMATIC FEVER

Chapter 1 : Rheumatic fever - Symptoms and causes - Mayo Clinic

Today rheumatic fever is still the most common cause of heart disease in children and young adults in developing countries. This disease is typically associated with poverty, in particular with poor housing, overcrowding and inadequate medical care.

This article has been cited by other articles in PMC. Abstract Rheumatic heart disease RHD is a disease of poverty, is almost entirely preventable, and is the most common cardiovascular disease worldwide in those under 25 years. RHD is caused by acute rheumatic fever ARF which typically results in cumulative valvular lesions that may present clinically after a number of years of subclinical disease. Therapeutic interventions, therefore, typically focus on preventing subsequent ARF episodes with penicillin prophylaxis. However, not all patients with ARF develop symptoms and not all symptomatic cases present to a physician or are correctly diagnosed. Therefore, if we hope to control ARF and RHD at the population level, we need a more reliable discriminator of subclinical disease. Recent studies have examined the utility of echocardiographic screening, which is far superior to auscultation at detecting RHD. However, there are many concerns surrounding this approach. Despite the introduction of the World Heart Federation diagnostic criteria in , we still do not really know what constitutes the most subtle changes of RHD by echocardiography. This poses serious problems regarding whom to treat and what to do with the rest, both important decisions with widespread implications for already stretched health-care systems. As a physical manifestation of poverty, children are particularly vulnerable and hard hit. Worldwide, more than three-quarters of those aged 15 years and younger live in high-prevalence regions,[2] with RHD accounting for the greatest cardiovascular-related loss of disability-adjusted life-years among 10â€”year olds We also relied on our familiarity with key literature. Pertinent review articles, book chapters, proceedings, and papers older than 20 years were used when judged important. ARF typically affects children of school-going age with a peak prevalence in the 5â€”14 years age group, and significant valvular damage is thought to accrue due to repeated episodes of ARF. However, nonspecific functional lesions such as mitral regurgitation MR and aortic regurgitation AR are frequently seen. Given the cumulative nature of valvular damage, classical thinking has been that a substantial period of disease latency of up to 20â€”30 years needs to be present from the initial ARF episode to clinically symptomatic RHD. This is exemplified by the fact that RHD often presents with moderate-to-severe multivalvular disease Primordial prophylaxis strategies to avoid GAS infection, e. A strategy of secondary prevention relies entirely on case detection and a successful therapeutic strategy. Therefore, if we hope to bridge the gap between the large number of incident RHD cases and the smaller number of patients who present with ARF there is a fold difference in endemic areas ,[7] we need a more robust strategy for detecting early RHD. Auscultation for a pathological murmur has been the traditional approach to screening school-aged children for RHD. However, it is neither sensitive[23] nor specific[24 , 25] as demonstrated in the seminal paper by Marijon et al. Furthermore, these guidelines were not universally accepted and many countries used alternative sometimes local guidelines for RHD diagnosis,[24 , 26 , 27 , 34 , 35 , 36 , 37] inadvertently resulting in a diagnostic potpourri that seriously undermined the validity and interchangeability of data from different countries. Inevitably, concerns began to emerge regarding diagnostic specificity: The World Heart Federation WHF criteria[38] [Box 1] were written to meet these needs and defined the lower limit of what constitutes RHD by echocardiography although this is highly debatable-see later. Auscultation is no longer required, and the guidelines are intended for screening patients with no history of ARF who live in endemic regions. The first two CoE criteria are met unequivocally: The gaps in our current state of knowledge and how this relates to the remaining three criteria are discussed here. Box 2 Open in a separate window Some general questions, such as the ideal screening age, also remain unanswered and lack concensus in the literature. It has been suggested that screening older age groups to include young adults and pregnant woman would increase pick-up rates of RHD and improve echocardiographic detection of disease. However, it is important to remember that RHD screening in its current form with echocardiographic case detection and the institution of secondary prophylaxis aims to prevent ARF recurrences rather than diagnose RHD per se. This explains the

