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Esophageal Columnar Metaplasia in Childhood: A Population-Based Case Series Analysis

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Title: Esophageal Columnar Metaplasia in Childhood: a Population-Based Case Series

Analysis

Short title: Pediatric Barrett's esophagus

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Abstract

Background Adults with Barrett's esophagus (BE) are often entered into surveillance for esophageal adenocarcinoma (EAC), although cancer risk is relatively low. BE can be detected in children (<16 years). Little is known about the epidemiology of pediatric BE, and it is unclear what the optimal surveillance regimes are in ~~these~~ children.

Aim

To evaluate the demographic and clinical characteristics, and future neoplastic progression risk in all pediatric BE patients diagnosed in Northern Ireland between 1993 and 2010.

Method

Data from the population-based Northern Ireland BE register were matched to the Northern Ireland Cancer Registry for EAC outcomes until end 2013. Age-adjusted incidence of pediatric BE was calculated, and characteristics between pediatric and adult BE patients compared using chi-square tests.

Results

Over 18 years, 42 pediatric BE patients (<16 years) were identified, equivalent to an age-adjusted incidence of <2 per 100,000 children. There was a clear age differential, with BE incidence increasing with age ~~even~~ within the pediatric population. The majority (85.7%) of patients were male, a significantly higher male: female ratio than adult BE patients ($p < 0.001$). No pediatric BE patients progressed to high grade dysplasia (HGD) or EAC, although the eldest patient was aged 34 years by the end of follow-up.

Conclusions

This is the largest series of pediatric BE ever ~~to be~~ reported. It demonstrates that pediatric BE is rare. The male preponderance of this condition is ~~even~~ more apparent in childhood

compared with adult cases. No children developed HGD/EAC during follow-up, suggesting that regular surveillance is not required, at least until adulthood.

Keywords: Barrett's esophagus; epidemiology; pediatrics; childhood; incidence.

Introduction

Barrett's esophagus (BE) is defined as an esophagus in which any portion of the normal distal squamous epithelial lining has been replaced by metaplastic columnar epithelium, which is clearly visible endoscopically above the gastro-esophageal junction and confirmed histopathologically from esophageal biopsies ^[1]. In adults, it results from prolonged exposure of the esophagus to gastro-esophageal reflux. Known risk factors include increasing age, Caucasian race, male gender, smoking, diet and central adiposity ^[2,3,4,5,6,7].

The increasing incidence of BE in adults has been a growing public health concern due to the increasing incidence of esophageal adenocarcinoma (EAC) ^[3]. BE is a precursor to EAC which often presents late and patients have poor prognosis from diagnosis. In a random sample of 1000 individuals in the Swedish population who underwent endoscopy, BE prevalence was observed to be 1.6% in these adults ^[8]. In Northern Ireland, the incidence of BE in adults increased by 159% from 21.9/100,000 to 62/100,000 between 1993-1997 and 2002-2005 ^[3]. However, relatively little is known about BE epidemiology in children.

Gastroesophageal reflux is physiological in new born infants ^[8]. At the age of 18 months, a reported 7-20% of infants have persistent reflux and may then be diagnosed with gastro-esophageal reflux disease (GERD) ^[9]. We have previously reported that out of a series of 118 pediatric cases of hiatus hernia, 53% had a persistent hiatus hernia into adulthood and 46% had ongoing reflux symptoms ^[10]. Research into pediatric BE has been limited to date, challenged by difficulties with study design and particularly by small sample sizes. The largest study to date was a retrospective case review which included 38 cases ^[9]. Many previous studies have relied on retrospective designs that may be affected by recall bias. A systematic

