Received: 7 March 2019 | Revised: 6 April 2019 | Accepted: 22 April 2019

DOI: 10.1111/chd.12782

EDUCATION FORUM

Congenital Heart Disease WILEY

Coronary artery disease in adults with tetralogy of Fallot

Alexander C. Egbe MD, MPH | Sindhura Ananthaneni MD | Raja Jadav MD | Srikanth Kothapalli MD | Charanjit S. Rihal MD | Muhammad Masood MD | Mounika Angirekula MD | Maria Najam MD | Numra Bajwa MBBS | Karim Tarek MBBS | Jessey Matthew MBBS | Heidi M. Connolly MD

Department of Cardiovascular Medicine, Mayo Clinic, Rochester, Minnesota

Correspondence
Alexander C. Egbe, MD, MPH, FACC, Mayo Clinic and Foundation, 200 First Street SW, Rochester, MN 55905.
Email: egbe.alexander@mayo.edu

Funding information
Dr Egbe is supported by National Heart, Lung, and Blood Institute (NHLBI) grant K23 HL141448-01

Abstract

Background: There are limited data about outcomes of coronary artery disease (CAD) in adults with repaired tetralogy of Fallot (TOF). The purpose of this study was to describe the prevalence and treatment of CAD in adults with TOF, and the impact of CAD on long-term survival.

Methods: Retrospective review of MACHD database for adults with repaired TOF who underwent aortic root/selective coronary angiogram, 1990-2017. Patients were categorized into three groups: (1) No CAD defined as normal coronary angiogram; (2) Mild CAD defined as ≤50% stenosis in all vessels; and, (3) Significant CAD defined as >50% stenosis in any vessel.

Results: We identified 105 (23%) of 465 TOF patients that had angiograms; mean age 47 ± 12 years. The prevalence of mild CAD and significant CAD was 19% (20 patients) and 15% (16 patients), respectively. Of these 16 patient with significant CAD, 9 (56%), 3 (19%), and 4 (24%) patients received guideline directed medical therapy, percutaneous coronary intervention, and coronary artery bypass grafting, respectively. Significant CAD was an independent risk factor for mortality (HR: 2.03, 95% CI 1.64-4.22, P = .022) after adjustment for differences in age, and prevalence of atrial fibrillation and renal dysfunction.

Conclusions: Based on a review of a selected cohort of 105 TOF patients, the prevalence of mild CAD and significant CAD was 19% and 15%, respectively. Significant CAD was an independent risk factor for mortality. There is need for more research to determine optimal noninvasive diagnostic strategies and optimal patient selections and methods for revascularization.

KEYWORDS
coronary artery disease, coronary intervention, tetralogy of Fallot

Abbreviations: ASCVD, atherosclerotic cardiovascular disease; CABG, coronary artery bypass grafting; CAD, coronary artery disease; CI, confidence interval; GDMT, guideline-directed medical therapy; HR, hazard ratio; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; TOF, tetralogy of Fallot.

1 | INTRODUCTION

There has been a significant improvement in the long-term survival of patients with congenital heart disease because of improvement in medical and surgical therapies since the first surgical palliation of congenital heart disease in the 1950s.[1-3] With the improvement in life expectancy, adults with congenital heart disease are now at risk for acquired heart diseases such as coronary artery disease (CAD).[4] Although CAD is well studied because it is a leading cause of cardiovascular mortality in the developed world,[5] very little is known about the prevalence, treatment and outcomes of CAD in adults with congenital heart disease.[4,6]

Tetralogy of Fallot (TOF) is the most common cyanotic heart disease, and it is the most common complex congenital heart disease seen in the adult congenital heart disease clinic.[7,8] Most of the adult TOF patients currently being followed underwent surgical repair in the 1980s or before, and at that time palliative shunts followed by late repair was the standard of care.[1-3] As a result these patients were exposed to prolonged duration of cyanosis and underwent cardiac surgery in an era when myocardial protection during cardiopulmonary bypass was still suboptimal.[1-3] It is unknown how these patients with abnormal myocardium will respond to superimposed myocardial ischemia due to CAD or how well they will respond to conventional CAD therapy. The purpose of the study was therefore to describe the prevalence and treatment of CAD in adults with TOF, and the impact of CAD on long-term survival.

