

Diagnostic yield of 24-hour Holter monitoring in children presenting with chest pain, palpitations, or syncope: A single-center retrospective study

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ABSTRACT

Objective: The aims of this study were to evaluate the diagnostic yield of 24-hour Holter monitoring (HM) in children referred for chest pain, palpitations, and/or syncope, to identify independent predictors of clinically significant dysrhythmia and of the need for clinical intervention, and to assess the value of repeat HM.

Materials and Methods: Single-center retrospective study of 584 patients aged 6–18 years who underwent 24-hour HM between February 2023 and February 2024, grouped by presenting symptom (isolated palpitations, isolated chest pain, isolated syncope, or combined [≥ 2 symptoms]). HM findings were classified as normal, clinically non-significant arrhythmia, or severe dysrhythmia. Independent predictors were assessed by multivariable Firth penalized-likelihood logistic regression.

Results: Mean age was 13.4 years; 64.6% were female. HM was normal in 83.4%, non-significant in 12.7%, severe in 3.9%. Findings differed across groups ($p=0.007$; only chest pain vs syncope, $p=0.002$). No isolated syncope patient had a severe dysrhythmia or required intervention (0/108). Intervention was most frequent in isolated chest pain (13.2%), but 7 of 12 were empirical beta-blockers for benign ectopy or normal HM. An abnormal ECG was the only independent predictor of intervention (OR 6.78, 95% CI 3.15–14.29; $p<0.001$) and of severe dysrhythmia (OR 13.32, 95% CI 5.46–33.16; $p<0.001$); presenting symptom was not independently associated with either. Arrhythmia on the initial HM predicted later actionable arrhythmia (27.1% vs 2.3%). Of those with a normal initial HM, 10.2% underwent repeat HM and 12.0% showed a new finding. Family history of sudden death was not associated with severe dysrhythmia ($p=0.406$).

Conclusion: The diagnostic yield of HM varies by presenting symptom, but this variation is explained by ECG findings rather than the symptom itself. Arrhythmia on the initial HM strongly predicts subsequent intervention. No patient with isolated syncope had a severe dysrhythmia, although the retrospective single-center design precludes a definitive exclusion of risk.

Keywords: Ambulatory, arrhythmias, cardiac, child, electrocardiography, syncope

Introduction

Chest pain, palpitations, and syncope are among the most common reasons for pediatric cardiology referral and a frequent source of parental anxiety (1–3). The cause is benign in most children: a cardiac etiology accounts for only 0.3–8.0% of pediatric chest pain (3,4), and about 80% of pediatric syncope is neurally mediated, with cardiac syncope in only 1.5–6% (5,6). Nevertheless, life-threatening conditions such as supraventricular tachycardia (SVT), ventricular tachycardia (VT), Wolff–Parkinson–White (WPW) syndrome, long QT syndrome, cardiomyopathy, and congenital heart disease can present this way, requiring the clinician to identify rare but serious cases within a high-volume, predominantly benign caseload (5,7,8).

Standard 12-lead electrocardiography (ECG), detailed history, and physical examination form the initial diagnostic framework (5,8,9). However, pediatric arrhythmias are often paroxysmal and patients are usually asymptomatic during recording, so a single

surface ECG frequently cannot establish the symptom–rhythm relationship (1,2,9). Twenty-four-hour ambulatory ECG (Holter monitoring) (HM) is a non-invasive, guideline-recommended tool used to bridge this gap (8,10,11). Its diagnostic yield varies with the indication, approaching 30% with structural heart disease or an abnormal ECG but remaining 5–11% in otherwise healthy children with a normal ECG (1,2,11,12). Which subgroups benefit most, and where clinically significant arrhythmias concentrate, remain open questions.

Pediatric referrals for cardiac symptoms have increased after the COVID-19 pandemic, attributed both to organic mechanisms (post-acute SARS-CoV-2 sequelae, inappropriate sinus tachycardia, POTS, myocarditis, pericarditis) and to psychosocial factors (isolation, reduced activity, anxiety, heightened parental awareness) (13–18). How these translate into the diagnostic yield of HM has not been examined in a single large cohort.

We therefore aimed to evaluate the diagnostic yield of 24-hour HM in a large single-center cohort of children evaluated for chest pain, palpitations, and/or syncope, and to identify the clinical and electrocardiographic factors associated with clinically significant arrhythmia and with the need for clinical intervention.

Material and Methods

Study design

This single-center, hospital-based, retrospective cross-sectional study was conducted in accordance with the Declaration of Helsinki and the principles of Good Clinical Practice. Because of the retrospective design, the requirement for individual informed consent was waived by the ethics committee.

