ilarly, GLP-1RA treatment produced a nonsignificant decrease in the risk for MACE, equal to 7% (RR 0.93, 95% CI 0.69 to 1.25, $I^2 = 49\%$), as shown in Figure 2.

Our meta-analysis updates the results of a previous meta-analysis performed by Mishriky et al,[20] incorporating data from the recently published CVOTs.

In conclusion, neither SGLT-2 inhibitors nor GLP-1RAs provide significant cardiovascular benefit in black patients, in terms of MACEs incidence. Future trials enrolling a larger proportion of black patients will clarify whether these observations are due to inadequate power of present CVOTs, or due to true racial disparities with the use of novel antidiabetics.

Disclosures

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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30 September 2020

1. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, Colagiuri S, Guariguata L, Motala AA, Ogurtsova K, Shaw JE, Bright D, Williams R, IDF Diabetes Atlas Committee. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. *Diabetes Res Clin Pract* 2019;157:107843.
2. Pham TM, Carpenter JR, Morris TP, Sharma M, Petersen I. Ethnic differences in the prevalence of type 2 diabetes diagnoses in the UK: cross-sectional analysis of the health improvement network primary care database. *Clin Epidemiol* 2019;11:1081–1088.
3. Clements JM, West BT, Yaker Z, Lauinger B, McCullers D, Haubert J, Tahboub MA, Everett GJ. Disparities in diabetes-related multiple chronic conditions and mortality: the influence of race. *Diabetes Res Clin Pract* 2020;159:107984.
4. Carnethon MR, Pu J, Howard G, Albert MA, Anderson CAM, Bertoni AG, Mujahid MS, Palaniappan L, Taylor HA Jr, Willis M, Yancy CW. American Heart Association Council on Epidemiology and Prevention. Council on Cardiovascular Disease in the Young. Council on Cardiovascular and Stroke Nursing; Council on Clinical Cardiology. Council on Functional Genomics and Translational Biology. Stroke Council. Cardiovascular Health in African Americans: a scientific statement from the American Heart Association. *Circulation* 2017;136:e393–e423.
5. Afkarian M, Katz R, Bansal N, Correa A, Kestenbaum B, Himmelfarb J, de Boer IH, Young B. Diabetes, kidney disease, and cardiovascular outcomes in the Jackson Heart Study. *Clin J Am Soc Nephrol* 2016;11:1384–1391.
6. Zinman B, Wanner C, Lachin JM, Fitchett D, Bluhmki E, Hantel S, Mattheus M, Devins T, Johansen OE, Woerle HJ, Broedl UC, Inzucchi SE, EMPA-REG OUTCOME Investigators. Empagliflozin, cardiovascular outcomes, and mortality in type 2 diabetes. *N Engl J Med* 2015;373:2117–2128.
7. Neal B, Perkovic V, Mahaffey KW, de Zeeuw D, Fulcher G, Erondu N, Shaw W, Law G, Desai M, Matthews DR, CANVAS Program Collaborative Group. Canagliflozin and Cardiovascular and renal events in type 2 diabetes. *N Engl J Med* 2017;377:644–657.
8. Wiviott SD, Raz I, Bonaca MP, Mosenzon O, Kato ET, Cahn A, Silverman MG, Zelniker TA, Kuder JF, Murphy SA, Bhatt DL, Leiter LA, McGuire DK, Wilding JPH, Ruff CT, Gause-Nilsson IAM, Fredriksson M, Johansson PA, Langkilde AM, Sabatine MS, DECLARE–TIMI 58 Investigators. Dapagliflozin and cardiovascular outcomes in type 2 diabetes. *N Engl J Med* 2019;380:347–357.
9. Cannon CP, Pratley R, Dagogo-Jack S, Mancuso J, Huyck S, Masiukiewicz U, Charbonnel B, Frederich R, Gallo S, Cosentino F, Shih WJ, Gantz I, Terra SG, Cherney DZI, McGuire DK, VERTIS CV Investigators. Cardiovascular outcomes with ertugliflozin in type 2 diabetes. *N Engl J Med* 2020. https://doi.org/10.1056/NEJMoa2004967. Epub ahead of print.
