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Clinical Research Paper

Progression of esophageal atresia associated Barrett's esophagus in adulthood — Is endoscopic surveillance worth it?

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ABSTRACT

Objectives: Barrett's esophagus (BE) is a late sequela after esophageal atresia (EA) repair. In order to assess the need for an endoscopic BE surveillance program, we studied evolution of BE in 71 adults with C-type EA in two successive endoscopies.
Methods: Endoscopic Prague Classification of BE was used. Endoscopic BE was graded as True BE (with goblet cell metaplasia) or BE without goblet cell metaplasia.
Results: The median patient age was 36 (IQR 28–43) years at E1 (index endoscopy) and 50 (41–58) years at E2 (follow-up endoscopy) with median interval of 16 (12–17) years. Prevalence of endoscopic BE increased from 15 % to 42 % ($p = 0.002$) and of true BE from 1.4 % to 15 %, ($p = 0.04$). Dysplasia and cancer were not observed. *De novo* true BE ($n = 10$) developed from previous BE without goblet cell metaplasia ($n = 4$) or from normal epithelium ($n = 6$). In four (5.6 %) patients true BE extended ≥ 3 cm. True BE was predicted by previous endoscopic BE (RR = 9.2; 95%CI 2.0-4-1, $p = 0.004$) and esophagitis (RR = 5.8; 95%CI 1.4–38, $p = 0.02$).
Conclusions: The prevalence of endoscopic and true BE increased by 3- and 10-fold, respectively, between median ages of 36 and 50 years. High-risk BE was rare and no dysplasia or cancer was found. Esophagitis and endoscopic BE predisposed to true BE. Endoscopic surveillance of patients with EA before the age 50 years seems unnecessary.

1. Introduction

Esophageal atresia (EA) occurs in one of 3500 live births. Currently even the most complicated anatomical variants of EA can be successfully repaired. After repair, however, deficient esophageal motility [1,2] predisposes patients to complications such as gastro-esophageal reflux (GER). The most common GER-associated esophageal mucosal changes are esophagitis and the transformation of the squamous epithelium of the distal esophagus into areas of gastric epithelium or intestinal goblet cell containing columnar epithelium [3,4] signifying development of Barrett's esophagus. Barrett's esophagus is a premalignant mucosal lesion preceding esophageal adenocarcinoma [5–7]. Although the increased occurrence of Barrett's esophagus in patients with EA is well established, little is known about potential progression of esophageal metaplasia in adults with repaired EA [8]. Thus, the need for endoscopic surveillance in adulthood remains unclear. In the present study we addressed progression of Barrett's esophagus in adult patients with repaired Gross type C EA [9] at two endoscopic evaluations with 16 years apart. Our main outcome measure was the prevalence of Barrett's esophagus. Secondary aim was to identify the patient characteristics that are associated with progression of Barrett's esophagus.

2. Patients and methods

2.1. Patients

One hundred and one patients who underwent primary repair of Gross-C type esophageal atresia between 1947 and 1985 underwent index upper gastrointestinal tract endoscopy (E1) at median age 36 years between 2005 and 2008 [4]. They were reinvited for repeated endoscopic examination (E2) and questionnaires median 16 years later between 2019 and 2023 (Fig. 1).

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What is known

After repair of esophageal atresia in neonates, complications of GER include Barrett's esophagus and esophageal cancer. Is routine endoscopic follow-up for Barrett's esophagus necessary after transition from pediatric age?

What is new

Prevalence of endoscopic and true Barrett's Esophagus increased from 15 % to 42 % and from 1.4 % to 15 % between age 36 and 50 years. Only 5.6 % of the patients had long high-risk (>3 cm) true Barrett's esophagus, none had dysplasia or cancer. Endoscopic assessment before the age of 50 years seems unnecessary.

2.2. Assessment of Barrett's esophagus

Assessment of Barrett's esophagus was performed according to the European Society of Gastrointestinal Endoscopy (ESGE) standards. Prague Classification symbols for description of the height (cm) of circumferential (C) and tongue shaped (M) columnar epithelial areas were used [10].

Multiple-level biopsies were taken as described previously [4,10,11]. Standard histopathological criteria of Barrett's esophagus, dysplasia and cancer were used [12–15]. All biopsies demonstrating columnar metaplasia were examined using immunohistochemistry for caudal type homeobox 2 (CDX2) and mucin 2 (MUC2) to ascertain metaplasia as described in detail previously [4,12–15]. Esophagitis was graded according to the Los Angeles classification and confirmed by biopsies [16].