2 | METHODS

2.1 | Patient selection and data collection

The ***M***ayo ***A***dult ***C***ongenital ***H***eart ***D***isease (MACHD) database was queried for patients (age ≥18 years) with repaired TOF who underwent aortic root angiogram or selective coronary angiogram from January 1, 1990 to December 31, 2017. The patients with pulmonary atresia were excluded. The Mayo Clinic institutional review board approved this study and waived informed consent for patients that provided research authorization. The electronic health records were extensively reviewed in these patients.

2.2 | Endpoints and definitions

Images and reports of all aortic root or selective coronary angiograms were reviewed, and patients were classified as: (1) No CAD defined as normal coronary angiogram; (2) Mild CAD defined as ≤50% stenosis in all vessels; and, (3) Significant CAD defined as >50% stenosis in any vessel. Based on the guidelines for management of stable ischemic heart disease,[5] we classified CAD therapy as: (1) guideline directed medical therapy (GDMT) for patients that were on at least two of these medications: antiplatelet therapy, beta-blocker therapy or angiotensin concerting enzyme inhibitor/angiotensin receptor blocker therapy; (2) percutaneous coronary interventions (PCI); and, (3) coronary artery bypass grafting (CABG). In order to assess the yield of coronary angiogram (prevalence of significant CAD on angiogram) based on age at the time of angiogram, we divided the cohort into three groups (<40 years, 40-59 year, ≥60 years). In order compare improvement (or lack thereof) after revascularization in patients with a significant CAD, we classified left ventricular ejection fraction (LVEF) as normal LV function (LVEF ≥ 50%), mild LV dysfunction (LVEF 40%-49%), and moderate/severe LV dysfunction (LVEF < 40%).

2.3 | Statistical analysis

Data were presented as mean ± standard deviation, median (interquartile range), or counts (%), and between-group comparisons were performed using *t*-test, Wilcoxon test, chi-square test, and Fisher's exact test as appropriate. Survival analysis was performed using the Kaplan-Meier's method and compared using log-rank test. The adjusted mortality risk due significant CAD was assessed using Cox proportional hazard model, and expressed as hazard ratio (HR) and 95% confidence interval (CI). Because of the small number of patients with significant CAD, and hence small number of potential mortality in this group, we adjusted only for the clinical variables that were different between the groups during assessment for the association between significant CAD and all-cause mortality. The at-risk period was calculated from the time of angiogram to time of death or last follow-up. All statistical analyses were performed with JMP software (version 13.0; SAS Institute Inc, Cary NC) and $P < .05$ was considered statistically significant.

3 | RESULTS

Of the 465 TOF patients in the MACHD database, 105 (23%) patients underwent aortic root angiogram or selective coronary angiogram, and these 105 patients comprised the study group. There were significant differences in patient demographics, comorbidities, and echocardiographic indices between the groups with and without angiograms, Supplementary Table 1.

Selective coronary angiograms and aortic root angiograms were performed in 98 (93%) and 7 (7%), respectively, and mean age at the time of angiogram was 47 ± 12 years. The primary indication for angiogram was preoperative evaluation for cardiac surgery 79 (75%), abnormal stress test 19 (18%), chest pain and/or dyspnea in the setting of known ischemic heart disease 1 (0.7%), non-ST segment elevation myocardial infarction 3 (3%), and left ventricular dysfunction 3 (3%). Of the 105 patients, 69 (66%) had no CAD, 20 (19%) had mid CAD, and 16 (15%) had significant CAD. The proportions of coronary angiograms with significant CAD stratified by age at the time of angiogram are shown in Figure 1. None of the patients that had angiogram before the age of 40 years had significant CAD. All 24 patients <40 years of age at the time of angiogram, also had concomitant left and right heart catheterization, and coronary angiogram was not performed as a *stand-alone* procedure. All seven aortic root angiograms were performed in this subgroup of patients <40 years of age at the time of angiogram.