10. Pfeffer MA, Claggett B, Diaz R, Dickstein K, Gerstein HC, Køber LV, Lawson FC, Ping L, Wei X, Lewis EF, Maggioni AP, McMurray JJ, Probstfield JL, Riddle MC, Solomon SD, Tardif JC, ELIXA Investigators. Lixisenatide in patients with type 2 diabetes and acute coronary syndrome. *N Engl J Med* 2015;373:2247–2257.
11. Holman RR, Bethel MA, Mentz RJ, Thompson VP, Lokhnygina Y, Buse JB, Chan JC, Choi J, Gustavson SM, Iqbal N, Maggioni AP, Marso SP, Öhman P, Pagidipati NJ, Poulter N, Ramachandran A, Zinman B, Hernandez AF, EXSCEL Study Group. Effects of once-weekly exenatide on cardiovascular outcomes in type 2 diabetes. *N Engl J Med* 2017;377:1228–1239.
12. Marso SP, Daniels GH, Brown-Frandsen K, Kristensen P, Mann JF, Nauck MA, Nissen SE, Pocock S, Poulter NR, Ravn LS, Steinberg WM, Stockner M, Zinman B, Bergenstal RM, Buse JB. LEADER Steering Committee. LEADER Trial Investigators. Liraglutide and cardiovascular outcomes in type 2 diabetes. *N Engl J Med* 2016;375:311–322.
13. Marso SP, Bain SC, Consoli A, Eliaschewitz FG, Jódar E, Leiter LA, Lingvay I, Rosenstock J, Seufert J, Warren ML, Woo V, Hansen O, Holst AG, Pettersson J, Vilsbøll T, SUSTAIN-6 Investigators. Semaglutide and cardiovascular outcomes in patients with type 2 diabetes. *N Engl J Med* 2016;375:1834–1844.
14. Hernandez AF, Green JB, Janmohamed S, D'Agostino RB Sr, Granger CB, Jones NP, Leiter LA, Rosenberg AE, Sigmon KN, Somerville MC, Thorpe KM, McMurray JJV, Del Prato S, Harmony Outcomes committees and investigators. Albiglutide and cardiovascular outcomes in patients with type 2 diabetes and cardiovascular disease (Harmony Outcomes): a double-blind, randomised placebo-controlled trial. *Lancet* 2018;392:1519–1529.
15. Gerstein HC, Colhoun HM, Dagenais GR, Diaz R, Lakshmanan M, Pais P, Probstfield J, Riesmeyer JS, Riddle MC, Rydén L, Xavier D, Atisso CM, Dyal L, Hall S, Rao-Melacini P, Wong G, Avezum A, Basile J, Chung N, Conget I, Cushman WC, Franek E, Hancu N, Hanefeld M, Holt S, Jansky P, Keltai M, Lanas F, Leiter LA, Lopez-Jaramillo P, Cardona Munoz EG, Pirags V, Pogosova N, Raubenheimer PJ, Shaw JE, Sheu WH, Temelkova-Kurktschiev T, REWIND Investigators. Dulaglutide and cardiovascular outcomes in type 2 diabetes (REWIND): a double-blind, randomised placebo-controlled trial. *Lancet* 2019;394:121–130.
16. Husain M, Birkenfeld AL, Donsmark M, Dungan K, Eliaschewitz FG, Franco DR, Jeppesen OK, Lingvay I, Mosenzon O, Pedersen SD, Tack CJ, Thomsen M, Vilsbøll T, Warren ML, Bain SC, PIONEER 6 Investigators. Oral Semaglutide and cardiovascular outcomes in patients with type 2 diabetes. *N Engl J Med* 2019;381:841–851.
17. Deeks JJ, Higgins JPT, Altman DG (2011) Chapter 9: analyzing data and undertaking meta-analyses. In: Higgins JPT, Green S (eds) Cochrane Handbook for Systematic Reviews of Interventions Version 5.1.0 [Updated March 2011], The Cochrane Collaboration.
18. *Review Manager (RevMan) [Computer program] Version [5.3]*. Copenhagen: The Nordic Cochrane Centre TCC; 2014.
19. Higgins JPT, Sterne JAC, Savović J, Page MJ, Hróbjartsson A, Boutron I, Reeves B, Eldridge S. A revised tool for assessing risk of bias in randomized trials. *Cochrane Database Syst Rev* 2016;10:29–31.
20. Mishriky BM, Powell JR, Wittwer JA, Chu JX, Sewell KA, Wu Q, Cummings DM. Do GLP-1RAs and SGLT-2is reduce cardiovascular events in black patients with type 2 diabetes? A systematic review and meta-analysis. *Diabetes Obes Metab* 2019;21:2274–2283.