2.3. Survival

Survival data were obtained from the Cause-of -Death Registry, maintained by Statistics of Finland using unique personal identification codes [17].

2.4. Questionnaires

Identical questionnaires were used at E1 and E2. The questionnaires inquired about the presence of GER symptoms, proton pump inhibitor (PPI) medication, smoking habits, alcohol consumption, weight and height.

2.5. Definitions

The term endoscopic Barrett's esophagus referred to an endoscopic view of the distal esophagus that was consistent with the presence of columnar metaplasia with height $\geq$1 cm. The term true Barrett's esophagus' was used, if biopsies from the metaplastic area contained goblet cells. If the metaplastic area contained gastric epithelium without goblet cells the term Barrett's esophagus without goblet cell metaplasia' was used. If the endoscopy did not disclose Barrett's esophagus, or, the biopsies suggested an incidental focal area of goblet cells term normal endoscopic finding' was used.

2.6. Statistical analysis

Statistical calculations were performed using StatView 512 software (Brain Power, California, United States). Data is presented as percentages, frequencies, or medians with interquartile ranges (IQR) or simple ranges. Categorial variables were compared using Fischer's exact test, continuous variables with Mann–Whitney U-test and risk ratios were analyzed using logistic regression analysis with 95 % confidence intervals. Possible risk factors for Barrett's esophagus were chosen from those previously known for GER disease (GERD) in general population including smoking, alcohol

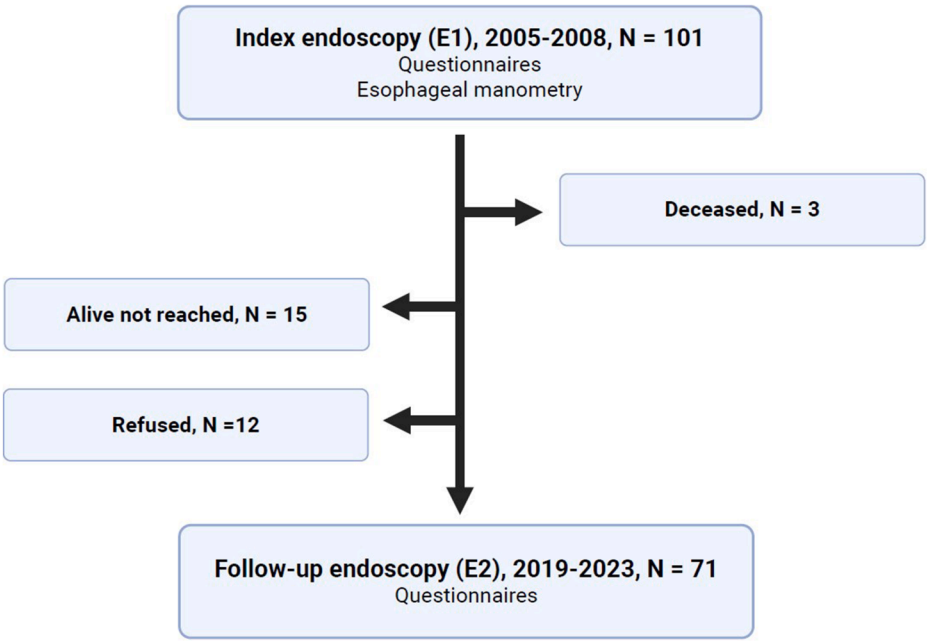

Fig. 1. Patient selection for follow-up endoscopy (E2).

consumption, body mass index (BMI), GER symptoms, PPI usage and age [6]. In addition, we chose risk factors that were found in our previous study to be statistically significantly associated with esophageal epithelial metaplasia in EA patients [4]. These included hiatal hernia, anastomotic complications after primary surgery (resection of stricture and/or recurrent tracheoesophageal fistula) and, fundoplication [4,5]. According to power analysis, to show an increase in prevalence of true Barrett's esophagus from 1.4 % at E1 to 15 % at E2 with power of 80 % and α-level of 0.05, required 69 patients. Statistical significance was set at $p < |0.05$.

2.7. Ethics

The study protocol was approved by the research ethics committee (HUS/447/E7/2004) and hospital review board (HUS/74/2023) and conformed to the Declaration of Helsinki. Informed consent was obtained from all the participants.