TABLE 1 Patients with significant CAD

		Vessels	LVEF	Therapy
#1	67 M	LAD and OM	55	LIMA to LAD graft and vein graft to OM during AVR
#2	50 M	LCX and RCA	50	History of 4 prior sternotomies. Implantation of a bare metal stent to proximal RCA and LCX one week prior to AVR
#3	41 F	LAD, RCA, and OM	30	Vein grafts to OM, mid LAD, and distal RCA during PVR. Had NSTEMI at the age of 59 years and received GDMT
#4	59 F	LAD	30	Underwent PVR, MVR, and TVR with bioprosthesis. CABG was not performed because patient had adequate perfusion of LAD territory by collateral arteries from the RCA. Patient died 14 days postop
#5	54 M	LAD and LCX	51	GDMT
#6	52 F	RCA	49	GDMT
#7	72 M	LCX	53	DES to the proximal and distal LCX
#8	66 M	LCX	50	GDMT
#9	72 M	LCX and diagonal	53	DES to the proximal and distal LCX
#10	62 M	RCA	50	GDMT
#11	84 M	RCA and LAD	60	GDMT
#12	51 M	LAD	55	GDMT
#13	50 M	LAD, LCX, and RCA	30	GDMT. Patient had anomalous LAD from RCA and severe pulmonary hypertension and was considered high risk for revascularization
#14	58 M	LAD and OM	30	LIMA to LAD graft and vein graft to OM during PVR
#15	65 F	LAD, RCA, and OM	40	Vein graft to LAD
#16	57 F	LCX, RCA, and OM	46	History of NSTEMI at 51 years and had vein graft to RCA during PVR at that time. Underwent TVR and PVR with bioprosthesis without revascularization after coronary angiogram. Patients died 23 days postop

Abbreviations: AVR, aortic valve replacement; DES, drug eluting stents; F, female; GDMT, guideline directed medical therapy; LAD, left anterior descending; LCX, left circumflex; LIMA, left internal mammary; LVEF, left ventricular ejection fraction; M, male; OM, obtuse marginal; PDA, posterior descending artery; PVR pulmonary valve replacement; RCA, right coronary artery.

Of the 105 patients, 7 (7%) had anomalous origin of coronary arteries, and these anomalies were as follows: anomalous origin of left anterior descending coronary artery from the right coronary artery (n = 5, 5%); anomalous origin of left circumflex coronary artery from the right coronary artery (n = 1, 1%); and, anomalous origin of right coronary artery from the left aortic sinus (n = 1, 1%). Coronary artery fistula was present in four (4%) patients, of these three patients had left circumflex coronary artery to pulmonary artery, fistula while one patient had right coronary artery to right coronary artery fistula.

3.1 | Significant CAD

Table 1 shows the clinical data of the 16 patients with significant CAD. Among these patients, seven (44%) had single vessel disease, while nine (56%) had multivessel disease (involvement of ≥2 vessels). The age at the time of coronary angiogram was 59 ± 11 years, the median LVEF was 49 (35%-45%), and eight patients had regional wall motion abnormalities.

Of the 16 patients with significant CAD, 9 (56%) received GDMT, 3 (19%) received GDMT and PCI, and 4 (24%) received

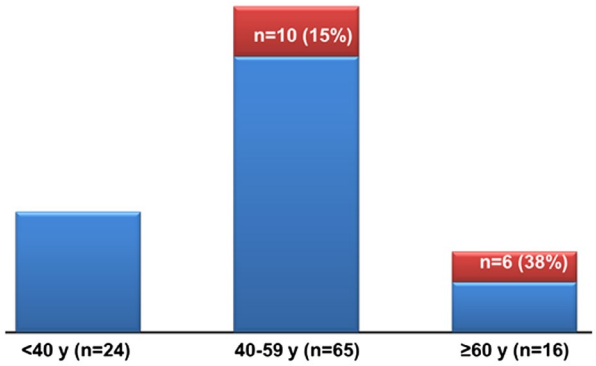

FIGURE 1 Bar graph showing the prevalence of significant coronary artery disease (CAD) categorized by age at the time of angiogram. Red represents patients with significant CAD, while blue represent patients with mild or no CAD