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Handgrip Strength and Risk of Atrial Fibrillation

Consistent evidence suggests inverse and independent associations between

handgrip strength (HGS) and cardiovascular outcomes. However, whether HGS is specifically related to future risk of atrial fibrillation (AF) is uncertain. We sought to assess the prospective association between HGS and risk of AF. HGS was assessed using a hand dynamometer in a general population-based cohort of 827 men and women aged 61 to 74 years with no history of AF at study entry. Absolute values of HGS were allometrically scaled to account for the effect of body weight (HGS/body weight$^{2/3}$) and to normalize the data. Hazard ratios (HRs) with 95% confidence intervals (CIs) were estimated for AF. During a median (interquartile range) follow-up of 15.7 (9.5 to 18.8) years, 265 AF cases were recorded. The HR (95% CI) for AF per 1 standard deviation increase in normalized HGS in age- and gender-adjusted analysis was 0.73 (0.61 to 0.86). The association remained similar in analyses adjusted for several established and emerging risk factors 0.77 (0.64 to 0.92). The corresponding adjusted HRs (95% CIs) were 0.53 (0.39 to 0.73) and 0.60 (0.44 to 0.83), respectively, when comparing the top versus bottom tertiles of normalized HG. In conclusion, normalized HGS is inversely associated with the future risk of AF in the general population.

Atrial fibrillation (AF) is the most commonly diagnosed arrhythmia in clinical practice and is a leading cause of cardio-metabolic stroke.[1] Major modifiable risk factors that increase the risk of AF include obesity, obstructive sleep apnoea, high blood pressure, smoking, excessive alcohol consumption, and physical inactivity. Strategies that target these modifiable risk factors can help lower the burden of AF. Although a significant amount of health resources have been invested in the prevention, detection, and management of AF, it is still on the increase, and reasons for this increase remain elusive.[1] Though the increase may partly be due to the aging population and higher detection, it appears there may be other factors that may explain the residual risk of AF. Handgrip strength (HGS) is well known to be a measure of muscular strength and a useful indicator for general health status. Several studies have demonstrated HGS to be inversely associated with the risk of chronic disease outcomes such as cardiovascular disease (CVD), diabetes, osteoporotic fractures, as well as all-cause mortality.[2–5] Additional evidence suggests that HGS improves the prediction of outcomes such as CVD mortality and type 2 diabetes beyond that of conventional risk factors.[4,6] However, no previous study has demonstrated the existence of an association between HGS and AF. Given that AF is a cardiovascular outcome and hence share similar risk factors with CVD, we hypothesized that increased HGS would be associated with a lower risk of AF. In this context, we sought to assess the prospective association between HGS and risk of AF in a general population-based cohort of Finnish men and women with no history of AF at study entry.

We employed the Kuopio Ischemic Heart Disease Risk Factor study, a population-based prospective cohort study designed to investigate potential risk factors for atherosclerotic cardiovascular outcomes and other related chronic disease outcomes. Methods for participant recruitment, baseline physical examinations, blood sampling, and measurements have been described in previous reports.[4,6–8] Baseline examinations and measurements were performed between March 10, 1998 and February 2, 2000. A hand dynamometer was used to measure the HGS of the dominant hand for each participant (in kPa; Martin-Balloon-Vigorimeter; Gebrüder Martin, Tuttlingen, Germany). The mean of 2 measurements was used for analysis. The dynamometers were calibrated at the beginning of each test, and there was a 1-minute resting gap between both handgrip measurements. Absolute values of HGS were allometrically scaled to account for the influence of body weight and to normalize the data (normalized HGS = HGS/body weight$^{2/3}$)[6,9]. All results were multiplied by 100 for easier readability. All incident cases of AF from study entry through to 2018 were included, and no losses to follow-up were recorded. Participants with a baseline history of AF were excluded. The diagnostic classification of AF cases was conducted according to the International Classification of Diseases-10 codes (I48.0 to I48.9).[8,10,11] Each participant provided written informed consent. The institutional review board of the University of Kuopio and Kuopio University Hospital, Kuopio, Finland, approved the protocol (License number 143/97) in line with the Declaration of Helsinki. Hazard ratios (HRs) with their 95% confidence intervals (CIs) for AF were calculated using Cox proportional hazard models. All statistical analyses were conducted using Stata version MP 16 (Stata Corp, College Station, Texas).

