Progress in Cardiovascular Diseases 62 (2019) 447–451

Contents lists available at ScienceDirect

Progress in Cardiovascular Diseases

journal homepage: www.onlinepcd.com

Tricuspid regurgitation is a public health crisis☆

Maurice Enriquez-Sarano *, David Messika-Zeitoun, Yan Topilsky, Christophe Tribouilloy, Giovanni Benfari, Hector Michelena

Cardiovascular Department, University of Ottawa, Canada
Cardiovascular Department, University of Tel-Aviv, Israel
Cardiovascular Department, University of Amiens, France

ARTICLE INFO

Article history:
23 October 2019
23 October 2019

Keywords:
Tricuspid regurgitation
Echocardiography

ABSTRACT

Tricuspid regurgitation (TR) has long been a forgotten valve disease of benign reputation. However, TR deserves higher attention and represents a growing public health crisis. Indeed, recent epidemiological data suggest that 1.6 million US residents are affected by moderate or severe TR. Furthermore, large recent cohorts demonstrate that higher degrees of TR are associated with considerable excess mortality, independent of all background clinical and hemodynamic contexts. Finally, analysis of recent cohorts also shows that >90% of patients with moderate or severe TR are never offered surgical treatment and remain untreated. Therefore, TR is frequent, severely impacts outcomes, and is rarely treated, justifying the development of new strategies and methods for its treatment.

Contents

Introduction

Tricuspid regurgitation (TR) is a valvular condition which is frequent and has long been perceived as being well-known and well-understood.[1] Therefore, the claim that TR represents a public health crisis is not simple to comprehend. Unfortunately, the concept of public health crisis is poorly defined and may be misunderstood for a public health emergency. In our opinion, the first element is the frequency of the condition; indeed, a rare condition may be very serious and warranting treatment but cannot amount to a public health crisis. The second element besides the frequency of the condition is its impact on outcome. A frequent condition that is benign cannot become a public health crisis. The third element to consider is the limited treatment received by the carriers of the condition, whether the treatment is often ineffective or whether an effective treatment is not administered.

TR is defined by the regurgitation of blood from the right ventricle (RV) to the right atrium, which is normally prevented by the coaptation of the tricuspid leaflets during systole.[1,2] Because presence of at least trivial TR is discovered in the vast majority of subjects, even with

Abbreviations and acronyms: HF, Heart failure; LV, Left ventricle; MR, Mitral regurgitation; PH, Pulmonary hypertension; RV, Right ventricle; TR, Tricuspid regurgitation.

☆ Statement of conflict of interest: see page 451.

* Address reprint requests to Maurice Enriquez-Sarano, MD, Cardiovascular Department, University of Ottawa, 2355 Hawthorne Hill Rd NE Rochester, MN, 55906, Canada.
E-mail address: sarano.maurice@gmail.com (M. Enriquez-Sarano).

https://doi.org/10.1016/j.pcad.2019.10.009
0033-0620/

completely normal hearts, it is indispensable to consider trivial TR as physiologic in most subjects and thus inconsequential.[1,2] Hence, the grading of TR by Doppler-echocardiography is crucial.[3] While the clinical significance of mild TR is most often considered benign, the TR that deserves the most attention is severe TR, and most recent data suggests that even moderate TR is also consequential and warrants our attention.[4] Therefore, the question to be examined is whether moderate or severe TR among our national community represents a public health crisis by fulfilling all three criteria of frequency, poor outcome and insufficient/ineffective treatment. Unfortunately, in view of its previous reputation of a benign condition,[5] the background knowledge regarding TR has remained quite scarce and because TR is also heterogeneous[6] the literature may be quite confusing and uncertain.

Epidemiology of TR: is TR a frequent condition?