GDMT and CABG. There were no postprocedural complications for the three patients that had PCI and no postoperative complications or in-hospital mortality for the four patients that had CABG. There were two postoperative deaths in two patients after valve surgery without CABG (Patient #4 and #16). The remaining 14 patients had transthoracic echocardiogram at 1-year postcoronary angiogram, and 3 (43%) of the 7 patients that initially had LV dysfunction (LVEF < 50%) had normalization of LVEF, and all 3 patients had revascularization.

3.2 | CAD and survival

There was a significant difference in the 10-year survival of the patients with no CAD, mild CAD, and significant CAD (unadjusted survival of 97% vs 85% vs 46%, respectively, $P < .001$), Figure 2. In comparison to patients without CAD, the patients with CAD were older and had more atrial fibrillation and renal dysfunction, Table 2. After adjustment for these baseline differences, significant CAD remained an independent risk factor for mortality (HR: 2.03, 95% CI 1.64-4.22, $P = .022$).

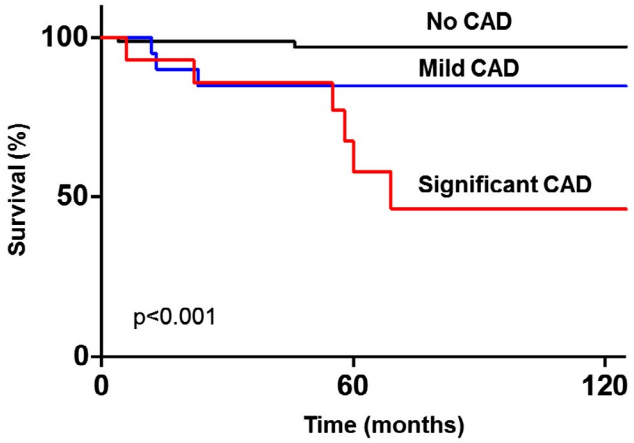

FIGURE 2 Kaplan-Meier curves comparing survival between patients with no CAD (black), mild CAD (blue), and significant CAD (red)

4 | DISCUSSION

In this study of a selected cohort of 105 adult TOF patients that underwent aortic root/selective coronary angiogram, the prevalence of mild and significant CAD were 19% and 15%, respectively. Although there is very robust literature about diagnosis, treatment, and outcomes of CAD in the general population,[5] there are limited data about CAD in adult with congenital heart disease.[4,6,9,10] There are two prior studies that reported the prevalence of CAD in patients with congenital heart disease.[4,9] One of these studies conducted with data from the multicenter CONgenital CORvitia (CONCOR) registry identified CAD in 55 patients out of 6904 patients (prevalence of 8%).[4] The second study identified 141 cases of CAD among 12, 124 patients (prevalence of 1%).[9] The wide variation in CAD prevalence in both studies reflect differences in how CAD was defined in both studies, demographics of the study population and maybe due to the different types of congenital heart disease included in both studies. In contrast to these two prior studies that reported CAD prevalence based on population adult congenital heart disease patients with predominance of simple congenital lesions, the CAD prevalence in the current study was obtained specifically from a cohort of TOF patients. However, it is important to highlight the fact that the prevalence of 19% and 15% for mild and significant CAD, respectively, reported in the current study is likely an overestimation of the true population risk since we studied only a selected cohort that had angiograms, and these patients had more atherosclerotic cardiovascular disease (ASCVD) risk factors in comparison to those without angiograms (Supplementary Table 1). The prevalence of ASCVD risk factors reported in a community-based study conducted in Olmsted County was comparable to the observed prevalence ASCVD risk factors in the current study suggesting that the risk profile of our study population was reflected that of the general population.[11]