The overall mean (standard deviation, SD) age of study participants at study entry was 69 (3) years, and 46% were males. The mean (SD) values of normalized HGS and body weight were 0.48 (0.23) kPa/kg$^{2/3}$ and 75.2 (12.9) kg respectively (Table 1). During a median

Table 1
Association between normalized handgrip strength and risk of atrial fibrillation

Normalized Handgrip Strength (kPa/kg$^{2/3}$)	Events/Total	Model 1		Model 2		Model 3	
		HR (95% CI)	p value	HR (95% CI)	p value	HR (95% CI)	p value
Per 1 SD increase	265 / 827	0.73 (0.61−0.86)	< .01	0.77 (0.64−0.92)	.004	0.77 (0.62−0.95)	.01
T1 (0.12−0.38)	103 / 276	ref		ref		ref	
T2 (0.39−0.52)	87 / 277	0.70 (0.53−0.94)	.02	0.75 (0.56−1.00)	.05	0.75 (0.49−1.13)	.17
T3 (0.53−4.03)	75 / 274	0.53 (0.39−0.73)	< .01	0.60 (0.44−0.83)	.002	0.61 (0.41−0.91)	.02

CI, confidence interval; HR, hazard ratio; ref, reference; SD, standard deviation; T, tertile.

Model 1: Adjusted for age and sex.

Model 2: Model 1 plus body height, total cholesterol, high-density lipoprotein cholesterol, systolic blood pressure, history of type 2 diabetes, heart rate, smoking status, prevalent coronary heart disease and physical activity.

Model 3: Model 2 plus alcohol consumption.

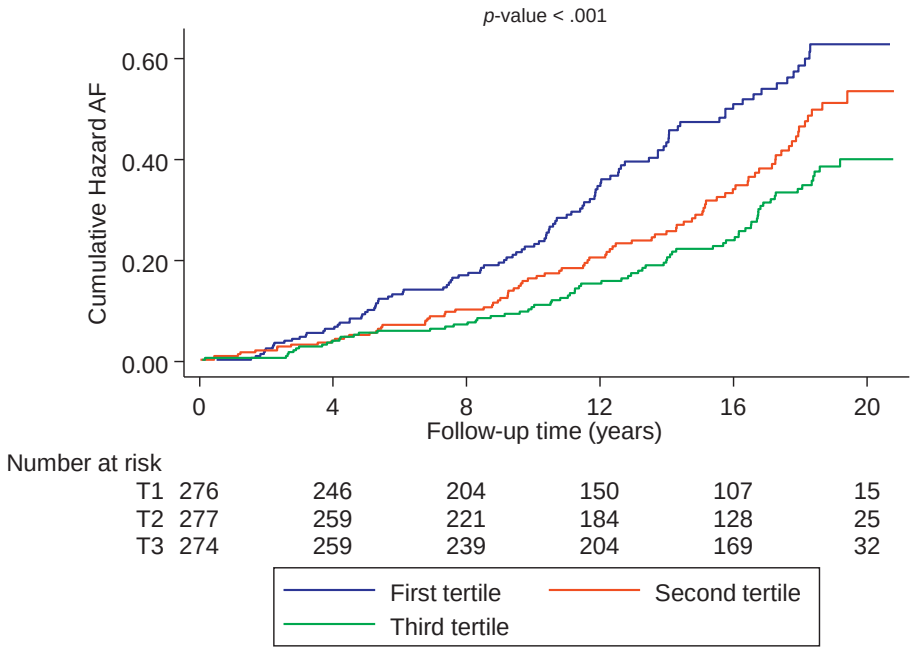

Figure 1. Cumulative hazard curves for atrial fibrillation according to the tertiles of normalized handgrip strength.

(interquartile range) follow-up of 15.7 (9.5 to 18.8) years, a total of 265 AF cases (annual rate 23.08/1,000 person-years at risk; 95% CI: 20.47 to 26.04) were recorded. Cumulative hazard curves demonstrated a lower risk of AF in patients in the top tertile of normalized HGS values compared with those in the bottom tertile ($p<0.001$ for log-rank test; Figure 1). The HR for AF per 1 SD increase in normalized HGS in age- and sex-adjusted analysis was 0.73 (95% CI: 0.61 to 0.86), which remained similar on further adjustment for established risk factors and other potential confounders (body height, total cholesterol, high-density lipoprotein cholesterol, systolic blood pressure, history of type 2 diabetes, resting heart rate, smoking status, prevalent coronary heart disease, and physical activity) 0.77 (95% CI: 0.64 to 0.92) (Table 1). The corresponding adjusted HRs were 0.53 (95% CI: 0.39 to 0.73) and 0.60 (95% CI: 0.44 to 0.83), respectively, when comparing the top versus bottom tertiles of normalized HGS. The HRs were unchanged in a third model that adjusted for alcohol consumption.