The epidemiology of TR is poorly known. Similar to other valve diseases the clinical examination is generally unrevealing[7,8] and many cases of confirmed TR are undetected clinically. This lack of sensitivity of clinical examination for TR makes Doppler-echocardiography the main tool for detecting TR in the population.[1] Color flow imaging is highly sensitive for TR, which has been shown to be quite frequent in the normal population. The American Society of Echocardiography has provided guidelines to grade TR into mild, moderate, and severe TR with the concept that severe and probably moderate TR are the grades of clinical significance.[3] TR can be quantified by the PISA method measuring the effective regurgitant orifice and the regurgitant volume, but this quantification remains insufficiently performed routinely.[1] This quantification, however, teaches us that for the same regurgitant orifice, the regurgitant volume is smaller with TR than mitral regurgitation (MR).[9] Hence, jets of patients with normal pulmonary pressure may be underestimated and great care needs to be taken to appropriately evaluate TR. This assumption that moderate TR is of clinical significance in terms of clinical status and outcome is recently being emphasized. Hence, trivial TR is considered valuable for calculation of pulmonary pressure by the Bernouilli equation[10] but not different from no TR in terms of outcome. Mild TR is of uncertain significance but is considered because of its frequency as a finding borderline of normal.[11] Hence, epidemiological studies have focused around the prevalence of moderate-severe TR. True prevalence estimation by systematic echocardiography of the general population has been rarely reported[12] due to the considerable effort and funding required but also due to the fact that echocardiography initially aimed at another endpoint (e.g. left ventricular function) may not be of perfectly reliable interpretation for valve disease in general and for TR detection in particular.[12] The most reliable evaluation was provided by the OxVALVE study in the UK, whereby ~2500 random subjects of age ≥65 were screened specifically for valve disease, finding moderate-severe TR in 2.7%.[13] The data provided is limited at present, without report of age or sex link to TR prevalence.

Existing US reports of systematic Doppler-Echocardiography in a sample of the general population were plagued with very few detected cases of moderate/severe TR and hence an impossibility of normalizing prevalence to that of the general US population.[12] However, it appeared that TR prevalence was greater in older subjects and in women.

In view of the limitations of attempts at systematic echocardiography, another approach is to consider prevalence of TR diagnosed in a geographically defined community, as long as all cases diagnosed can be defined.[14] In those circumstances prevalence can be calculated with the denominator being the community population and the numerator being the number of cases diagnosed. Prevalence can be adjusted to that of the entire US population based on age and sex. Up to now the only community where such a measurement could be performed was Olmsted County, Minnesota, whereby a single echocardiographic laboratory, a relatively isolated population with very little care performed outside of the county, allowed detection of all (or almost all) cases diagnosed in the community. These assumptions have been validated by the similarity of patterns of left-sided valve diseases in the general population and in the county population.[14] Results in regard to TR have been recently reported[6] and indicate TR prevalence overall of 0.55%[0.50–0.60] and of 0.47% [0.39–0.55] in men lower than in women 0.59%[0.52–0.67]. Another important feature is the strong link between TR prevalence and age, (Fig 1) very similar to the link between age and left sided valve diseases.[14] Importantly, there is a very strong similarity between OxVALVE and Olmsted studies as the 2.7% prevalence in OxVALVE is well within the 1.09% for the 65–74 age range and the 3.96% in the 75+ age range.[13]

Using this clinical prevalence and the distribution of the US population per the US census bureau, the total burden of TR diagnosed in the community can be evaluated at approximately 1.6 million US residents. Such burden of cases diagnosed is quite close to that of aortic stenosis[14] warranting a similar attention. Recent data suggest a considerable underdiagnosis of valve diseases in the US[14] and the UK[13]whereby diagnosed cases of left sided valve diseases represent only 2/3 of total prevalence. While the underdiagnosis may be higher in TR due to the frequency of lack of murmur,[6] a conservative estimate of the total burden of moderate or severe TR in the US is 2.4 million residents both diagnosed and undiagnosed.

One important information on TR epidemiology is the incidence of new cases per year that may allow judging the need for treatment and whether it is met or not. There has been no such direct measurement performed yet, but an estimation is possible based on a stable prevalence in the community and on the mortality rate per year. While mortality associated with moderate severe TR is variable, one can estimate the yearly mortality at 10 to 15% based on community data.[6] Hence, based on those values one can estimate the moderate or severe TR incidence of new cases between 160,000 and 240,000 per year in the US alone.