The yield of coronary angiogram (proportion of angiogram that showed significant CAD) was 15% and 38% for patients aged 40-59 year and ≥60 years, respectively. None of the patients that had angiogram before the age of 40 years had significant CAD. Coronary angiogram is recommended prior to valve surgery in men older than 40 years and postmenopausal women, or earlier for patients with other ASCVD risk factors.[12,13] The indication for aortic root/coronary angiogram in the 24 patients (23% of the cohort) that were <40 years of age was mostly due to the presence of other ASCVD risk factors or LV dysfunction. It is also important to highlight that all these angiograms were performed during comprehensive left and right heart catheterization (not as a *stand-alone* procedure), which arguably did not significantly increase the risk of the procedure. Notwithstanding, the results of this study suggests very low yield for coronary angiogram in this age groups because of low pretest probability of CAD in these patients.

The guidelines for the management of adults with congenital heart disease and the bulk of clinical research in this population focus primarily on the identification, treatment, and outcomes of therapy for residual/recurrent lesions due to the underlying congenital heart disease.[14-16] Although, CAD is an expected morbidity that occurs with aging, there is still very little research on CAD outcomes in the aging

TABLE 2 Baseline characteristics of patients that had coronary/aortic root angiogram

	No CAD (*n* = 69)	Mild CAD (*n* = 20)	Significant CAD (*n* = 16)	*P*	*P**
Age at angiogram, years	44 ± 11	52 ± 10	59 ± 11	.002	<.001
Male	40 (58%)	12 (60%)	12 (75%)	.342	.208
Body mass index, kg/m^2	28 ± 8	30 ± 9	27 ± 4	.382	.537
Body surface area, m^2	1.9 ± 0.2	2.0 ± 0.3	2.0 ± 0.1	.157	.111
Age at TOF repair, years	6 (2-10)	10 (6-32)	15 (4-28)	.007	.042
Prior palliative shunt	40 (58%)	10 (50%)	6 (38%)	.527	.139
Comorbidities					
Atrial fibrillation	21 (30%)	12 (60%)	11 (69%)	.016	.004
Atrial flutter/ tachycardia	26 (38%)	8 (40%)	4 (25%)	.851	.339
Hypertension	29 (42%)	13 (65%)	6 (38%)	.070	.740
Hyperlipidemia	37 (54%)	17 (85%)	12 (75%)	.011	.119
Current or prior smoker	15 (22%)	6 (30%)	3 (20%)	.444	.789
Diabetes mellitus	12 (17%)	7 (35%)	3 (19%)	.091	.897
Sleep apnea	27 (39%)	83 (23%)	6 (38%)	.015	.904
Creatinine, mg/dl	0.94 ± 0.21	1.11 ± 0.32	1.35 ± 0.41	.052	.003
Echocardiography					
≥Moderate RV systolic dysfunction*	28 (41%)	12 (60%)	6 (38%)	.124	.821
Lateral E/e'	7 ± 3	8 ± 3	7 ± 2	.579	.597
LV ejection fraction	54 ± 10	52 ± 13	51 ± 8	.268	.092

Abbreviations: CAD, coronary artery disease; LV, left ventricle; RV, right ventricle; TOF, tetralogy of Fallot.
P: comparison between No CAD and mild CAD; *P**: comparison between No CAD and significant CAD.

population of adults with congenital heart disease. The current study highlights the importance of CAD both in terms of disease prevalence and its impact on mortality. Since CAD prevalence will continue to rise in this population because of aging, and the current study demonstrates its negative impact on survival, perhaps there should be more emphasis of ASCVD risk factor modification and early diagnosis and treatment in this population. About 56% (9 of 16) of the patients with significant CAD had LV dysfunction of which 38% (6 of 16) had moderate/severe LV dysfunction at the time of a diagnosis. Although the current study was not designed or powered to determine the specific cause of LV dysfunction (as some patients might have LV dysfunction due to previous cardiac surgery unrelated to their CAD diagnosis) or the impact of LV dysfunction on CAD outcomes, we speculate that perhaps, early detection and revascularization may improve survival in these patients.