review published by Jurnink *et al* in 2011 included a total of 14 studies, of which 4 were cohort studies and 10 were retrospective ^[9]. This systematic review found that the prevalence of BE in children with GERD varied from 0.3-4.8%, which is significantly lower than reported corresponding rates in the adult population, with 6-12% of adults with GERD reported to be diagnosed with BE ^[9,11]. Looking at trends within the pediatric BE population, Hassel *et al* found that similar to adulthood, childhood BE has a high male to female ratio, with one study reporting a 10:1 ratio ^[12]. In another paper from the same cohort, 100% of pediatric BE patients had symptoms and laboratory findings of GERD, most of which developed in the first year of life ^[13]. A recent systematic review by Raicevic and Saxena (2018) incorporating 130 patients (with a confirmed diagnosis of BE at < 18 years of age) from 18 studies carried out between 1985 and 2017 found that 82.2% of patients with a diagnosis of Barrett's esophagus during childhood, had previously confirmed GERD ^[14]. Other reported risk factors for BE in childhood included the presence of a hiatus hernia or a gastric tube, learning difficulties, chemotherapy treatment ^[12, 15,16]. It should be noted that these studies were published in the 1980s and 1990s, and further evaluation of pediatric BE epidemiology in more recent years is warranted.

The justification for such studies is also merited by a need to inform clinical guidelines. The current British Society of Gastroenterology guidelines indicate that adult BE patients with a length of columnar epithelium <3cm require surveillance every 3-5 years, and those with a length ≥3cm require surveillance every 2-3 years ^[1]. It remains unclear whether the application of these guidelines to children is appropriate.

The aims of this study are to analyse a large, population-based case series to evaluate the epidemiology of this rare condition in children, to compare and contrast clinical features between the pediatric and adult BE population, and to evaluate the risk of neoplastic progression for children with BE.

Materials and Methods

Barrett's esophagus patient identification

The patient data analyzed in this study were collected from the Northern Ireland Barrett's esophagus Register (NIBR); a robust database compiled of every patient diagnosed with BE in Northern Ireland (population 1.8 million) since 1993, as previously described [3,17,18,19]. The NIBR uses the British Society of Gastroenterology (BSG) definition of BE [1] and therefore to be included in the register a patient's pathology report must have had evidence for columnar lined epithelium (including but not limited to specialised intestinal metaplasia; SIM) in the esophagus. Information in the NIBR includes dates of biopsies, patient demographics, and information extracted via a manual review of pathology reports including BE length where reported in the clinical summary, and pathology information on the presence of SIM and/or dysplasia. Reports relating to biopsies from the esophago-gastric junction are excluded.

Follow-up information and outcomes

The proportion of pediatric BE patients (< 16 years) who had repeat esophageal biopsies after their initial BE diagnosis were evaluated for follow-up outcomes. Unfortunately, it was not possible to capture information on children who had a follow-up endoscopy that did not result in a biopsy, but it is assumed that any such investigations were deemed to reveal 'normal' findings. The number of repeated biopsies and their reported pathological findings subsequent to the index BE (normal, BE or progression to low or high grade dysplasia) were evaluated. Data were then matched using the unique Health and Social Care Number to the Northern Ireland Cancer Registry to assess cancer outcomes. Ethical approval for the NIBR research database (including the waiving of need for individual patient consent) and data analysis was granted by ORECNI (15/NI/0203) [20].

Statistical Analysis

All BE patients were organised into categories according to their age at incident BE diagnosis. The age categories used were <16, 16-<35, 35-<55, 55-<75 and ≥ 75 years. Within the under 16 year olds, distribution of age and year of diagnosis were investigated to further evaluate pediatric BE epidemiology. Combining census data from the Northern Ireland's Statistical and Research agency, age and year of BE diagnosis, it was possible to calculate the age-adjusted incidence per 100,000 for males and females for each year ^[19]. Years of diagnosis were then grouped together and an average calculated for each group. Using Stata (version 14, StataCorp, College Station, TX, USA), chi-squared tests were applied to compare the distribution of categorical variables such as sex, dysplasia, and presence of hiatus hernia between age categories. Numbers of cases less than 5 within categories been suppressed and reported as <5 to avoid disclosure of potentially identifiable patient data.

Results

Between 1993 and 2010, the total number of incident BE patients in the NIBR was 13,336. This included 42 pediatric cases and 13,294 adult cases, therefore pediatric BE cases represented only 0.31% of all BE cases diagnosed during this 18 year timeframe.

