

Rheumatic heart disease in rural Pakistan

E L Kaplan

In the developing countries of the world, rheumatic fever and rheumatic heart disease remain significant medical and public health problems

Heart 2004;90:361–362. doi: 10.1136/hrt.2003.026013

In this issue of *Heart*, Rizvi and colleagues describe the current situation in Pakistan regarding rheumatic fever and rheumatic heart disease.¹ These noteworthy data likely are representative of many countries even at the beginning of the 21st century. While one might question how quantitatively these data represent the other parts of Pakistan, and while one might wish to delve further into the epidemiology of this important cardiovascular disease in Pakistan, nevertheless the point which cannot be overlooked is that this theoretically preventable cardiovascular disease is unacceptably too prevalent. In the developing countries of the world, countries which constitute approximately two thirds of the world's population, rheumatic fever and rheumatic heart disease (and by inference group A streptococcal upper respiratory tract infections) remain significant medical and public health problems. Uncounted numbers of children and young adults are in need of valve surgery, a procedure which few individuals or ministries of health in those countries can afford.

While there have been recent reports addressing the incidence of acute rheumatic fever and the prevalence of rheumatic heart disease in many countries where the magnitude of the problem is similar (for example, India²), the sentinel reports authored by Strasser and colleagues from the World Health Organization three or four decades ago adequately documented the cost effectiveness of control programmes, and how the lack of attention even to secondary prophylaxis resulted in higher morbidity (including hospitalisations) and mortality.^{3–4} Yet, little seems to have changed during these past four decades.⁵ Furthermore, recent published data by Veasy and colleagues from middle class US populations in Utah clearly document that the potential for medical and epidemiologic problems caused by streptococcal infections, rheumatic fever, and rheumatic heart disease is not confined to socially and economically disadvantaged populations in the still industrialising countries. Since 1985, there have been more than 600 proven cases of acute rheumatic fever in Utah.^{6–8}

WHY IS RHEUMATIC HEART DISEASE UNCONTROLLED?

The obvious question is why has this disease remained essentially uncontrolled around the

world? The irrefutable studies by Wannamaker and colleagues in the early 1950s showed that prompt diagnosis and adequate penicillin treatment of group A streptococcal throat/tonsil infections prevent rheumatic fever (primary prophylaxis).⁹ During the past 10 or 15 years, although significant new microbiologic and immunologic information has been generated from studies using sophisticated molecular techniques in many basic science laboratories, the pathogenetic mechanism(s) by which the group A streptococcus incites the development of rheumatic fever and rheumatic heart disease remain, at best, incompletely defined. Furthermore, there has been little if any translation of laboratory discoveries (either relating to the organism or to its human host) to the clinic or to the bedside.

Effective control of streptococcal infections continues to be further complicated by the extraordinary and intense competition for health related funds within ministries of health in all countries. Recent commitments by international organisations such as WHO toward the control of coronary heart disease, hypertension, HIV/AIDS, tuberculosis, malaria, and others appear to have made it even more difficult for adequate attention to be given and for subsequent allocation of adequate resources for cost effective and inexpensive control of an essentially preventable cardiovascular disease, rheumatic heart disease. The effort expended and the resources required for rheumatic fever/rheumatic heart disease control are relatively minimal when compared with most other cardiovascular diseases, and the effort can be rewarded in a relatively short period of time as has been noted.

SITUATION IN PAKISTAN

What should be attempted about a current situation such as that described by Rizvi and colleagues in Pakistan? As has been the case with so many other consequences of infectious diseases, the development of vaccines and implementation of wide immunisation programmes have at least the theoretical potential for controlling group A streptococcal infections just as they have impacted smallpox, poliomyelitis, measles, diphtheria (which also has cardiovascular sequelae), tetanus, and many others. However, although there are currently candidate vaccines in very early clinical trials, it is probably accurate to speculate that a group A streptococcal vaccine ready for widespread use will not be available for a number of years, perhaps even a decade. Whether a vaccine—either one leading to effective type-specific immunity or one based upon conserved antigens of the group A streptococcus—will prove sufficiently cost effective to

Correspondence to:
Professor Edward L Kaplan,
Department of Pediatrics,
University of Minnesota
Medical School, 420
Delaware Street SE,
Minneapolis, MN 55455,
USA; kapla001@umn.edu

allow it to be distributed among the most at-risk populations in the developing countries has not been sufficiently discussed by global health authorities. This issue should be addressed. Research into the still elusive pathogenetic mechanism(s) should be supported.

Thus, physicians and other clinicians are left with having to continue the current clearly unsatisfactory control methods and procedures as has been the case in the past, and having to make efforts to implement new laboratory developments into the every day practical clinical diagnosis and management of streptococcal infections. The importance of the roles of primary care physicians and of public health authorities around the world in implementing a relatively simple and cost effective approach surely requires additional consideration and resources.

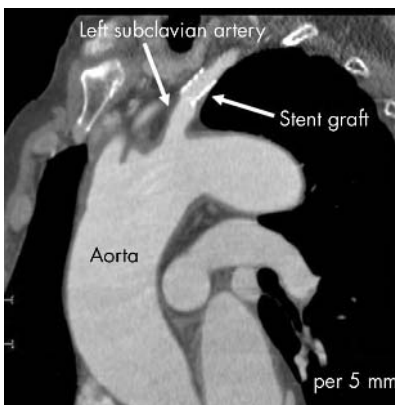
It is the responsibility of the medical and public health communities to raise these important questions in a meaningful way in the hope that until more effective techniques (perhaps a cost effective vaccine) are available, control of situations such as that described by Rizvi and colleagues can be more effectively attempted.