Study population and inclusion/exclusion criteria

Children aged 6–18 years who presented to the Pediatric Cardiology Clinic of Ankara Bilkent City Hospital with chest pain, palpitations, and/or syncope and underwent 24-hour HM between February 2023 and February 2024 were eligible, and were identified retrospectively through the hospital information management system. Exclusion criteria were: (i) inaccessible patient records or study variables; (ii) hemodynamically significant structural heart disease, collagen tissue disease, hyperthyroidism, anemia, or comparable comorbidity; (iii) lack of regular follow-up; and (iv) ongoing antiarrhythmic, antihypertensive, or steroid medication at presentation. Hemodynamically insignificant findings (patent foramen ovale, trivial or mild valvular regurgitation, other minor echocardiographic findings) were not exclusion criteria. After applying these criteria, 584 patients constituted the final analysis population.

Importantly, patients with high-risk syncope features were not excluded. All consecutive patients meeting these criteria were analyzed, including those with exertional syncope ($n=6$; 4 isolated syncope, 1 palpitations-plus-syncope, 1 chest-pain-plus-syncope), a family history of sudden death ($n=44$; 3 within isolated syncope), and an abnormal ECG. No patient had a history of syncope during swimming or competitive athletics.

The decision to perform HM was made by the treating pediatric cardiologist on clinical grounds, not by a standardized protocol; because only patients in whom HM was performed are included, the cohort is subject to referral and selection bias (addressed in the Limitations).

Data collection and variables

Demographic, clinical, and laboratory data were extracted retrospectively from the hospital information management system and patient records. Presenting symptoms were categorized as isolated palpitations, isolated chest pain, isolated syncope, or combined (≥ 2 symptoms). Associated complaints, family history (arrhythmia, sudden death, other cardiac disease, with degree of consanguinity), COVID-19-related data where available, physical examination findings, and laboratory parameters were recorded as detailed in Tables I and II.

Electrocardiography and echocardiography

All patients underwent a standard 12-lead surface ECG at presentation. ECG findings were classified as normal, clinically non-significant arrhythmia (e.g., isolated sinus tachycardia,

occasional supraventricular or ventricular ectopy, sinus arrhythmia), or clinically significant dysrhythmia (atrioventricular [AV] blocks, WPW or pre-excitation pattern, significant ST-segment changes, etc.). Transthoracic echocardiography was performed by a pediatric cardiologist in all patients; left ventricular systolic function, valvular regurgitation, and the presence of congenital heart disease were documented.

Holter monitoring and arrhythmia classification

All patients underwent three-channel digital 24-hour ambulatory ECG (Holter) monitoring (DMS 300-4A; DM Software Inc., Stateline, NV, USA). Recordings were processed by the device's software, then manually reviewed by a single pediatric cardiologist. Minimum, maximum, and mean heart rates and the type and frequency of arrhythmias were recorded, and patients/families time-stamped any symptoms during recording.

On the basis of the PACES/HRS expert consensus statement and the pediatric arrhythmia literature (10), HM findings were classified as (a) normal; (b) clinically non-significant arrhythmia; or (c) clinically significant (severe) dysrhythmia. The non-significant group included rare or very rare supraventricular and ventricular ectopy (SVE/VES), rare monomorphic VES, sinus arrhythmia, isolated first-degree AV block, wandering atrial pacemaker, and diurnal heart-rate variation. The significant group included frequent ($>10\%$) or polymorphic VES, VES couplets, non-sustained VT (nsVT), VT, SVT, WPW or pre-excitation, second-degree Mobitz II and third-degree AV block, and significant sinus pauses.

Separately from the HM classification, cases requiring medical antiarrhythmic therapy, electrophysiological study (EPS), catheter ablation, or implantable cardioverter-defibrillator (ICD) placement were classified as 'requiring clinical intervention.' This is a clinical outcome variable and is distinct from the Holter finding itself.

Repeat Holter monitoring and follow-up

In patients with a normal initial HM but persistent symptoms, repeat HM was performed at the discretion of the treating physician. The incremental yield of repeat HM was assessed among patients with a normal initial study who actually underwent at least one further recording. Follow-up duration, defined as the time from initial presentation to the last outpatient visit, was categorized as <6 months, ≥ 6 months to <1 year, and ≥ 1 year.

Outcomes

The primary outcome was the frequency and type of arrhythmias on 24-hour HM by presenting-symptom group. Secondary outcomes were independent predictors of clinically significant dysrhythmia and of the need for clinical intervention, the relationship between arrhythmia on the initial HM and subsequent intervention, the incremental yield of repeat HM after a normal initial study, and the association of a family history of sudden death with HM findings.

Statistical analysis

Data were analyzed using IBM SPSS Statistics (version 22.0; IBM Corp., Armonk, NY, USA) and R (version 4.3). Normality was assessed with the Shapiro–Wilk test. Normally distributed

variables are presented as mean $\pm$ SD, non-normally distributed variables as median (interquartile range, IQR), and categorical variables as number (%). Categorical comparisons used the Pearson chi-square test, with Monte Carlo exact p values when expected frequencies were inadequate and Fisher's exact test for selected 2 $\times$ 2 comparisons. Continuous variables were compared across groups by one-way ANOVA or Kruskal-Wallis test. Where significant, post-hoc pairwise comparisons used Bonferroni-adjusted Mann-Whitney U or chi-square tests, with significance set at $p < 0.0083$.