Temporal trends in pediatric Barrett's Esophagus incidence

The highest average number of diagnoses were made in 2008-2010 and the average lowest number of diagnoses were made from 2002-2004. However, over the entirety of the 18 year study period, there was no clear trend indicating changing crude incidence of pediatric BE. The age-adjusted incidence was less than 2/100,000 children.

Age and sex distribution of incident pediatric Barrett's Esophagus

Figure 1 shows the male and female breakdown of pediatric BE incidence, demonstrating a strong male predominance with the age adjusted incidence/100,000 reaching a maximum of 1.54 in males whereas the maximum in females was 0.9. The average age of diagnoses in the 42 pediatric BE cases was 10.1 (SD 4.04) years. The trends in the age of diagnosis across the pediatric patients are outlined in Figure 2. There is a clear increase in incidence with increasing age with <5 children being diagnosed at <2 years old and 11 children being diagnosed at 14-<16 years old.

Comparison of pediatric and adult Barrett's Esophagus features

Table 1 compares features such as sex, presence of a hiatus hernia, or dysplasia in the pediatric BE population with adult counterparts. Significant differences were observed in the male to female ratio across age groups, and this became more apparent as age increased. In

pediatric BE, 85.7 %of cases were male, compared with 69.7% of 16-35 year olds ($p=0.02$) and decreasing to 45.6% of over 75 year olds ($p<0.001$).

The proportion of patients with dysplasia increased significantly as age increased, with no dysplasia reported in under 16 year olds with BE, rising to up to 6.2% of ≥ 75 year olds having dysplastic BE at their incident diagnosis ($p<0.001$). There was no clear difference in the proportion of patients with hiatus hernias across age categories (Table 1).

Follow-up and risk of neoplastic progression in pediatric Barrett's Esophagus

Follow up data were available up until 2010 for the BE register and 2013 for the cancer registry. Accordingly, the average age of patients from the pediatric BE cohort is 18 years in the NIBR and 21 years in the cancer registry, with the youngest children being 4 and 7 years and oldest aged 31 and 34 years old, respectively, by the end of follow-up.

Out of the 42 children with a diagnosis of BE, 29 cases did not have any subsequent esophageal biopsies. Thirteen children had subsequent endoscopy and biopsies, Excluding the four children who had a repeat biopsy within 6 months, the mean follow-up time was 3 (SD 2.4) years (reflecting typical time to surveillance endoscopy) which ranged from 0.9-9 years.

Of the 9 patients that underwent follow-up biopsy > 6months following their diagnosis, columnar mucosa was observed in all 9 patients and six had SIM detected and three did not. Less than 5 patients developed low grade dysplasia in early adulthood (age range 22-24 years), but none progressed to high grade dysplasia or EAC.

Discussion

The findings from this large, population-based study confirm that pediatric BE is a rare disease. In Northern Ireland between 1993 and 2010, there were 42 diagnosed cases of pediatric BE, accounting for only 0.31% of all BE diagnoses over this 18 year timeframe. Even amongst this young age group, there was a predominance of male BE cases. The male to female ratio of BE reduces slightly in older categories. No pediatric BE cases had dysplasia. Follow-up data revealed that of the 42 pediatric BE patients, <5 continued to have BE into adulthood; none had progressed to high grade dysplasia or EAC, although one had a low grade dysplastic BE.

The data in this study focuses on pediatric cases diagnosed in the 1990s and 2000s, with follow-up to 2013 therefore limiting the conclusions that can be drawn for pediatric BE into later adulthood. The eldest patient was 34 years by the end of the study follow-up period. Continued monitoring of the incidence of dysplasia and EAC in the 42 pediatric cases into adulthood will provide useful data regarding the future surveillance of these patients.

This study demonstrated the existence of a steady increase in the incidence of pediatric BE with increasing age throughout childhood. This age distribution fits with the pattern seen in adult BE of increasing incidence with age, albeit at a much lower incidence ^[3].