Therefore, to the first question of whether TR is frequent, recent data soundly provide a positive answer.

Outcome of TR: is TR a serious condition?

This question is the most difficult to answer as historical data provided blanket answers that were contradictory. Indeed, on one hand TR was recommended as benign enough to require only conservative management[5] and it was even estimated that complete surgical removal of the tricuspid valve was associated with excellent tolerance and outcome.[15] On the other hand TR diagnosed by echocardiography, irrespective of its cause, was found overall to severely impact outcome.[16] These contradictory suggestions cannot be accepted as blanket statements because TR is a heterogeneous condition.[6] In those diverse circumstances poor outcomes may be related to the causal disease or to the comorbidity associated with the TR cause and not to the TR itself. What is the most recent evidence

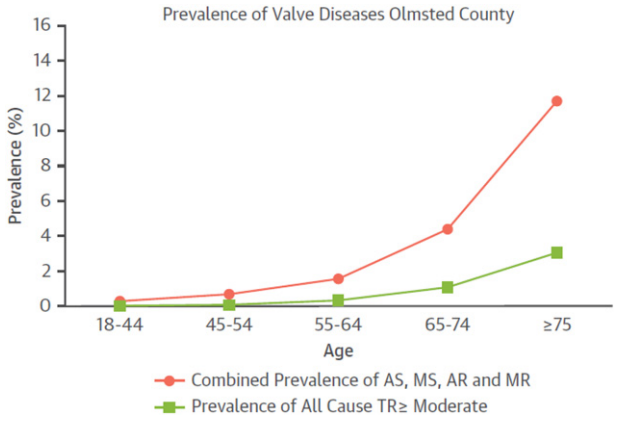

Fig 1. Prevalence of tricuspid regurgitation diagnosed in the community compared to left sided valve diseases. The green curve represents the prevalence of tricuspid regurgitation increasing with age similarly to left sided valve disease (red curve). Reproduced with authorization from reference.[6]

regarding the risk attached to TR, in and by itself, within well-defined uniform clinical contexts?

In the community, outcome analysis suggests a high mortality and cardiac morbidity associated with all clinical contexts.[6] There is some variability in absolute rates of events depending on the clinical context but due to relatively small samples in most subsets, ascertaining the risk attached to TR in context requires larger, homogeneous cohorts.

Some of the TR clinical contexts are relatively seldom, such as congenital heart diseases[17] or organic TR[18] or post-pacemaker/defibrillator TR.[19] While small case series suggest that flail leaflet causing TR is associated with poor outcome, [18] data remain scarce. Similarly, iatrogenic TR due to lead interference with tricuspid valve appears to have poor outcomes[19,20] although these are difficult to separate from the reasons for implantation. Other specific contexts, all with TR called functional or secondary with structurally normal leaflets, have been the subject of more in-depth evaluation of the impact of TR on outcome. The most investigated has been functional TR associated with heart failure (HF) with reduced ejection fraction. Although few studies with generally small samples did not observe an independent impact of functional TR on clinical outcome of HF,[21] most studies did,[22,23] with also possible impact in patients with HF with preserved ejection fraction.[24] Functional TR is associated with excess mortality that increases with the degree of TR, thus highly suggestive of a causal effect. Hence, it is not just severe functional TR that affects outcome but also moderate TR (Fig 2). This poor outcome linked to functional TR, despite the normal tricuspid leaflets, appears independently of the severity of the left ventricular (LV) and RV dysfunction and of pulmonary hypertension (PH). The data regarding TR occurring in the context of left sided valve disease is more scarce. Limited series addressed mitral stenosis post-balloon valvuloplasty[25] MR of various causes[26] and aortic stenosis[27] and are all notably biased but tend to suggest that also in the context of left-sided valve disease TR may be linked to poor clinical outcome. Finally, one group of particular interest is "isolated" TR, a functional TR without left sided valve disease, without LV dysfunction and without PH.[28,29] In this group, often associated with atrial arrhythmia and characterized by a primary right atrial dilatation with TR mechanism primarily related to isolated tricuspid annular dilatation,[30] there is little cardiac comorbidity that may explain the poor outcome instead of the TR and nevertheless these patients incur with severe TR considerable excess mortality and high rate of cardiac events whether atrial fibrillation or sinus rhythm is present (Fig 3).