To determine whether the higher event rates observed in specific symptom groups were independent of potential confounders, multivariable logistic regression models were constructed with clinically significant dysrhythmia and, separately, the need for clinical intervention as dependent variables. Age, sex, presenting-symptom group, family history of sudden death, and the presence of an ECG abnormality were entered as independent variables, with isolated palpitations as the reference category. These predictors were selected a priori on the basis of clinical relevance and their established associations with pediatric arrhythmia, rather than by data-driven (stepwise) selection. Because no events occurred in the isolated syncope group, conventional maximum-likelihood estimation was subject to complete separation and did not converge; Firth's penalized-likelihood method was therefore used, and p values were derived from the penalized likelihood-ratio test. Odds ratios (OR) are reported with 95% confidence intervals (CI). Overall statistical significance was set at $p < 0.050$.

Results

Demographic and clinical characteristics

A total of 584 patients were analyzed. Mean age was 13.41 ± 3.11 years (range 6.0–18.0 years), and 64.6% ($n=377$) were female. Most presentations occurred in 2023 (79.6%, $n=465$), and 83.7% ($n=489$) were referred from within Ankara Bilkent City Hospital. Isolated palpitations were the most common presentation (39.7%, $n=232$), followed by isolated syncope (18.5%, $n=108$) and isolated chest pain (15.6%, $n=91$); 26.2% ($n=153$) presented with ≥ 2 symptoms simultaneously. The distribution of presenting symptoms is shown in Figure 1.

Demographic, clinical, and family-history characteristics by symptom group are summarized in Table I. Males were most frequent in isolated chest pain (49.5%), whereas females predominated in the other groups ($p=0.022$; post-hoc: isolated chest pain differed from both isolated palpitations and the combined group, $p=0.008$ for each). Age, year of presentation, and referral source did not differ. Follow-up duration was shortest in isolated syncope ($p < 0.001$; post-hoc: isolated chest pain versus isolated syncope). Symptom duration of >6 weeks was most common in the combined group (52.3%; $p < 0.001$; post-hoc: isolated syncope versus both isolated palpitations and the combined group). ≥ 2 HM recordings were most frequent in isolated chest pain (24.2%; $p=0.005$; post-hoc: isolated chest pain versus isolated syncope and versus the combined group). Comorbidity rates were similar ($p=0.127$). A family history of sudden death (14.3%; $p=0.018$; post-hoc: isolated chest pain versus isolated syncope) and of other cardiac disease (25.3%;

$p=0.014$; no pairwise comparison significant after Bonferroni correction) were both highest in isolated chest pain.

Associated complaints, physical examination, laboratory, ECG, and echocardiographic findings

Associated complaints included dizziness in 13.5%, dyspnea in 11.1%, and visual blackouts in 9.2%. Visual blackouts ($p < 0.001$), dizziness ($p < 0.001$), and exertional syncope ($p=0.011$) were significantly more frequent in the isolated syncope group (32.4%, 38.0%, and 3.7% respectively), whereas dyspnea was most frequent in the isolated chest pain (18.7%) and combined (16.3%) groups ($p < 0.001$). In post-hoc testing, visual blackouts and dizziness were each significantly more frequent in the isolated syncope group than in every other group (all $p < 0.001$), and dyspnea differed between isolated chest pain and isolated syncope and between isolated syncope and the combined group; the pairwise differences for exertional syncope did not remain significant after Bonferroni correction.

Physical examination, laboratory, ECG, and echocardiographic findings are shown in Table II. Median apical heart rate ($p=0.003$) and diastolic blood pressure ($p=0.016$) differed across groups, both localizing post-hoc to the isolated palpitations versus isolated syncope contrast. Cardiac enzyme elevation (2.4% overall) was concentrated in isolated chest pain (11.0%; $p < 0.001$; post-hoc: isolated chest pain versus each other group). On ECG, 14.4% ($n=84$) had a rhythm abnormality; this was most frequent in isolated chest pain (24.2%) and least in isolated syncope (3.7%; $p < 0.001$; post-hoc: isolated chest pain versus each other group), and no isolated syncope patient had a clinically significant ECG abnormality. Mild valvular regurgitation (25.3%; $p=0.014$; post-hoc: isolated chest pain versus isolated syncope) and hospitalization (17.6%; $p=0.007$; post-hoc: isolated chest pain versus isolated syncope) were also highest in isolated chest pain; left ventricular dysfunction (3.8%) and congenital heart disease (6.2%) did not differ.

COVID-19 history records were available for only 5.7% ($n=33$) of patients; a history of COVID-19 was documented in 3.3% ($n=19$), exposure history in 1.6%, and antibody positivity in 1.6%. Because of extensive missing data, COVID-19 status could not be tested as an independent variable.