rationale for screening children rather than adults as the rate of ARF recurrence in the latter group is very low. Most guidelines support this point of view with recommendations to stop prophylaxis at the age of 18â€”21 years in individuals with mild valve involvement and without excessive risk. The ideal timing for screening has to carefully balance picking up more cases by screening later with picking up less cases by screening as early as possible to allow maximal time for prophylaxis to make a difference. Cost-effectiveness of RHD screening It remains to be determined if echocardiography screening is cost-effective. The decisions regarding how and where limited resources should be focused in developing countries is an important ethical question[45] and the decision regarding whether to invest in echocardiographic RHD screening programs at the expense of other, possibly more robust evidence-based interventions for other conditions, warrants due consideration. The use and delivery of secondary prophylaxis Secondary penicillin prophylaxis intramuscular BPG is superior to oral penicillin plays an important role in preventing ARF recurrences[46] and in doing so reduces the severity of RHD by slowing, stopping, or regressing valvular disease[47 , 48] CoE criterion 4. In those with mild disease treated with penicillin prophylaxis, for example, the vast majority will have no detectable disease 5â€”10 years later. It has also yet to be proven that diagnosing subclinical RHD and instituting prophylaxis through a screening program will lead to better outcomes compared with intervention when the disease becomes clinically symptomatic CoE criterion 5. They are expensive, cumbersome to transport, and have limited battery capacity. Handheld HAND devices help address these issues although their limited functionality e. It is now possible to attach probes to smart devices which should help improve portability and reduce costs. There is no perfect diagnostic test for rheumatic heart disease Although echocardiography has become the gold standard for RHD diagnosis, it relies on criteria that must balance sensitivity and specificity and as such invariably remains imperfect at diagnostic categorization CoE criterion 3. In particular, detecting early valvular lesions that have no diagnostic or prognostic precedent raises many questions including that we still do not truly know what constitutes the lower limit of RHD the earliest or slightest changes recognizable as being due to RHD by echocardiography. Part of the problem here is that RHD encompasses pathological changes that exist on a continuum, and delineating the transition point that separates mild disease from a normal variant can be very difficult. Although echocardiography is presently the most discriminating tool, a deeper understanding of the disease mechanisms that underlie morphological changes will no doubt facilitate a more rational diagnostic criteria. A false-positive diagnosis will expose the patient to inappropriate and lengthy treatment usually 10 years or longer , potentially create psychological harm and stigmatization by association with a disease there is even evidence that echocardiographic RHD screening alone lowers quality of life scores for both the caregiver and screened child ,[69] and unnecessarily add to the financial and manpower burden of the already stretched healthcare systems of many developing countries. Conversely, a false-negative result risks missing the opportunity to prevent a potentially fatal disease. The significance of borderline rheumatic heart disease Understanding the natural history of borderline RHD in particular is crucial because screening studies tend to uncover a burden of disease that is double or more that of definite RHD. However, one-third of these children were receiving secondary prophylaxis, which may have altered the natural course of the disease. They also demonstrated that patients with nonspecific valvular abnormalities e. These findings again bring into focus the questions regarding the definition of what constitutes the lower limit of RHD by echocardiography and also raises the issue of how best to manage these patients, whether to treat with secondary prophylaxis, opt for enhanced surveillance or repeat echocardiography, decisions that will have potentially significant implications for already stretched health-care systems. Another recent study,[73] again using WHF criteria, re-examined 44 South African patients with borderline or definite RHD around 5 years after the initial diagnosis. Half of the participants In this series, only two patients 4. Slightly earlier studies from Nicaragua,[27] India,[28 , 74] Uganda,[75] and New Caledonia[76] also detail the natural history of borderline RHD. However, the studies from Nicaragua and India used nonstandardized diagnostic criteria which are associated with widely varying estimates of the prevalence of RHD and many of these studies reported variable use of secondary prophylaxis, again potentially altering the course of the disease. One has to question the mechanism of improvement of rheumatic involvement in all these studies and more work is undoubtedly required to tease out true disease