Therefore, the most recent data, generally based on large series tend to show that in all clinical contexts investigated, TR, even functional, is associated with poor outcome not only when it is severe but most remarkably when it is moderate and independently of all baseline characteristics. Hence, to the question regarding the survival impact of TR, mounting evidence shows that increasing severity of TR is associated with higher and considerable excess-mortality, despite medical treatment.

Treatment of TR: is there an unmet need for treatment?

The only currently approved treatment for TR is cardiac surgery with tricuspid valve repair or replacement.[31] The only class I indication for TR surgery is for severe TR in the context of mitral surgery. To consider whether the need for treatment is met or not, one must consider effectiveness of surgical treatment and the consistency and frequency of treatment application.

Effectiveness of tricuspid surgery

Tricuspid valve surgery has a bad reputation although in some subsets of patients such as tricuspid flail leaflets valve repair can be performed in a majority of patients with low operative mortality and good long-term outcome.[18] However, when tricuspid surgery in all types of cases, even isolated, is considered, many single-center studies report considerable operative mortality reaching 25 to 30%.[32] Nationally, reports in large numbers of patients mention operative mortality slightly decreasing recently but remaining very high around 8 to 10%.[33] Survival following tricuspid surgery appears to be reduced although long-term data remain scant.[34] Such poor reputation of tricuspid valve surgery is in part tainted by the advanced stage at which patients are referred to surgery. Indeed, survival after isolated tricuspid surgery is much better in patients who are operated before they reach the stage of intractable HF[34] or have a repair before persistent atrial fibrillation has occurred.[18] Because the indication of isolated tricuspid surgery is frequently made at such desperate stages, [35] the comparison with medical treatment is markedly biased even after "matching"[36] and should not discourage future conduct of clinical trials to assess the intrinsic efficacy of tricuspid surgery, in particular in preventing the recurrence of HF. Another issue is the mediocre results of tricuspid valve repair. Indeed, while complex repair can be done for organic TR,[18] most repairs are simple annuloplasties for functional TR.[33] Such repairs often fail with recurrent TR in 25% or more of patients operated over 2 to 5 years post-surgery.[37] This high failure rate is due to valve tenting, most frequent in patients with PH or, RV marked dilatation or

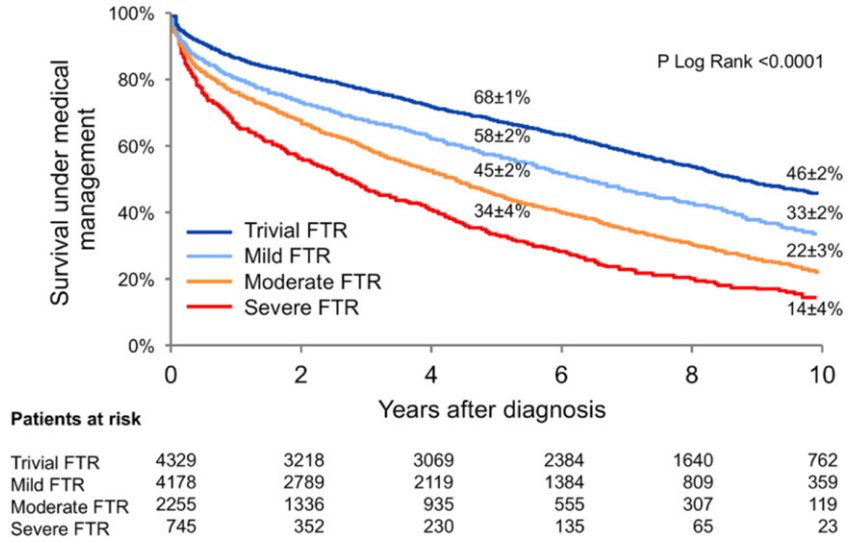

Fig 2. Survival of patients with heart failure with reduced ejection fraction according to tricuspid regurgitation severity. Note that each grade of tricuspid regurgitation is associated with increased long-term mortality. Reproduced with authorization from reference.[4]