3. Results

A flow chart demonstrating patient recruitment for E2 is shown in Fig. 1. Of the 101 original patients, 98 were alive at the end of enrollment in 2019, while three patients had died of unrelated causes. Of the remaining 98 patients, 15 did not respond and 12 refused. Overall, 71(70 %) patients underwent E2 and completed questionnaires. The median interval between E1 and E2 was 16 [14–17] years.

Comparison of clinical patient characteristics between E1 and E2 is shown in Table 1 for patients who underwent both endoscopies. There was a fourfold increase in patient reported PPI usage from 7 % at E1 to 28 % at E2.

3.1. Evolution of endoscopic and histopathological findings at E1 and E2

As described earlier, at E1 none of the patients (n = 101) had cancer or dysplasia, 25 had histological esophagitis, and 21 had endoscopic Barrett's esophagus [4].

Table 1
Clinical data of 71 patients with type-C esophageal atresia who underwent endoscopic examination at E1 (index endoscopy, 2005-08) and E2 (follow -up endoscopy, 2019-23).

	E1	E2	p-value
Sex, males:females	40:31	40:31	
Age, years	36 (28–43)	50 (41–58)	<0.001
Associated morbidity			
Anorectal malformation n (%)		5 (7)	
Congenital heart disease (open heart surgery), n (%)		6 (8)	
Redo esophageal anastomotic surgery, n (%)		3 (4)	
Recurrent tracheoesophageal fistula surgery, n (%)		9 (13)	
Significant tracheomalacia, n (%)		12 (17)	
Fundoplication, n (%)		9 (13)	
Gastroesophageal reflux symptoms, n (%)	25 (35)	36 (51)	0.09
Proton pump inhibitor users, n (%)	7 (10)	28 (39)	<0.001
Smokers, n (%)	25 (35)	15 (21)	0.09
Smoking years, y	9 (5–14)	20 (15–23)	0.02
Smoking exposure[a]	3.6 (1.6–6.4)	7.8 (3.2–15)	0.06
Alcohol consumption			
None or light (monthly), n (%)	47 (66)	51 (72)	0.58
Moderate (weekly) or heavy (daily), n (%)	24 (34)	20 (28)	
Body mass index, kg/m^2	23 (21–27)	25 (23–27)	0.17

Data are frequencies or medians with interquartile ranges.
[a] Smoking years x 365 x (cigarettes/day) x 10^{-3}.

The endoscopic and histopathological findings of the 71 study patients at E1 and E2 are outlined in Table 2. During the 16-year interval between E1 and E2 there was a statistically significant increase in the prevalence of both endoscopic and true Barrett's esophagus from 15 % to 42 % ($p < 0001$) and 1.4 % to (15 %) ($p = 0.01$).

All patients with true Barrett's esophagus stained positive for both CDX2 and MUC2. Of the 30 cases with Barrett's esophagus without goblet cell metaplasia, 29 stained positively for CDX2 and four showed weak MUC2 positivity. No dysplasia or cancer was observed.

The prevalence of true Barrett's esophagus increased with time by 0.9 %/patient year (1077 patient years). Four of 11 (36 %) patients with endoscopic Barrett's esophagus without goblet cell metaplasia (174 patient years) and six (10 %) of 59 patients with normal endoscopy (903 patient years) developed *de novo* true Barrett's esophagus corresponding to an increase of 2.3 % and 0.7 %/patient year.

Of the 59 patients with normal endoscopy at E1, 21 (36 %) developed either true Barrett's esophagus (n = 6) or Barrett's esophagus without goblet cell metaplasia. Prague Classification of endoscopic Barrett's esophagus did not change statistically significantly.

The prevalence of esophagitis was similar at E1 and E2 (20 %). However, only two (2.8 %) patients had esophagitis at both endoscopies whereas 12 patients had esophagitis at E1 only and 12 patients had esophagitis at E2 only. All cases of esophagitis were classified as Los Angeles grade A, except for one case at E2 as grade B. At E2 two patients had esophagitis with moderate eosinophilic infiltration (20 eosinophils/high power field), but neither had symptoms nor endoscopic appearance consistent with eosinophilic esophagitis. The prevalence of esophagitis stratified by PPI medication is outlined in Table 2. Among the 14 patients with esophagitis at E2 nine had endoscopic Barrett's esophagus (true

Table 2
Endoscopic and histopathological findings in 71 patients with type-C esophageal atresia at E1 (index endoscopy 2005-08) and E2 (follow-up endoscopy 2019-23).