This study found 85% of childhood BE patients were male, which is consistent with a previous literature review by Hassel et al which found that 91% of cases were male and Jeurnink *et al's* systematic review in which 66% of patients were male ^[9,11]. The high male to female ratio in the pediatric population could dispel the hypothesis that estrogen could be protective for BE or EAC, as this ratio still exists before puberty ^[22].

The mean age of patients in this study was similar to that of Jeurnink *et al's* (2011) review which found the mean age to be 9.5 years ^[12]. The follow up data of the current study was also in keeping with Beddow *et al's* retrospective case review study. Reviewing surveillance in 38 cases, they found that 39% of diagnosed patients continued to have BE but none were diagnosed with dysplasia or EAC with a mean follow up time of 3 years and 7 months ^[23].

None of the patients within the current study had SIM observed at initial BE diagnosis, with only one in 7 (14.2%) going on to develop SIM in adulthood. A study carried out by El-Serag *et al.* (2002) in 402 patients with GERD aged between 18 months and 25 years (mean age 9.7 years) who underwent endoscopy showed similar findings with none of the 11 patients diagnosed of BE having SIM present ^[26]. As factors associated with an increased risk of BE in adulthood, such as smoking status ^[27], are not applicable in childhood, it is important that other risk factors for BE are considered within the pediatric population. In a systematic review by Jeurnick *et al*, 45% of 176 pediatric BE patients had predisposing risk factors including congenital abnormalities, neurological impairment, chronic lung disease, hiatus hernia and a family history of GERD, BE or EAC ^[9]. Hassel *et al*, studied 11 patients in a retrospective study and found that 10 patients had symptoms of recurrent vomiting and pulmonary infections in their first year of life ^[13].

Current adult guidelines suggest repeating endoscopy and biopsy every 3-5 years for a length of columnar epithelium <3cm and every 2-3 years with a length >3cm ^[1]. On the basis of our data it may be possible to delay any surveillance endoscopy of pediatric cases of BE until adulthood as no patients diagnosed in childhood developed dysplasia or EAC under the age of 16. This finding is supported by Beddow *et al* who found that none of their 38 pediatric BE

patients had progressed to dysplasia or malignancy and concluded that surveillance for pediatric cases could be infrequent ^[23]. Singendonk et al also had similar findings with most studies reporting no progression to BE in childhood in patients with a GERD diagnosis ^[11]. The recent systematic review carried out by Raicevic and Sexena (2018) found that only one patient progressed to adenocarcinoma at the age of 23 years ^[14].

Our study has many strengths, due to its study size relative to existing very limited literature, and its population-based approach. The previous largest study in the UK included 38 cases, making our current cohort the largest in which pediatric BE epidemiology has been studied ^[10]. One limitation of this study was that it was not possible to access patients' full clinical reports. Information including birth weight, weight at diagnosis, onset and duration of symptoms, past medical history and pre-existing co-morbidities such as mental impairment, could have helped to increase our understanding for the mechanisms behind BE in the pediatric population. Limited evidence from Scandinavia has been published on this topic. Forssell *et al* investigated the relationship between preterm birth or low birth weight and the risk of developing BE ^[24]. That study found that a low birth weight significantly increased the patient's likelihood of developing BE compared to infants with a normal birth weight for gestational age (OR 8.22 95% CI 2.83-23.88) ^[21]. In another study, Forssell *et al* found that babies born before full term were at an increased risk of EAC ^[25].

Another limitation of this study is that follow-up biopsies at >6months after the index biopsy (where BE was confirmed) were only available for 9 patients (21.4%) therefore it is possible that a portion of the remaining 33 patients could have gone on to develop SIM in the absence of follow-up endoscopy and biopsy.

Future research should focus on risk factors and mechanisms specific to pediatric cases of BE. Furthermore, following each of the 42 patients with pediatric BE and any newly diagnosed patients into adulthood for outcomes will help to confirm if pediatric BE increases the overall lifetime risk of dysplasia and EAC and if surveillance is warranted.