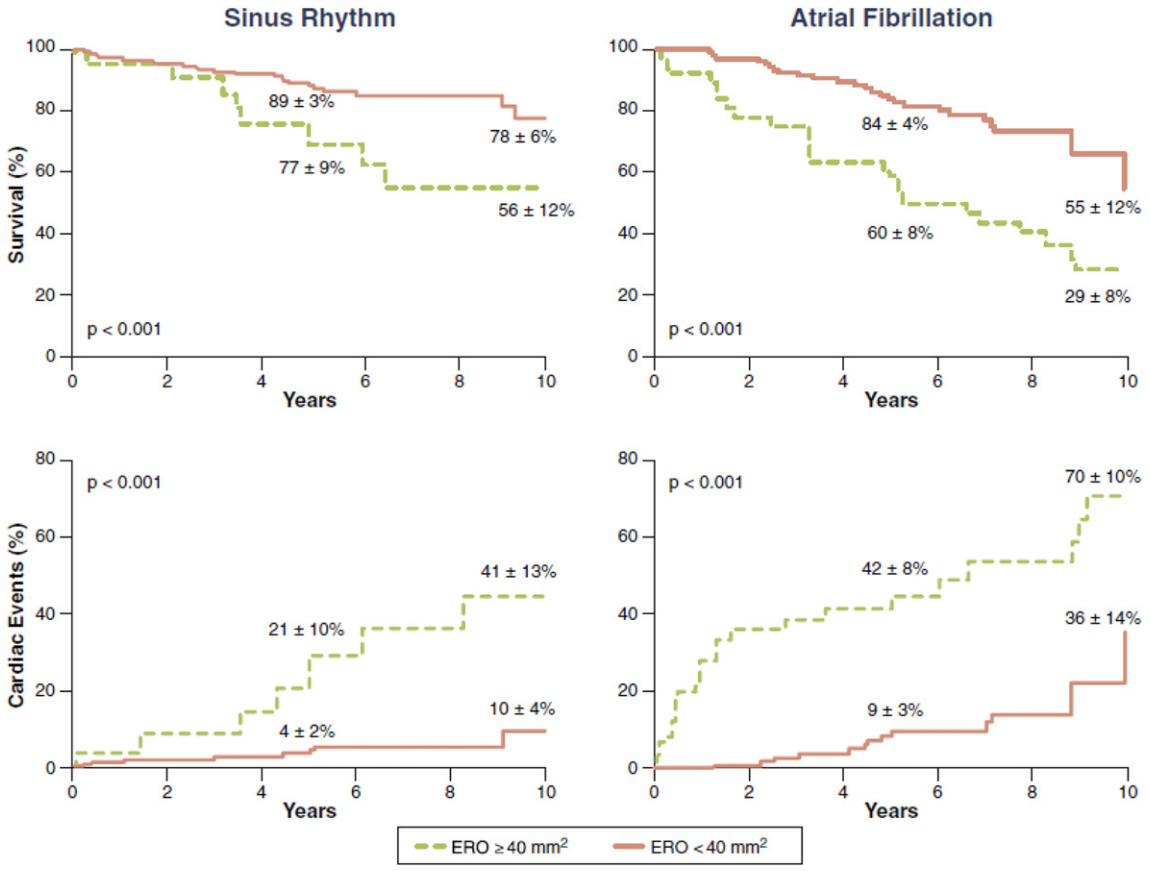

Fig 3. Clinical outcome of isolated tricuspid regurgitation. The outcomes analyzed are survival (top row) and cardiac events (lower row) in sinus rhythm (left graphs) and in atrial fibrillation (right graphs). Note that for both endpoints in sinus rhythm or atrial fibrillation, severe isolated tricuspid regurgitation, despite absence of left ventricular dysfunction, left sided valve disease and pulmonary hypertension, is associated with much worse clinical outcome. Reproduced with authorization from reference.[28]