	E1		E2		p-value
Endoscopic BE, n (%)	12 (15)		30 (42)		0.002
• With goblet cell metaplasia, (= true BE), n (%)	1 (1.4)		11 (15)		0.04
• Without goblet cell metaplasia, n (%)	11 (15)		19 (27)		0.15
Prague Classification					
Endoscopic BE					
• C, cm	1 (0–2)		1 (0–2)		0.86
• M, cm	1 (1–3)		2 (1–2)		0.42
True BE					
• C, cm	3		2 (0–2)		0.32
• M, cm	3		2 (1–4)		0.74
• Maximum height≥3 cm, n	1		4		0.42
Esophagitis	12 (17)				
Esophagitis			12 (17)		
Esophagitis either at E1or E2	26 (37)				
Esophagitis both at E1 and E2	2 (2.8)				
Proton pump inhibitor users	Yes	No	Yes	No	
Esophagitis n (%)	1 (14)	13(20)	8 (29)	6 (14)	
No esophagitis	6 (86)	51(80)	20 (71)	37 (86)	
p-value	0.99		0.22		

Data are frequencies or medians with interquartile ranges. BE = Barrett's esophagus; C, circumferential height of Barrett's esophagus area; M, height of Barrett's esophagus tongue.

Barrett's esophagus n = 5, Barrett's esophagus without goblet cell metaplasia n = 4).

3.1.1. Predictors of Barrett's esophagus

The risk factors for development of endoscopic and true Barrett's esophagus at E2 are outlined in Table 3. According to logistic regression analysis, risk factors for both endoscopic and true Barrett's esophagus included pre-existing endoscopic Barrett's esophagus or Barrett's esophagus without goblet cell metaplasia. Additional risk factors for endoscopic Barrett's esophagus included male sex and esophagitis at E1 and, for true Barrett's esophagus, esophagitis at E2. Smoking exposure and alcohol consumption approached but did not reach statistical significance for predicting endoscopic or true Barrett's esophagus. Statistical significance was not reached for the rest of the potential risk factors, including age, hiatal hernia, GER symptoms, PPI usage, fundoplication, anastomotic reoperation and BMI.

4. Discussion

The main findings of the present study are outlined as follows. Among adults who have undergone repair of Gross C-type esophageal atresia, the prevalence of endoscopic and true Barrett's esophagus increased by three-fold and ten-fold during a median interval of 16 years between median age 36 and 50 years. Extensive lesions (≥10 cm) of true Barrett's Esophagus having the highest risk for malignant transformation [10,18] were not observed. None of the patients had esophageal mucosal dysplasia or carcinoma. The main predictors of true Barrett's Esophagus were pre-existing endoscopic Barrett's Esophagus and esophagitis. It is noteworthy that one third of the patients with normal index endoscopy developed endoscopic Barrett's esophagus. Thus, a normal endoscopy and lack of GER symptoms in an EA patient at the time of transition from pediatric to adult care do not eliminate the risk of Barrett's esophagus later in life.

Although the prevalence of Barrett's esophagus was relatively high in EA patients, it remains unknown whether they could benefit from routine endoscopic surveillance. An endoscopic surveillance program should not overburden patients with repeated sessions or unnecessarily worry them. Although we observed a steep rise in the prevalence of true Barrett's esophagus between the ages of 36 and 50 years, no extensive endoscopic Barrett's lesions, dysplasia or adenocarcinoma were encountered. Thus, routine endoscopic surveillance before the age 50 years seems unnecessary based on our findings.

The value of routine endoscopic follow-up in patients with EA remains controversial. A Dutch study performed repeated [1–3] endoscopies in 271 EA patients at median age <30 years and diagnosed true Barrett's esophagus in 7 % of patients while none had esophageal dysplasia or cancer. (21) The 7 % prevalence of true Barrett's esophagus around the median age of 30 is comparable to our previous study (6 %) with less strict criteria for true Barrett's esophagus [4,19].

Currently, it is unclear whether the risk of Barrett derived adenocarcinoma among EA patients differs from the rest of Barrett population. Moreover, our understanding of esophageal adenocarcinoma in EA patients is limited. A recent systematic review identified 13 esophageal cancers (9 squamous cell carcinomas and 4 adenocarcinomas) among 1727 EA patients who underwent endoscopic follow-up at median age 40 years. Of the four patients with adenocarcinoma, two had synchronous Barrett's esophagus, one had no evidence of Barrett's esophagus, and one had adenocarcinoma in an interposed colonic segment. In addition, one patient with squamous cell carcinoma had Barrett's esophagus [8].