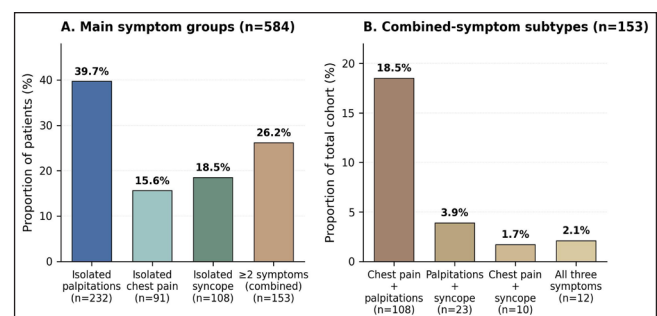


Figure 1: Distribution of presenting symptoms in 584 children referred for 24-hour Holter monitoring. (A) Main symptom groups: isolated palpitations, isolated chest pain, isolated syncope, and a combined group with ≥ 2 symptoms. (B) Subtypes within the combined group ($n=153$), expressed as a proportion of the total cohort ($n=584$).

Table I: Demographic, clinical, and family-history characteristics stratified by presenting-symptom group

Variable	Total	Isolated palpitations	Isolated chest pain	Isolated syncope	≥2 symptoms	p
Total number of patients	584	232	91	108	153	-
Age (years)*	13.41 ± 3.11	13.36 ± 3.45	13.06 ± 2.97	13.54 ± 2.59	13.60 ± 2.99	0.573
Female†	377 (64.6)	156 (67.2)	46 (50.5)	70 (64.8)	105 (68.6)	0.022
Year 2023†	465 (79.6)	187 (80.6)	74 (81.3)	87 (80.6)	117 (76.5)	0.731
Follow-up <6 months†	417 (71.4)	165 (71.1)	51 (56.0)	90 (83.3)	111 (72.5)	<0.001
Follow-up ≥1 year†	122 (20.9)	47 (20.3)	32 (35.2)	12 (11.1)	31 (20.3)	0.001
Symptom duration >6 weeks†	266 (45.6)	114 (49.1)	37 (40.7)	35 (32.4)	80 (52.3)	<0.001
≥2 Holter recordings†	79 (13.5)	34 (14.7)	22 (24.2)	10 (9.3)	13 (8.5)	0.005
Comorbidity present†	127 (21.7)	62 (26.7)	16 (17.6)	21 (19.4)	28 (18.3)	0.127
Family history of arrhythmia†	57 (9.8)	24 (10.3)	10 (11.0)	8 (7.4)	15 (9.9)	0.819
Family history of sudden death†	44 (7.5)	15 (6.5)	13 (14.3)	3 (2.8)	13 (8.6)	0.018
Family history of other cardiac disease†	105 (18.0)	35 (15.1)	23 (25.3)	12 (11.1)	35 (23.0)	0.014
Symptom-mimetic medication use†	11 (1.9)	6 (2.6)	0 (0.0)	0 (0.0)	5 (3.3)	0.095†
Hospitalization†	56 (9.6)	19 (8.2)	16 (17.6)	4 (3.7)	17 (11.2)	0.007

*: mean ± SD (One-way ANOVA), †: n(%) (Pearson chi-square test), ‡: Monte Carlo exact p value

Table II: Physical examination, laboratory, electrocardiographic, and echocardiographic findings stratified by presenting-symptom group

Variable	Total	Isolated palpitations	Isolated chest pain	Isolated syncope	≥2 symptoms	p
Total number of patients	584	232	91	108	153	-
Apical heart rate (bpm)*	82.0 (74.0–91.0)	83.5 (76–94)	80.5 (71–91)	79.0 (72–87)	82.0 (72–90)	0.003
Systolic BP (mmHg)*	110.0 (103.0–118.0)	111 (104–119)	110 (104–115)	109 (101–115)	110.5 (102–120)	0.098
Diastolic BP (mmHg)*	62.0 (58.0–69.0)	62 (58–70)	63.5 (59–69)	60 (55–67)	61 (58–69)	0.016
Murmur present†	12 (2.1)	5 (2.2)	2 (2.2)	3 (2.8)	2 (1.3)	0.865
Hemoglobin (g/dL)*	13.6 (13.0–14.4)	13.6 (13.0–14.5)	13.9 (13.2–14.6)	13.7 (13.2–14.4)	13.6 (12.9–14.4)	0.279
Cardiac enzyme elevation†	14 (2.4)	1 (0.4)	10 (11.0)	1 (0.9)	2 (1.3)	<0.001
ECG						
Normal†	500 (85.6)	198 (85.3)	69 (75.8)	104 (96.3)	129 (84.3)	0.004
Non-significant arrhythmia†	61 (10.4)	26 (11.2)	16 (17.6)	4 (3.7)	15 (9.8)	0.004
Clinically significant dysrhythmia†	23 (4.0)	8 (3.4)	6 (6.6)	0 (0.0)	9 (5.9)	0.004
LV systolic dysfunction†	22 (3.8)	8 (3.4)	7 (7.7)	3 (2.8)	4 (2.6)	0.190
Mild valvular regurgitation†	106 (18.2)	43 (18.5)	23 (25.3)	9 (8.3)	31 (20.3)	0.014
Congenital heart disease†	36 (6.2)	18 (7.8)	7 (7.7)	2 (1.9)	9 (5.9)	0.181