4.1 | Clinical application and future directions

The current study raises three important issues regarding clinical practice and future research. First is the need for adequate ASCVD risk factor modification. More that two-thirds of the patients that underwent coronary angiogram had at least one ASCVD risk factors. Similarly, more than one-thirds of the rest of the TOF patients (Supplementary Table 1) had a least one ASCVD risk factor. Considering the high prevalence of ASCVD in this population, pharmacologic and nonpharmacologic strategies to modify these risk factors should be a central focus of daily clinical practice and clinical research. Pharmacologic interventions should include a more aggressive use of statin therapy in patients with appropriate indication for primary and secondary prevention therapy. Next, since more than half of the patients with significant CAD already had LV dysfunction at the time of coronary angiogram, there is need for more research to assess diagnostic and prognostic use of noninvasive screening tests for early identification of CAD. Unlike the noncongenital population, the use of exercise electrocardiogram, stress echocardiogram or nuclear imaging may results in false positive readings in adults with congenital heart disease because of prior myocardial injury related to previous cardiac surgeries, hence the need for more research to identify the optimal screening strategies. Third, there is need for more

research to improve patient selection, and identify optimal timing and methods of revascularization. The current guidelines recommend revascularization in the setting of ischemic symptoms, LV dysfunction, or involvement of the left main coronary artery,[5] but we do not know how well these recommendations will apply to patients with preexisting subtle or overt myocardial injury from previous cardiac surgeries.

4.2 | Limitations

This study was based on a selected cohort of adult TOF patients that underwent aortic root/selective coronary angiogram, and whose clinical characteristics differ from that of the target population of all adult TOF patients thereby limiting generalizability of the results. Notwithstanding, the take home message is that CAD is an emerging problem in this population, and that ASCVD risk factor modification, and early detection and treatment will potentially improve survival. The study was unable to determine relative effectiveness of the different CAD therapy because of small sample size and selection bias in the allocation of therapy.

5 | CONCLUSIONS

Based on a retrospective review of a selected cohort of 105 TOF patients that underwent aortic root and coronary angiograms, the prevalence of mild CAD and significant CAD was 19% and 15%, respectively. The yield of coronary angiogram (proportion of patients with significant CAD) was 0%, 15%, and 38% for patients aged <40 years, 40-59 years, and ≥60 years, respectively, at the time of angiogram. Significant CAD was an independent risk factor for mortality. The results of the current study supports the importance of optimal ASCVD risk factor modification, and the need for more research to address the issue of optimal noninvasive diagnostic strategies as well as optimal patient selections and methods for revascularization.

AUTHOR CONTRIBUTIONS

Study design: Egbe, Ananthaneni, Jadav, Kothapalli
Data collection: Egbe, Ananthaneni, Jadav, Kothapalli
Data analysis: Egbe, Ananthaneni, Jadav, Kothapalli
Manuscript drafting: Egbe, Ananthaneni, Jadav, Kothapalli
Critical revision: Egbe, Ananthaneni, Jadav, Kothapalli, Rihal, Masood, Angirekula, Najam, Bajwa, Tarek, Matthew, Connolly
Final review/approval: Egbe, Ananthaneni, Jadav, Kothapalli, Rihal, Masood, Angirekula, Najam, Bajwa, Tarek, Matthew, Connolly
Manuscript drafting: Rihal, Masood, Angirekula, Najam, Bajwa, Tarek, Matthew, Connolly