IMAGES IN CARDIOLOGY

Patency of the left subclavian artery following implantation of stent graft to rectify a stenosis, as demonstrated by multislice computed tomography

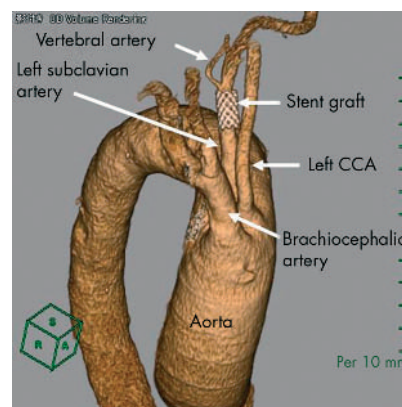
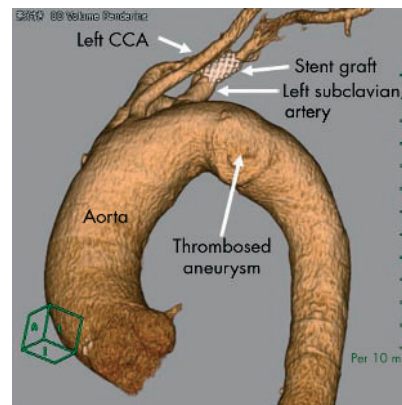
A 67 year old woman with a thrombosed aortic arch aneurysm, presented with numbness in her left arm and a substantial decrease in blood pressure (122/94 mm Hg), which compared unfavourably with the blood pressure of her right arm (156/90 mm Hg). A conventional angiogram revealed a prominent stenosis in the left subclavian artery, more proximal than the site of the juncture with the vertebral artery. Percutaneous transluminal angioplasty was performed, a stent was successfully implanted, and the discrepancy in blood pressure was resolved (left arm 146/92 mm Hg, right arm 150/90 mm Hg). Enhanced multislice computed tomography (MSCT) (Light Speed Ultra 16, General Electric, Milwaukee, USA) was performed (slice thickness 0.625 mm; helical pitch 3.25), using the following technique. The patient was given an intravenous injection of 100 ml of iodinated contrast medium (350 mgI/ml) and MSCT scanning was carried out after a 30 second delay. Volume data were extracted at the end of diastole and transferred to a workstation (Virtual Place Office Azemoto, Tokyo, Japan). Three dimensional, contrast enhanced, volume rendered images were reconstructed through retrospective ECG gating.

A multiplanar, reconstructed image clearly showed the patent lumen of the



proximal portion of the left subclavian artery, surrounded by the implanted stent graft (panel above). Volume rendered images revealed the implanted stent graft on the proximal portion of the left subclavian artery, and displayed the spatial relationship of the graft to the vertebral artery and other branches of the aortic arch, such as the left common carotid artery (CCA) (right upper and lower panels). Furthermore, the volume rendered images allowed the thrombosed aneurysm to be easily visualised (right upper panel).

N Funabashi, T Sekine, I Komuro
komuro-iky@umin.ac.jp



REFERENCES

- 1 Rizvi SF, Khan MA, Kundi A, *et al.* Status of rheumatic heart disease in rural Pakistan. *Heart* 2004;**90**:394-9.
- 2 Vijaykumar M, Narula J, Reddy KS, *et al.* Incidence of rheumatic fever and prevalence of rheumatic heart disease in India. *Int J Cardiol* 1994;**43**:221-8.
- 3 Strasser T, Dondog N, El Kholy A, *et al.* The community control of rheumatic fever and rheumatic heart disease: report of a WHO international cooperative project. *Bull WHO* 1981;**59**:285-94.
- 4 Strasser T. Rheumatic fever and rheumatic heart disease in the 1970s. *WHO Chronicle* 1978;**49**:88-93.
- 5 Kaplan EL. T. Duckett Jones Memorial Lecture. Global assessment of rheumatic fever and rheumatic heart disease at the close of the century. Influences and dynamics of populations and pathogens: a failure to realize prevention. *Circulation* 1993;**88**:1964-72.
- 6 Veasy LG, Wiedmeier SE, Orsmond GS, *et al.* Resurgence of acute rheumatic fever in the intermountain area of the United States. *N Engl J Med* 1987;**316**:421-7.
- 7 Veasy LG, Tani LY, Hill HR. Persistence of acute rheumatic fever in the intermountain area of the United States. *J Pediatr* 1994;**125**:673-4.
- 8 Veasy LG, Tani LY, Daly JA, *et al.* Temporal association of mucoid strains of *Streptococcus pyogenes* with a continuing high incidence of rheumatic fever in Utah. Proceedings of the Pediatric Academic Societies Pediatric Research, Seattle, May 2003. *Pediatric Res* 2003;**53**:329a [abstract 1881].
- 9 Wannamaker LW, Rammelkamp CH Jr, Denny FW, *et al.* Prophylaxis of acute rheumatic fever by treatment of the preceding streptococcal infection with various amounts of depot penicillin. *Am J Med* 1951;**10**:673-95.