*: median (Q1–Q3) (Kruskal–Wallis test), †: n(%) (Pearson chi-square test), BP: blood pressure, ECG: electrocardiogram, LV: left ventricular

Holter monitoring findings

On 24-hour HM, 83.4% (n=487) of patients had a normal rhythm, 12.7% (n=74) had a clinically non-significant arrhythmia, and 3.9% (n=23) had a clinically significant dysrhythmia. The most common findings were rare or very rare supraventricular ectopy (5.8%) and rare or very rare ventricular ectopy (5.5%); WPW or pre-excitation, moderate-frequency VES, and AV block each occurred in ≤1.4%, and SVT (0.7%) and VT (0.3%) were rare.

Holter findings by symptom group are shown in Table III. The three-category distribution differed across groups (p=0.007); in post-hoc testing only the isolated chest pain versus isolated syncope contrast remained significant (p=0.002). Clinically non-significant arrhythmias were most frequent in isolated chest pain (23.1%); clinically significant dysrhythmia occurred in 3.9% of isolated palpitations, 5.5% of isolated chest pain, and 5.9% of the combined group, but in no isolated syncope patient (0.0%; one-sided 95% upper confidence limit 3.4%). Dichotomized, the difference in severe dysrhythmia was not significant (Monte Carlo exact p=0.088). Minimum, maximum, and mean heart rates did not differ (p=0.091, 0.074, and 0.216, respectively).

Clinical intervention and its indications

Thirty-seven patients (6.3%) required clinical intervention. The specific interventions were: medical antiarrhythmic therapy alone in 15; diagnostic EPS without ablation in 3; EPS with ablation alone in 12; medical therapy followed by EPS and ablation after treatment failure in 5; and EPS with ablation followed by medical therapy for symptom recurrence in 2. Overall, 22 patients received medical therapy at some point (metoprolol in 17, propranolol in 2, propafenone in 2, and verapamil subsequently changed to sotalol in 1), 22 underwent EPS, and 19 underwent ablation. No patient underwent ICD implantation.

The need for clinical intervention differed across groups (p=0.002), being highest in isolated chest pain (13.2%, 12/91) and absent in isolated syncope (0/108); isolated palpitations and combined groups were 6.9% and 5.9%. Because the 13.2% rate exceeds what the reported prevalence of clinically significant arrhythmia would predict, we reviewed each of the 12 interventions. Two patients had WPW or intermittent pre-excitation and underwent EPS with ablation; one had >10% supraventricular ectopy, one frequent supraventricular with rare

Table III: Twenty-four-hour Holter monitoring findings stratified by presenting-symptom group

Variable	Total	Isolated palpitations	Isolated chest pain	Isolated syncope	≥2 symptoms	p
Total number of patients	584	232	91	108	153	-
Holter result*						
Normal	487 (83.4)	197 (84.9)	65 (71.4)	96 (88.9)	129 (84.3)	0.007
Clinically non-significant	74 (12.7)	26 (11.2)	21 (23.1)	12 (11.1)	15 (9.8)	
Clinically significant dysrhythmia	23 (3.9)	9 (3.9)	5 (5.5)	0 (0.0)	9 (5.9)	
Specific arrhythmia subtype*						
Supraventricular ectopy	51 (8.7)	16 (6.9)	14 (15.4)	10 (9.3)	11 (7.2)	0.089
Ventricular ectopy	53 (9.1)	23 (9.9)	10 (11.0)	6 (5.6)	14 (9.2)	0.527
Supraventricular tachycardia	4 (0.7)	2 (0.9)	0 (0.0)	0 (0.0)	2 (1.3)	0.503
WPW / pre-excitation	8 (1.4)	2 (0.9)	2 (2.2)	0 (0.0)	4 (2.6)	0.245
Ventricular tachycardia	2 (0.3)	1 (0.4)	0 (0.0)	0 (0.0)	1 (0.7)	0.760
Other rhythm abnormality	38 (6.5)	16 (6.9)	8 (8.8)	5 (4.7)	9 (5.9)	0.680
Minimum HR (bpm) [†]	49.1±7.2	49.8±7.9	47.9±6.0	48.2±6.6	49.2±7.2	0.091
Maximum HR (bpm) [†]	155.9±17.7	155.4±19.1	153.0±16.1	155.2±17.9	158.8±15.8	0.074
Mean HR (bpm) [†]	82.8±12.0	83.8±12.8	82.7±15.5	80.9±10.6	82.7±8.7	0.216

*: (%) (Pearson chi-square test), †: mean±SD (one-way ANOVA), HR: heart rate, SD: standard deviation, WPW: Wolff-Parkinson-White

Table IV: Prognostic value of initial holter findings, incremental yield of repeat holter monitoring, and association of family history of sudden death with holter findings