dysfunction, which is often under-appreciated pre-operatively, is not corrected by the annuloplasty and yields tricuspid malcoaptation post-annuloplasty.[38] While some surgical techniques may be used to address this type of valvular dysfunction, it is not detectable intra-operatively when the heart is unloaded of flaccid and post-operative recurrence of TR remains frequent. Tricuspid valve replacement with mechanical prosthesis is rarely used due to the concern with valve thrombosis.[34] Biological valve replacement is not devoid of the risk of thrombosis and tissue degeneration but the new development of valve in valve implantation allows potential therapeutic approaches. Irrespective of these considerations, tricuspid valve surgery for TR is not considered an optimal and safe treatment.

With no optimal surgical solution patients are often treated with increasing diuretic treatment, initially with some success but ultimately complicated by renal failure, which is another dis-incentive to surgical treatment.[34] Therefore, physicians even with the best of intentions are undertreating TR.

Is TR undertreatment established as a fact?

1. Nationally the confrontation of a prevalence of 1.6 million patients with prevalent moderate severe TR vs. the 8 to 10,000 tricuspid surgeries performed yearly is highly suggestive of a massive undertreatment. Considering the incident cases estimated in the nation no more than 4 to 5% of incident cases are ever operated.
2. In the privileged community of Olmsted county, MN, the rate of lifetime tricuspid surgery for moderate severe TR diagnosed in the 1990's has been measured at 2.5%.[6]
3. In the privileged but small subset of flail tricuspid leaflets, around 50% of cases underwent tricuspid surgery.[18]
4. In the large subset of patients with functional TR secondary to LV dysfunction, those operated for TR represent only 4% of the patients with moderate or severe TR five years after diagnosis.[4]
5. In the context of isolated TR, despite the absence of LV dysfunction, left-sided valve disease or PH, only 8% ever get tricuspid surgery performed.[28]

Therefore, despite the doubts on risks and efficacy of tricuspid valve surgery, the immense majority of patients with moderate or severe TR throughout their lifetime is left untreated by the only treatment currently approved in the US for TR and are only receiving medical treatment, which does not prevent the occurrence of considerable excess mortality. Hence, we believe that this situation qualifies as undertreatment or unmet need for treatment for TR.

TR public health crisis: the way forward

With TR now proven to bear the hallmarks of a public health crisis as a condition that is frequent in the population, affecting millions of mostly older patients, complicated by severe HF and excess mortality and treated only in a minimal fraction of those affected, it is abundantly clear that a plan of action is necessary. The way forward involves:

1. Paying more attention to diagnosing moderate and severe TR, which ideally encompasses increased use of quantitative Doppler-echocardiographic methods.
2. Avoid being falsely reassured by an initial improvement with diuresis and keep a continued contact with patients who have challenging symptoms and clinical features.

3. Early consideration of tricuspid surgery before the occurrence of intractable HF. At best that consideration should occur within a multidisciplinary Heart Team.
4. Pursue the current development of new therapies, surgical and mostly percutaneous.[39] Current transcatheter therapies appear to reduce TR in half on average leaving often TR remaining severe and more effective therapies are warranted.[40]
5. Test efficacy of therapies in appropriately designed randomized clinical trials.

The burden of TR is large, almost unaddressed and warrants our full attention.

Statement of conflict of interest

None of the authors have any conflicts of interests with regard to this publication.