ORCID

Alexander C. Egbe https://orcid.org/0000-0002-8810-3631

REFERENCES

1. Lillehei CW, Cohen M, Warden HE, et al. Direct vision intracardiac surgical correction of the tetralogy of Fallot, pentalogy of Fallot, and pulmonary atresia defects; report of first ten cases. *Ann Surg*. 1955;142:418-442.
2. Murphy JG, Gersh BJ, Mair DD, et al. Long-term outcome in patients undergoing surgical repair of tetralogy of Fallot. *N Engl J Med*. 1993;329:593-599.
3. Nollert G, Fischlein T, Bouterwek S, Bohmer C, Klinner W, Reichart B. Long-term survival in patients with repair of tetralogy of Fallot: 36-year follow-up of 490 survivors of the first year after surgical repair. *J Am Coll Cardiol*. 1997;30:1374-1383.
4. Bokma JP, Zegstroo I, Kuijpers JM, et al. Factors associated with coronary artery disease and stroke in adults with congenital heart disease. *Heart*. 2018;104:574-580.
5. Fihn SD, Gardin JM, Abrams J, et al. 2012 ACCF/AHA/ACP/AATS/PCNA/SCAI/STS Guideline for the diagnosis and management of patients with stable ischemic heart disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, and the American College of Physicians, American Association for Thoracic Surgery, Preventive Cardiovascular Nurses Association, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *J Am Coll Cardiol*. 2012;60:e44-e164.
6. Bokma JP, Winter MM, Kuijpers JM, et al. Role of acquired cardiovascular disease in tetralogy of Fallot patients >50 years of age. *J Am Coll Cardiol*. 2017;69:2465-2466.
7. Egbe A, Uppu S, Stroustrup A, Lee S, Ho D, Srivastava S. Incidences and sociodemographics of specific congenital heart diseases in the United States of America: an evaluation of hospital discharge diagnoses. *Pediatr Cardiol*. 2014;35:975-982.
8. Gilboa SM, Devine OJ, Kucik JE, et al. Congenital heart defects in the United States: estimating the magnitude of the affected population in 2010. *Circulation*. 2016;134:101-109.
9. Yalonetsky S, Horlick EM, Osten MD, Benson LN, Oechslin EN, Silversides CK. Clinical characteristics of coronary artery disease in adults with congenital heart defects. *Int J Cardiol*. 2013;164:217-220.
10. Stulak JM, Dearani JA, Burkhart HM, Ammash NM, Phillips SD, Schaff HV. Coronary artery disease in adult congenital heart disease: outcome after coronary artery bypass grafting. *Ann Thorac Surg*. 2012;93:116-122; discussion 122-123.
11. Gerber Y, Weston SA, Redfield MM, et al. A contemporary appraisal of the heart failure epidemic in Olmsted County, Minnesota, 2000 to 2010. *JAMA Intern Med*. 2015;175:996-1004.
12. Baumgartner H, Falk V, Bax JJ, et al. 2017 ESC/EACTS guidelines for the management of valvular heart disease. *Eur Heart J*. 2017;38:2739-2791.
13. Nishimura RA, Otto CM, Bonow RO, et al. AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2014;2014(63):2438-2488.
14. Stout KK, Daniels CJ, Aboulhosn JA, et al. 2018 AHA/ACC guideline for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2019;73(12):e81-e192.
15. Baumgartner H, Bonhoeffer P, De Groot NM, et al. Task Force on the Management of Grown-up Congenital Heart Disease of the European Society of C, Association for European Paediatric C and guidelines ESCCfP. ESC guidelines for the management of grown-up congenital heart disease (new version 2010). *Eur Heart J*. 2010;31:2915-2957.
16. Silversides CK, Marelli A, Beauchesne L, et al. Canadian Cardiovascular Society 2009 Consensus Conference on the management of adults with congenital heart disease: executive summary. *Can J Cardiol*. 2010;26:143-150.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

How to cite this article: Egbe AC, Ananthaneni S, Jadav R, et al. Coronary artery disease in adults with tetralogy of Fallot. *Congenital Heart Disease*. 2019;14:491–497. https://doi.org/10.1111/chd.12782