Outcome	n	Values	p
Clinical intervention not required*			
Initial HM normal	488	477 (97.7)	<0.001
Initial HM dysrhythmia	96	70 (72.9)	
Clinical intervention required*			
Initial HM normal	488	11 (2.3)	-
Initial HM dysrhythmia	96	26 (27.1)	
Repeat holter monitoring [†]	488		
Underwent ≥1 repeat HM		50/488 (10.2)	-
New dysrhythmia among those repeated		6/50 (12.0) - (5.6–23.8)	
New dysrhythmia during follow-up, whole subgroup		9/488 (1.8) - (1.0–3.5)	
Normal*			
No history of sudden death	539	450 (83.5)	0.612
History of sudden death	44	36 (81.8)	
Clinically non-significant*			
No history of sudden death	539	69 (12.8)	0.406
History of sudden death	44	5 (11.4)	
Clinically significant dysrhythmia*			
No history of sudden death	539	20 (3.7)	0.406
History of sudden death	44	3 (6.8)	
Normal / clinically non-significant*			
No history of sudden death	539	519 (96.3)	0.406
History of sudden death	44	41 (93.2)	

*: n(%) (Pearson chi-square test or Fisher's exact test), †: n/N(%) (95% CI)

ventricular ectopy, and one moderate-frequency ventricular ectopy with a short ventricular run, all receiving medical therapy; the remaining seven received empirical beta-blocker therapy for rare ventricular ectopy (n=3) or an entirely normal HM (n=4). Thus only 2 of 91 patients (2.2%) underwent intervention for an unequivocally life-threatening arrhythmia, and the elevated aggregate rate reflects a low threshold for empirical therapy rather than a high burden of serious arrhythmia.

Recurrent dysrhythmia (p=0.001), the presence of dysrhythmia on the initial HM (p=0.022), and development of new dysrhythmia during follow-up (p=0.020) all peaked in the isolated chest pain group (post-hoc: for recurrent dysrhythmia, isolated chest pain versus isolated syncope; for dysrhythmia on the initial HM and for new dysrhythmia during follow-up, no pairwise comparison remained significant after Bonferroni correction).

Initial Holter findings, repeat Holter, and family history of sudden death

Initial HM findings, the incremental yield of repeat HM, and the association of family history of sudden death with HM findings are shown in Table IV. Among patients with dysrhythmia on the initial HM, 27.1% (26/96; 95% CI 19.2–36.7) subsequently required intervention versus 2.3% (11/488; 95% CI 1.3–4.0) with a normal initial HM (p<0.001).

Of the 488 patients with a normal initial HM, only 50 (10.2%) underwent at least one further recording. Among these 50 selected patients, 6 (12.0%; 95% CI 5.6–23.8) demonstrated a new dysrhythmia on repeat Holter monitoring. Across the whole subgroup with a normal initial study, a new dysrhythmia was identified during follow-up in 9 of 488 patients (1.8%; 95% CI 1.0–3.5) by any route; these 9 comprised the 6 detected on repeat Holter together with 3 additional patients in whom a new

Table V: Independent predictors of the need for clinical intervention and of clinically significant dysrhythmia: multivariable Firth penalized-likelihood logistic regression (n=583)

Variable	Intervention (37 events): OR (95% CI)	p	Severe dysrhythmia (23 events): OR (95% CI)	p
Abnormal ECG	6.78 (3.15–14.29)	<0.001	13.32 (5.46–33.16)	<0.001
Isolated chest pain	1.58 (0.67–3.61)	0.287	0.82 (0.24–2.56)	0.742
Isolated syncope	0.08 (<0.01–0.59)	0.007	0.17 (<0.01–1.46)	0.126
Combined (≥2 symptoms)	0.88 (0.36–2.01)	0.772	1.58 (0.58–4.24)	0.364
Family history of sudden death	0.97 (0.24–2.89)	0.963	1.94 (0.45–6.50)	0.342
Age (per year)	1.00 (0.90–1.11)	0.953	0.96 (0.85–1.10)	0.584
Male sex	0.90 (0.42–1.82)	0.766	1.35 (0.55–3.25)	0.507

OR: odds ratio, **CI:** confidence interval, **ECG:** electrocardiogram. Reference category for presenting symptom is isolated palpitations. Because no events occurred in the isolated syncope group, conventional maximum-likelihood estimation was subject to complete separation; Firth's penalized-likelihood method was used and *p* values were derived from the penalized likelihood-ratio test. Family-history data were unavailable in 1 patient (n=583).

dysrhythmia was identified by other clinical evaluation without a repeat Holter recording. Patients in the isolated chest pain group underwent ≥2 HM recordings more frequently than the other groups (24.2%, *p*=0.005; post-hoc: isolated chest pain versus isolated syncope and versus the combined group).

A family history of sudden death was documented in 44 patients (data unavailable in 1, leaving 583). It was not significantly associated with HM findings in either the three-category (*p*=0.612) or dichotomized analysis; severe dysrhythmia was numerically about two-fold higher with a positive family history (6.8% versus 3.7%) but not significant (Fisher's exact *p*=0.406).