improvement from the known measurement variability of mild or subclinical disease. This is an issue because borderline RHD often encompasses minor heart valve abnormalities that are open to subjective assessment e. Finally, researchers who screened low- and high-risk Australian children for RHD found that high-risk Australians were 3. Simplifying the World Heart Federation criteria for rheumatic heart disease screening programs Implementing the WHF criteria is time-consuming, potentially complex, requires highly trained operators,[38 , 53] and may be impractical for in-field application. Therefore, the development of a uniform, simplified criteria with acceptable sensitivity and specificity that allows for a single-stage screening process by nonexperts using HAND, and which can be implemented within a preexisting health care program would significantly improve feasibility. This may circumvent some of the criticism regarding simplification of the criteria and echocardiographic interpretation. Removing the morphological criteria entirely and simplifying the functional criteria usually measuring MR jet length only is a strategy that researchers have recently begun to employ, with HAND devices being increasingly used for this purpose. A minimally defined jet length is used as a marker of pathological MR and by extension presence of RHD. A reasonable but unproven assumption here is the rationale that criteria-defined pathological MR found during screening of high-risk RHD populations is more likely to represent RHD than either normal variants or other pathologies. Possible problems with this simplified approach are that MR has many causes and we are removing the morphological features meant to add specificity, hoping that from a screening perspective, we maintain enough sensitivity to include all possible RHD cases. It is, however, unclear at this stage that the group with isolated morphological change is insignificant and can be ignored. Since , several studies[70 , 81 , 82 , 83] have examined the performance of MR jet length as the single echocardiographic criterion against a reference approach [Table 1]. Sensitivity for all disease i. Sensitivity for definite RHD was much better, ranging between Table 1 Summary of recent screening studies examining the sensitivity and specificity of simplified diagnostic criteria when compared to the reference approach images obtained using standard portable echocardiography and interpreted by experienced cardiologists with expertise in rheumatic heart disease using the full World Heart Federation criteria Open in a separate window One has to be mindful of the figures in all these studies looking at MR as a single criterion. The gold standard WHF criteria used to define what constitutes RHD in these studies required significant MR as an important diagnostic ingredient for the most common lesion. This risks introducing an important bias into these analyses which become a self-fulfilling prophecy. However, jet length measurement is quick and reproducible, which suits the requirements of large-volume screening programs and thus remains an important avenue to explore. Cardiology practice guidelines place more importance on the proximal jet width assessment vena contracta than length assessment when assessing MR severity. The latter, as a measured marker of MR severity, has all but disappeared from recent guidelines due to its known variability with technical factors e. However, with suboptimal specificity rates, this single criterion may require modification or risk over-treatment. Alternatively, a HAND-positive patient could undergo confirmatory testing with STAND which, although not a flawless approach, will still reduce the number of in-depth echocardiograms that need to be performed. Moreover, when compared to auscultation alone, the case for HAND is very powerful, even if it missed almost one-third of borderline RHD in the Godown study: In developing countries ravaged by disease, some intervention, one could argue, is better than none. The obvious problem here is that all isolated morphological deficits, even if relatively gross, would be missed by necessity. This again addresses the less obvious sensitivity issues with such a simplified approach. Take the example of a patient with a valve area reduced by half due to rheumatic commissural fusion. Such a valve area may still be above the 2. The importance of functional versus morphological aspects of the criteria must be weighed carefully and future recommendations based on study evidence. Task-shifting in rheumatic heart disease screening Task-shifting i. However, while reducing costs and freeing physicians to perform other tasks,[90] it may actually require additional resources for successful implementation, particularly in the short term. Studies examining task-shifting using nurses, having previously received focused echocardiographic training, were first conducted in [Table 1]. Overall sensitivity for all disease ranged from Again, sensitivity for definite RHD was high at However, the amount of echocardiographic experience of the nurses before training for the studies varied significantly in some cases. Medical students may also make suitable candidates

for task-shifting in some developing countries. One possible option for standardized training is an international team of trainers that could act as accredited instructors, implementing these training protocols with competency testing. A cheaper alternative and which may reach a wider audience is web-based learning that is open to everyone such as that devised by Engelman et al. The WHO guidelines also recommend continuous monitoring and evaluation as vital components of the task-shifting process. Effective strategies that encourage the regular uptake of secondary prophylaxis, a deeper understanding of the natural history of subclinical RHD and its response to penicillin prophylaxis, advancements in portable echocardiography, and a simplified criteria that is based on disease mechanisms, that can be applied by nonexperts, and adequately balances sensitivity and specificity, are desperately needed. Until then, routine widespread screening for RHD cannot be endorsed.

Chapter 2 : Rheumatic heart disease screening: Current concepts and challenges

acute attack acute rheumatic fever adults antigens aortic valve arthralgia Assoc ation attack of rheumatic attack rate bed rest benzathine penicillin cardiac carditis cause cells chronic Circulation clinical manifestations control of rheumatic cross-reaction decrease developing countries diagnosis of rheumatic dose effective Engl erythema.