To our knowledge, only one prospective study has so far evaluated the association between HGS and risk of cardiac arrhythmias, including AF. Andersen and colleagues using a large Swedish cohort of military conscriptees demonstrated higher muscle strength (assessed by HGS) to be associated with a lower risk of arrhythmias.[12] However, they found no association with the specific outcome of AF. Furthermore, the study did not account for the effect of body weight on HGS values, given that body size is a key factor that explains muscle strength. Several cohort studies have demonstrated associations between high HGS and lower risk of vascular disease and mortality.[4,5] It has been suggested these effects may be mediated by a reduction in the incidence of vascular risk factors such as chronic inflammation, weight gain, abdominal adiposity, insulin resistance, and inflammation.[13] Hence, given that AF is also a vascular-related outcome, we postulate that similar pathways may explain the relation between high HGS and lower risk of AF. The current findings are relevant and add to the accumulating literature on the relation between muscular strength and vascular outcomes.

In conclusion, normalized HGS is inversely associated with the future risk of AF in the general population.

Disclosures

The authors declare that they have no known competing financial interests or personal relations that could have appeared to influence the work reported in this study.

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12 September 2020
2 October 2020

1. Morillo CA, Banerjee A, Perel P, Wood D, Jouven X. Atrial fibrillation: the current epidemic. *J Geriatr Cardiol* 2017;14:195–203.
2. Kunutsor SK, Isiozor NM, Khan H, Laukkanen JA. Handgrip strength - a risk indicator for type 2 diabetes: systematic review and meta-analysis of observational cohort studies. *Diabetes Metab Res Rev* 2020:e3365.
3. Laukkanen JA, Kunutsor SK, Yates T, Willeit P, Kujala UM, Khan H, Zaccardi F. Prognostic Arelevance of cardiorespiratory fitness as assessed by submaximal exercise testing for all-cause mortality: a UK biobank prospective study. *Mayo Clin Proc* 2020;95:867–878.
4. Laukkanen JA, Voutilainen A, Kurl S, Araujo CGS, Jae SY, Kunutsor SK. Handgrip strength is inversely associated with fatal cardiovascular and all-cause mortality events. *Ann Med* 2020;52:109–119.
5. Laukkanen JA, Voutilainen A, Kurl S, Isiozor NM, Jae SY, Kunutsor SK. Handgrip strength is inversely associated with sudden cardiac death. *Mayo Clin Proc* 2020;95:825–828.
6. Kunutsor SK, Voutilainen A, Laukkanen JA. Handgrip strength improves prediction of type 2 diabetes: a prospective cohort study. *Ann Med* 2020;52:471–478.
7. Kunutsor SK, Makikallio TH, Voutilainen A, Laukkanen JA. Handgrip strength is not associated with risk of venous thromboembolism: a prospective cohort study. *Scand Cardiovasc J* 2020;54:253–257.
8. Kunutsor SK, Laukkanen JA, Bluemke DA, Butler J, Khan H. Baseline and long-term gamma-glutamyltransferase, heart failure and cardiac arrhythmias in middle-aged finnish men: prospective study and pooled analysis of published evidence. *Eur J Prev Cardiol* 2016;23:1354–1362.
9. Jacobson BH, Thompson BJ, Conchola EC, Glass R. A comparison of absolute, ratio and allometric scaling methods for normalizing strength in elite American football players. *J Athl Enhancement* 2013;2:2.
10. Kunutsor SK, Laukkanen JA, Kurl S, Makikallio TH, Khan H. Leisure-time cross-country skiing and risk of atrial fibrillation and stroke: a prospective cohort study. *Eur J Prev Cardiol* 2020. 2047487319901040.
11. Khan H, Kella D, Rauramaa R, Savonen K, Lloyd MS, Laukkanen JA. Cardiorespiratory fitness and atrial fibrillation: a population-based follow-up study. *Heart rhythm* 2015; 12:1424–1430.
12. Andersen K, Rasmussen F, Held C, Neovius M, Tynelius P, Sundstrom J. Exercise capacity and muscle strength and risk of vascular disease and arrhythmia in 1.1 million young Swedish men: cohort study. *BMJ* 2015;351: h4543.
13. Artero EG, Lee DC, Lavie CJ, Espana-Romero V, Sui X, Church TS, Blair SN. Effects of muscular strength on cardiovascular risk factors and prognosis. *J Cardiopulm Rehabil Prev* 2012;32:351–358.