Independent predictors of dysrhythmia and intervention

The multivariable Firth models are shown in Table V. After adjustment for age, sex, family history of sudden death, and ECG findings, an abnormal ECG was the only independent predictor of the need for intervention (OR 6.78, 95% CI 3.15–14.29; *p*<0.001) and of clinically significant dysrhythmia (OR 13.32, 95% CI 5.46–33.16; *p*<0.001). Presenting-symptom group was not independently associated with either outcome; the crude excess in isolated chest pain did not persist after adjustment (intervention OR 1.58, 0.67–3.61, *p*=0.287; severe dysrhythmia OR 0.82, 0.24–2.56, *p*=0.742). Isolated syncope remained protective against intervention (OR 0.08, 95% CI <0.01–0.59; *p*=0.007); because no intervention occurred in this group, its confidence interval was derived by the profile penalized-likelihood method, consistent with the likelihood-ratio *p* value. Age, sex, and family history of sudden death were not independently associated with either outcome.

Discussion

This single-center retrospective cohort study of 584 children evaluated with 24-hour HM for chest pain, palpitations, and/or syncope provides four main findings. First, the diagnostic yield of HM varied with the presenting symptom: HM was normal in 83.4%, clinically non-significant in 12.7%, and clinically significant in 3.9%, with differences across symptom groups driven by the isolated chest pain versus isolated syncope contrast. Second, no patient presenting with isolated syncope had a clinically significant dysrhythmia or required clinical intervention. Third, arrhythmia on the initial HM was strongly associated with subsequent clinically actionable arrhythmia (27.1% versus 2.3%). Fourth, and central to the interpretation of the first finding, multivariable analysis showed that the apparent symptom-based variation

in yield is explained by ECG findings rather than by the presenting symptom itself.

Reported HM yields in pediatric series vary widely. Hallioğlu et al. (7) found arrhythmia rates of 39.6% in syncope, 37.1% in palpitations, and 23.5% in chest pain in an eight-year retrospective series. Hegazy et al. (1) reported an overall HM yield of 10.7% in 1,319 patients, with much lower yields in palpitations (5.7%) and syncope (0.4%). Our overall yield (16.6% including non-significant arrhythmias; 3.9% clinically significant) falls between these reports. The 23.1% non-significant arrhythmia rate in our isolated chest pain group is comparable to the 23.5% reported by Hallioğlu et al. (7). Methodologically, a four-group by three-category chi-square shows only whether distributions differ, not where; our post-hoc testing localized the significant result to the chest pain versus syncope contrast, so we describe the distribution as heterogeneous rather than uniformly higher in any single group.

The isolated chest pain group: high testing, low arrhythmic burden

The interpretation of our isolated chest pain findings requires care. Although this group had the highest aggregate arrhythmia detection and intervention rate (13.2%), most detected arrhythmias were benign (23.1% non-significant versus 5.5% significant), and our case-by-case review found only two interventions for an unequivocally life-threatening arrhythmia (WPW ablation), with seven being empirical beta-blocker prescriptions for benign ectopy or a normal HM. The apparently high rate therefore reflects the breadth of our intervention definition and a low prescribing threshold, not a concentration of dangerous arrhythmia—reinforced by the multivariable analysis, in which isolated chest pain was not independently associated with either outcome once ECG findings were considered.

Consistent with this, cardiac enzyme testing (11.0%), mild valvular regurgitation (25.3%), a family history of cardiac disease (25.3%), hospitalization (17.6%), and repeat HM (24.2%) were all enriched in isolated chest pain, despite the low cardiac etiology reported in the literature (3,4,19,20). This is best explained by referral behavior: chest pain prompts a more extensive, more selective work-up that concentrates both benign findings and empirical treatment. Heightened post-pandemic somatic awareness and parental anxiety are documented drivers of such referrals (13–15,18,21). For practice, the chest pain group is the principal target for reducing unnecessary testing and empirical therapy, not one needing intensified surveillance.

The prognostic value of the initial Holter and the role of repeat monitoring

An important observation from our data is the prognostic value of the initial HM. Patients with dysrhythmia on the initial study were more than ten-fold more likely to require subsequent clinical intervention than those with a normal initial study (27.1% versus 2.3%; $p < 0.001$). This is consistent with prior reports indicating that a negative HM is associated with a low yield of new arrhythmia over one-year follow-up (12). For the practicing clinician, this finding supports the use of an initial 24-hour HM as a triage tool: a normal study identifies a low-risk subgroup.

Our repeat-HM data require careful framing. At the population level, a new dysrhythmia was identified during follow-up in only 1.8% of patients with a normal initial HM, and only 10.2% underwent any repeat recording. Among those repeated for persistent symptoms, however, the yield was 12.0% (95% CI 5.6–23.8)—comparable to the 13.1% reported for standard HM in an unselected symptomatic pediatric population (22). Repeating a 24-hour recording thus appears to reproduce the first recording's performance rather than add information beyond it. Since event recorders sampling over 14–30 days achieve yields approaching 62%, longer-duration recording is the more rational next step after a normal initial HM (22). The wide confidence interval ($n=50$) means this should be regarded as hypothesis-generating.