Considering endoscopic surveillance of EA patients beyond 50 years of age, endoscopic surveillance of our patients with true Barrett's esophagus is currently performed according to ESGE recommendations (5 years interval for lesions ≤ 3 cm and 3 years for 3–10 cm lesions) -. For patients who are not diagnosed true Barrett's esophagus, ESGE does not recommend further endoscopic surveillance, and whether this practice is adequate for EA patients remains unknown. EA patients whose endoscopic status at the age of 50 years do not warrant further surveillance according to ESGE, may, however, remain at risk for development of true Barrett's esophagus.

In patients with GER, PPI medication has been shown to decrease the prevalence of esophagitis and consequently of Barrett's esophagus [20]. The data in the present study is insufficient to assess true effect of PPI usage in the evolution of Barrett's esophagus.

The main limitations of the present study include the retrospective conversion of the endoscopic findings into the Prague Classification at E1 based on endoscopy reports. Only presence or absence of GER symptoms was reported rather than qualitative data. The strengths of the study include a relatively long follow-up period, adherence to ESGE recommendations for the management of Barrett's Esophagus [10], and recruitment of a relatively high percentage of patients from the index endoscopy to the follow-up endoscopy.

Table 3
Risk factors for progression to endoscopic and true Barrett's esophagus between E1 (index endoscopy, 2005–08) and E2 (follow-up endoscopy 2019–2023) in 71 adult patients with type-C esophageal atresia.

	Endoscopic BE at E2		True BE at E2	
	RR (95 %CI)	p-value	RR (95 %CI)	p-value
Endoscopic BE (E1)	16 (1.9–134)	0.01	9.2 (2.0–41)	0.004
No BE (E1)	0.1 (0.0–0.5)	0.01	0.1 (0.0–0.5)	0.004
Male sex	3.9 (1.4–11)	0.01	3.6 (0.7–18)	0.12
Esophagitis (E2)	2.9 (0.8–9.7)	0.09	5.8 (1.4–24)	0.02
Esophagitis (E1)	4.3(1.2–15)	0.03	1.9 (0.4–8.7)	0.38
Smoking exposure[a]	1.0 (1.0–1.1)	0.41	1.1 (1.0–1.1)	0.06
Alcohol consumption (weekly or daily)	2.5 (0.9–6.8)	0.08	2.2 (0.6–8.6)	0.25
Prague Classification of endoscopic Barrett's esophagus (E1)				
• C, cm	1.2 (0.6–2.8)	0.59	0.8 (0.1–4.8)	0.78
• M, cm	1.4 (0.8–2.2)	0.23	0.4 (0.1–1.9)	0.22
Age (E2)	1.0 (1.0–1.1)	0.29	1.0 (0.9–1.1)	0.25
Hiatal hernia	1.8 (0.3–4.3)	0.89	1.8 (1.2–3.1)	0.33
Gastroesophageal reflux symptoms (E2)	1.9 (0.6–6.0)	0.25	2.1 (0.5–9.3)	0.31
Proton pump inhibitor usage (E2)	0.7 (0.3–2.0)	0.55	0.8 (0.2–3.4)	0.73
Fundoplication	1.3 (0.3–5.8)	0.70	2.2 (0.4–13)	0.37
Anastomotic reoperation	1.4 (0.4–5.1)	0.66	0.6 (0.1–5.7)	0.69
Body mass index (E2)	1.0 (0.9–1.1)	0.69	1.1 (0.0–1.2	0.48

Data are frequencies or medians with interquartile ranges. RR, risk Ratio; CI, confidence interval; BE, Barrett's esophagus; Endoscopic BE, endoscopically diagnosed BE with (true BE) or without goblet cell metaplasia; C, circumferential height of endoscopic BE; M, height of BE tongue.

[a] Smoking years x 365 x (cigarettes/day) x 10^{-3}.

5. Conclusion

Our endoscopic follow-up study detected a 10-fold increase in the prevalence of true Barrett's esophagus in adult EA patients between median age 36 and 50 years, respectively. No dysplasia or cancer was detected. Based on our findings, routine endoscopic surveillance before age 50 years seems unnecessary.

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Conflicts of interest

None of the authors.

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