References

1. Badano LP, Muraru D, Enriquez-Sarano M. Assessment of functional tricuspid regurgitation. Eur Heart J 2013;34:1875-1885.
2. Hung J. The pathogenesis of functional tricuspid regurgitation. Semin Thorac Cardiovasc Surg 2010;22:76-78.
3. Zoghbi WA, Adams D, Bonow RO, et al. Recommendations for noninvasive evaluation of native valvular regurgitation: a report from the American Society of Echocardiography Developed in Collaboration with the Society for Cardiovascular Magnetic Resonance. J Am Soc Echocardiogr 2017;30:303-371.
4. Benfari G, Antoine C, Miller WL, et al. Excess mortality associated with functional tricuspid regurgitation complicating heart failure with reduced ejection fraction. Circulation 2019;140:196-206.
5. Braunwald NS, Ross Jr J, Morrow AG. Conservative management of tricuspid regurgitation in patients undergoing mitral valve replacement. Circulation 1967;35:I63-I69.
6. Topilsky Y, Maltais S, Medina Inojosa J, et al. Burden of tricuspid regurgitation in patients diagnosed in the community setting. JACC Cardiovasc Imaging 2019;12:433-442.
7. Marijon E, Ou P, Celermajer DS, et al. Prevalence of rheumatic heart disease detected by echocardiographic screening. N Engl J Med 2007;357:470-476.
8. Gardezi SKM, Myerson SG, Chambers J, et al. Cardiac auscultation poorly predicts the presence of valvular heart disease in asymptomatic primary care patients. Heart 2018;104:1832-1835.
9. Tribouilloy CM, Enriquez-Sarano M, Capps MA, Bailey KR, Tajik AJ. Contrasting effect of similar effective regurgitant orifice area in mitral and tricuspid regurgitation: a quantitative Doppler echocardiographic study. J Am Soc Echocardiogr 2002;15:958-965.
10. Currie P, Seward J, Chan K. Continuous wave Doppler determination of right ventricular pressure: a simultaneous Doppler-catheterization study in 127 patients. J Am Coll Cardiol 1985;6:750-756.
11. Yang L, Chen H, Pan W, et al. Analyses for prevalence and outcome of tricuspid regurgitation in China: an echocardiography study of 134,874 patients. Cardiology 2019;142:40-46.
12. Singh JP, Evans JC, Levy D, et al. Prevalence and clinical determinants of mitral, tricuspid and aortic regurgitation. Am J Cardiol 1999;83:897-902.
13. d'Arcy JL, Coffey S, Loudon MA, et al. Large-scale community echocardiographic screening reveals a major burden of undiagnosed valvular heart disease in older people: the OxVALVE Population Cohort Study. Eur Heart J 2016;37:3515-3522.
14. Nkomo VT, Gardin JM, Skelton TN, Gottdiener JS, Scott CG, Enriquez-Sarano M. Burden of valvular heart diseases: a population-based study. Lancet 2006;368:1005-1011.
15. Arbulu A, Holmes RJ, Asfaw I. Tricuspid valvulectomy without replacement. Twenty years' experience. J Thorac Cardiovasc Surg 1991;102:917-922.
16. Nath J, Foster E, Heidenreich PA. Impact of tricuspid regurgitation on long-term survival. J Am Coll Cardiol 2004;43:405-409.
17. Attenhofer Jost CH, Connolly HM, Scott CG, Burkhart HM, Warnes CA, Dearani JA. Outcome of cardiac surgery in patients 50 years of age or older with Ebstein anomaly: survival and functional improvement. J Am Coll Cardiol 2012;59:2101-2106.
18. Messika-Zeitoun D, Thomson H, Bellamy M, et al. Medical and surgical outcome of tricuspid regurgitation caused by flail leaflets. J Thorac Cardiovasc Surg 2004;128:296-302.
19. Lin G, Nishimura RA, Connolly HM, Dearani JA, Sundt 3rd TM, Hayes DL. Severe symptomatic tricuspid valve regurgitation due to permanent pacemaker or implantable cardioverter-defibrillator leads. J Am Coll Cardiol 2005;45:1672-1675.
20. Van De Heyning CM, Elbarasi E, Masiero S, et al. Prospective study of tricuspid regurgitation associated with permanent leads after cardiac rhythm device implantation. Can J Cardiol 2019;35:389-395.