Isolated syncope

The absence of any clinically significant dysrhythmia or intervention in the isolated syncope group (0/108; one-sided 95% upper confidence limit 3.4%) is consistent with the predominance of neurally mediated mechanisms in pediatric syncope (~80%; cardiac syncope 1.5–6%) (5,6). This is strengthened because high-risk patients were not excluded: those with exertional syncope, a family history of sudden death, and any ECG abnormality were all eligible and analyzed. Notably, no patient in this group had a clinically significant ECG abnormality, consistent with our regression finding that ECG, not symptom, carries the predictive information.

From a practical standpoint, this may be among the more useful observations of our study: in a child with isolated syncope, a normal ECG, and no high-risk features, routine 24-hour HM is unlikely to alter management. This does not support a relaxed approach to high-risk syncope: exertional syncope, a family history of sudden death, or an abnormal ECG should still prompt detailed evaluation for channelopathies, hypertrophic cardiomyopathy, and conduction disease (5,8,23). Our family-history analysis was limited by the small subgroup ($n=44$): severe dysrhythmia was numerically about two-fold higher with a positive family history (6.8% versus 3.7%) but not significant ($p=0.406$) and likely underpowered.

The post-pandemic context

Our study period (2023–2024) immediately followed the COVID-19 pandemic, and post-acute cardiac autonomic dysregulation, impaired heart-rate variability, inappropriate sinus tachycardia, and POTS have been described in children after SARS-CoV-2 infection (16,17,24–26). However, objective

COVID-19 history was available in only 5.7% of our patients and no pre-pandemic comparator was available. Any link between the arrhythmias observed here and prior SARS-CoV-2 infection is therefore speculative, and we do not draw one.

Limitations

The principal strengths of this study are its relatively large pediatric cohort ($n=584$) and its focus on clinically actionable questions: symptom-stratified yield, the prognostic value of the initial HM, the incremental value of repeat HM, and the identification of independent predictors. Several limitations should be acknowledged.

First, the retrospective, single-center design limits generalizability. Second, and most importantly, the cohort comprises only patients in whom HM was performed, not all symptomatic children, and HM indications were not protocolized; the study is therefore subject to referral and selection bias, the most plausible explanation for the concentration of testing, benign findings, and empirical therapy in isolated chest pain, and a constraint on generalizing our yield estimates. Third, objective COVID-19 records were absent in most patients, precluding causal inference about pandemic-related changes and any pre-pandemic comparison. Fourth, some subgroups were small (e.g., family history of sudden death, $n=44$) and the multivariable models rested on only 37 and 23 events, giving a low events-per-variable ratio, wide confidence intervals, and hypothesis-generating estimates. Fifth, patients with hemodynamically insignificant minor echocardiographic findings were not excluded. Sixth, all HM tracings were read by a single pediatric cardiologist, so inter-observer agreement could not be assessed and observer bias cannot be excluded. Finally, the 24-hour window is inherently limited for detecting rare paroxysmal arrhythmias.

Conclusion

In children evaluated with 24-hour HM for chest pain, palpitations, and/or syncope, the diagnostic yield varies with the presenting symptom, but this is attributable to ECG findings rather than to the symptom itself: an abnormal ECG was the only independent predictor of both clinically significant dysrhythmia and the need for intervention.

In this cohort, no patient with isolated syncope had a clinically significant dysrhythmia or required intervention, even though high-risk patients (exertional syncope, family history of sudden death, abnormal ECG) were not excluded; this supports a limited role for routine HM in isolated syncope with a normal ECG and no high-risk features, although the single-center retrospective design means it should not be read as a definitive exclusion of risk.

Arrhythmia on the initial HM strongly predicts subsequent intervention, whereas repeat 24-hour HM after a normal study reproduces rather than exceeds the first recording's yield; longer-duration event recorders are the more rational next step. An individualized approach—incorporating ECG findings first, together with presenting symptom and family history—is likely to optimize yield and minimize unnecessary testing and empirical therapy, particularly in isolated chest pain. Confirmation will require multicenter prospective studies with prospectively recorded COVID-19 status.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study was approved by Ankara Bilkent City Hospital (19/03/2025, reference number: TABED 2-25-1026).

Contribution of the authors

Conception and design: AU, BK, SK, HAG, AEKG. Data acquisition: AU, BK, SK. Analysis and interpretation of data: AU, BK, SK, HAG, AEKG. Drafting of the manuscript: AU, BK, SK. Critical revision for important intellectual content: BK, SK, HAG, AEKG. Statistical analysis: AU, BK, SK. Supervision: AEKG. All authors read and approved the final version of the manuscript.

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Conflict of interest

The authors declare that there is no conflict of interest.

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