Print Overview Rheumatic fever is an inflammatory disease that can develop as a complication of inadequately treated strep throat or scarlet fever. Strep throat and scarlet fever are caused by an infection with streptococcus bacteria. Rheumatic fever is most common in 5- to year-old children, though it can develop in younger children and adults. Although strep throat is common, rheumatic fever is rare in the United States and other developed countries. However, rheumatic fever remains common in many developing nations. Rheumatic fever can cause permanent damage to the heart, including damaged heart valves and heart failure. Treatments can reduce damage from inflammation, lessen pain and other symptoms, and prevent the recurrence of rheumatic fever. Rheumatic fever care at Mayo Clinic Symptoms Rheumatic fever symptoms vary. You can have few symptoms or several, and symptoms can change during the course of the disease. The onset of rheumatic fever usually occurs about two to four weeks after a strep throat infection. Rheumatic fever signs and symptoms â€" which result from inflammation in the heart, joints, skin or central nervous system â€" can include: Fever Painful and tender joints â€" most often in the knees, ankles, elbows and wrists Pain in one joint that migrates to another joint Red, hot or swollen joints Small, painless bumps nodules beneath the skin Chest pain Fatigue Flat or slightly raised, painless rash with a ragged edge erythema marginatum Jerky, uncontrollable body movements Sydenham chorea, or St. Proper treatment of strep can prevent rheumatic fever. Also, have your child see a doctor if he or she shows other indications of rheumatic fever. Request an Appointment at Mayo Clinic Causes Rheumatic fever can occur after an infection of the throat with a bacterium called group A streptococcus. Group A streptococcus infections of the throat cause strep throat or, less commonly, scarlet fever. Group A streptococcus infections of the skin or other parts of the body rarely trigger rheumatic fever. The strep bacterium contains a protein similar to one found in certain tissues of the body. This immune system reaction results in inflammation. Risk factors Factors that can increase the risk of rheumatic fever include: Some people carry a gene or genes that might make them more likely to develop rheumatic fever. Type of strep bacteria. Certain strains of strep bacteria are more likely to contribute to rheumatic fever than are other strains. A greater risk of rheumatic fever is associated with overcrowding, poor sanitation and other conditions that can easily result in the rapid transmission or multiple exposures to strep bacteria. Complications Inflammation caused by rheumatic fever can last a few weeks to several months. In some cases, the inflammation causes long-term complications. Rheumatic heart disease is permanent damage to the heart caused by rheumatic fever. It usually occurs 10 to 20 years after the original illness. Problems are most common with the valve between the two left chambers of the heart mitral valve , but the other valves can be affected. The damage can result in: This narrowing of the valve decreases blood flow. This leak in the valve allows blood to flow in the wrong direction. Damage to heart muscle. The inflammation associated with rheumatic fever can weaken the heart muscle, affecting its ability to pump. Damage to the mitral valve, other heart valves or other heart tissues can cause problems with the heart later in life. Resulting conditions can include: An irregular and chaotic beating of the upper chambers of the heart atrial fibrillation An inability of the heart to pump enough blood to the body heart failure Prevention The only way to prevent rheumatic fever is to treat strep throat infections or scarlet fever promptly with a full course of appropriate antibiotics.

Chapter 3 : Rheumatic Fever - Angelo V. Taranta, Milton Markowitz - Google Books

Rheumatic fever is an inflammatory connective tissue disease due to bacterial toxins after an infection with group A streptococci. It is a non-suppurative sequelae of the infection. It is a non-suppurative sequelae of the infection.

Chapter 4 : Rheumatic Fever | Taranta / Markowitz | 2nd ed. , | Buch | calendrierdelascience.com

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Acute rheumatic fever is caused by an autoimmune response to throat infection with Streptococcus calendrierdelascience.comc involvement during acute rheumatic fever can result in rheumatic heart disease, which can cause heart failure and premature mortality.

Chapter 5 : Rheumatic Fever

Read "Rheumatic Fever A Guide to its Recognition, Prevention and Cure with Special Reference to Developing Countries" by Angelo Taranta with Rakuten Kobo. It has become commonplace to say that the decline of rheuÂ- matic fever in Europe and North America has little, if anyÂ- t.