21. Mutlak D, Lessick J, Khalil S, Yalonetsky S, Agmon Y, Aronson D. Tricuspid regurgitation in acute heart failure: is there any incremental risk? Eur Heart J Cardiovasc Imaging 2018;19:993-1001.
22. Topilsky Y, Inojosa JM, Benfari G, et al. Clinical presentation and outcome of tricuspid regurgitation in patients with systolic dysfunction. Eur Heart J 2018;39:3584-3592.
23. Kazum SS, Sagie A, Shochat T, et al. Prevalence, echocardiographic correlations, and clinical outcome of tricuspid regurgitation in patients with significant left ventricular dysfunction. Am J Med 2019;132:81-87.
24. Santas E, Chorro FJ, Minana G, et al. Tricuspid regurgitation and mortality risk across left ventricular systolic function in acute heart failure. Circ J 2015;79:1526-1533.
25. Sagie A, Schwammenthal E, Newell J, et al. Significant tricuspid regurgitation is a marker for adverse outcome in patients undergoing percutaneous balloon mitral valvuloplasty. J Am Coll Cardiol 1994;24:696-702.
26. Varadarajan P, Pai RG. Tricuspid regurgitation in patients with severe mitral regurgitation and normal left ventricular ejection fraction: risk factors and prognostic implications in a cohort of 895 patients. J Heart Valve Dis 2010;19:412-419.
27. Amano M, Izumi C, Taniguchi T, et al. Impact of concomitant tricuspid regurgitation on long-term outcomes in severe aortic stenosis. Eur Heart J Cardiovasc Imaging 2019;20:353-360.
28. Topilsky Y, Nkomo VT, Vatury O, et al. Clinical outcome of isolated tricuspid regurgitation. J Am Coll Cardiol Img 2014;7:1185-1194.
29. Fender EA, Petrescu I, Ionescu F, et al. Prognostic importance and predictors of survival in isolated tricuspid regurgitation: a growing problem. Mayo Clin Proc 2019;94:2032-2039.
30. Topilsky Y, Khanna A, Le Tourneau T, et al. Clinical context and mechanism of functional tricuspid regurgitation in patients with and without pulmonary hypertension. Circ Cardiovasc Imaging 2012;5:314-323.
31. Nishimura RA, Otto CM, Bonow RO, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation 2014;129:e521-e643.
32. Anselmi A, Ruggieri VG, Harmouche M, et al. Appraisal of long-term outcomes of tricuspid valve replacement in the current perspective. Ann Thorac Surg 2016;101:863-871.
33. Kilic A, Saha-Chaudhuri P, Rankin JS, Conte JV. Trends and outcomes of tricuspid valve surgery in North America: an analysis of more than 50,000 patients from the Society of Thoracic Surgeons database. Ann Thorac Surg 2013;96:1546-1552 [discussion 1552].
34. Topilsky Y, Khanna AD, Oh JK, et al. Preoperative factors associated with adverse outcome after tricuspid valve replacement. Circulation 2011;123:1929-1939.
35. Dreyfus J, Ghalem N, Garbarz E, et al. Timing of referral of patients with severe isolated tricuspid valve regurgitation to surgeons (from a French nationwide database). Am J Cardiol 2018;122:323-326.
36. Axtell AL, Bhambhani V, Moonsamy P, et al. Surgery does not improve survival in patients with isolated severe tricuspid regurgitation. J Am Coll Cardiol 2019;74:715-725.
37. McCarthy PM, Bhudia SK, Rajeswaran J, et al. Tricuspid valve repair: durability and risk factors for failure. J Thorac Cardiovasc Surg 2004;127:674-685.
38. Fukuda S, Gillinov AM, McCarthy PM, et al. Determinants of recurrent or residual functional tricuspid regurgitation after tricuspid annuloplasty. Circulation 2006;114:I582-I587.
39. Taramasso M, Pozzoli A, Guidotti A, et al. Percutaneous tricuspid valve therapies: the new frontier. Eur Heart J 2017;38:639-647.
40. Nickenig G, Kowalski M, Hausleiter J, et al. Transcatheter treatment of severe tricuspid regurgitation with the edge-to-edge MitraClip technique. Circulation 2017;135:1802-1814.