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Among Pediatric Patients Hospitalized for Influenza Infection, Pre-Existing Cardiomyopathy Confers Significantly Higher Morbidity and Mortality

Influenza infection generally presents as an acute self-limited and uncomplicated infection in healthy children, but children <5 years and those with chronic medical conditions are at risk of serious complications of influenza including pneumonia and acute respiratory failure. Recently we reported that in-hospital mortality and morbidity were increased in those with congenital heart disease hospitalized with influenza.[1] Although there are reports of increased mortality and complications in influenza in children with other chronic diseases[2,3] the literature is lacking information on complications of influenza infection in children with cardiomyopathy. Here, we report a recent nationwide retrospective study from Kids' Inpatient Database (KID) of the United States to examine risk of mortality and in-hospital complications of influenza infection in children with cardiomyopathy.

We analyzed data of hospital discharge records of patients with the nationally representative KID for years 2003, 2006, 2009, 2012, and 2016. Using International Classification of Disease, Ninth and Tenth Revision, Clinical Modification, we identified children hospitalized with cardiomyopathy, influenza infection and other variables studied. Cardiomyopathy includes dilated cardiomyopathy, hypertrophic cardiomyopathy, restrictive cardiomyopathy and other unspecified cardiomyopathy. We excluded children with myocarditis secondary to influenza to only include the children with pre-existing cardiomyopathy. The outcomes of interest were comparison of in-hospital mortality and morbidities between those children with influenza infection with and without concomitant cardiomyopathy. We adjusted for age, gender, race/ethnicity, hospital discharge quarter, year of admission and history of asthma, and presence of structural congenital heart disease for a logistic regression model to calculate outcome of interest. All statistical analyses were performed using R version 3.6.0,[4] R Studio 1.2[5] (http://www.R-project.org) and *survey* package.[6]

We retrieved records of 171,933 children who were admitted with a diagnosis of influenza over the 5 years of available data; 231 cases of influenza-related myocarditis were excluded from analysis. Of the remainder, 491 (0.3%) had a diagnosis of cardiomyopathy prior to the influenza admission. The in-hospital mortality rate for children hospitalized with influenza was 0.47% (n = 817). In-hospital mortality was higher in children with cardiomyopathy 6.5% (n = 32) compared with those without cardiomyopathy 0.5%(n = 785) [adjusted odds ratio 8.1(5.0 to 13.3), p <0.001]. Acute respiratory failure was more common among those with cardiomyopathy compared to those without cardiomyopathy, 28.0% (n = 137) versus 4.9% (n = 8,469) [aOR of 5.1(3.9 to 6.6), p <0.001]. Similarly, acute kidney injury was more common in those with cardiomyopathy versus without, 7.5% (n = 37) versus 0.9%(n = 1586) [aOR 3.9(2.4 to 6.3), p <0.001]. Children with cardiomyopathy were more likely to experience other complications as well (Table 1). Hospital stay in children with cardiomyopathy was longer than those without cardiomyopathy at median 6(the interquartile range 3 to 15) days versus 2 (the interquartile range 2 to 4) days, p <0.001.

Ours is the first study to report in-hospital outcomes in children with pre-existing cardiomyopathy who were hospitalized for influenza infection. We found that the presence of cardiomyopathy significantly increased the risk of in-hospital mortality and morbidity in children with influenza infection. The methodology does not allow us to pinpoint the exact etiology of this difference, but it is likely that co-morbid pulmonary disease and limited cardiac reserves are important factors. In addition, acute systemic inflammatory response caused by an influenza infection may lead to acute decompensated heart failure to an already failing myocardium.[7]

Influenza vaccination offers significant protection against severe influenza disease and influenza-related hospitalization.[8,9] Annual influenza vaccination is recommended for all children, but particularly for those with chronic diseases to prevent the serious complications from influenza.