In conclusion, pediatric BE is a rare condition and in this large, population based study it was found to have an incidence of <2 per 100,000. This study has further emphasised the high male to female ratio in BE. Our limited evidence on follow-up shows that no patients diagnosed in childhood developed dysplasia or EAC under the age of 16 and therefore suggests that it may be possible to delay any surveillance endoscopy of pediatric cases of BE until at least early adulthood. Further investigation into the optimal timing of endoscopic surveillance in pediatric BE is required.

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References

- [1] Fitzgerald RC, di Pietro M, Ragunath Ket al. British Society of Gastroenterology guidelines on the diagnosis and management of Barrett's oesophagus. *Gut* 2014 Jan;63(1):7-42.
- [2] Anderson LA, Monaghan P, Murray L et al. Risk factors for Barrett's oesophagus and oesophageal adenocarcinoma: results from the FINBAR study. *World J Gastroenterol* 2007;13(1):146-151.
- [3] Coleman HG, Bhat S, Murray LJ, McManus D, Gavin AT, Johnston BT. Increasing incidence of Barrett's oesophagus: a population-based study. *Eur J Epidemiol* 2011;26(9):739-745.
- [4] Ford AC, Forman D, Reynolds PD, Cooper BT, Moayyedi P. Ethnicity, gender, and socioeconomic status as risk factors for esophagitis and Barrett's esophagus. *Am J Epidemiol* 2005 Sep 1;162(5):454-460.
- [5] Andrici J, Cox MR, Eslick GD. Cigarette smoking and the risk of Barrett's esophagus: A systematic review and meta-analysis. *J Gastroenterol Hepatol* 2013;28(8):1258-1273.
- [6] Winberg H, Lindblad M, Lagergren J, Dahlstrand H. Risk factors and chemoprevention in Barrett's esophagus—an update. *Scand J Gastroenterol* 2012;47(4):397-406.
- [7] Hassall E. Columnar-lined esophagus in children. *Gastroenterol Clin North Am* 1997;26(3):533-548.
- [8] Ronkainen, J, Pertti, A, Storskrubb, T et al (2005) Prevalence of Barrett's Esophagus in the General Population: An endoscopic study. *Gastroenterology*: 129:1825-1831.

- [9] Jeurnink SM, van Herwaarden-Lindeboom MY, Siersema PD, Fischer K, Houwen RH, van der Zee, David C. Barrett's esophagus in children: does it need more attention? *Digestive and Liver Disease* 2011;43(9):682-687.
- [10] Johnston, BT, Carré, IJ, Thomas, PS, Collins, BJ. Twenty to forty year follow-up of infantile hiatal hernia. *Gut*. 1995; 36:809-812.
- [11] Singendonk MM, Tabbers MM, Benninga MA, Langendam MW. Pediatric Gastroesophageal Reflux Disease: Systematic Review on Prognosis and Prognostic Factors.. *J Pediatr Gastroenterol Nutr*. 2018 Feb;66(2):239-243
- [12] Hassall E. Barrett's esophagus: new definitions and approaches in children. *J Pediatr Gastroenterol Nutr* 1993;16(4):345-364.
- [13] Hassall E, Weinstein WM, Ament ME. Barrett's esophagus in childhood. *Gastroenterology* 1985;89(6):1331-1337.
- [14] Raicevic, M and Sazena, A. Barrett's esophagus in children: what is the evidence? *World Journal of Pediatrics*. 2018; 14; 330-334.
- [15] Dahms, B, Greco, M, Stranfjord, S, Rothstein, F. Barrett's Esophagus in three children after antileukaemia chemotherapy. *Cancer*. 1987; 60:2896-900
- [16] Lindahl, H, Rintala, R, Sariola, H, Louhimo, I. Cervical Barrett's Esophagus: a common complication of gastric tube reconstruction. *J Pediatr Surg*. 1990;25:446-8.

[17] Bhat S, Coleman HG, Yousef F et al. Risk of malignant progression in Barrett's esophagus patients: results from a large population-based study. *J Natl Cancer Inst* 2011;103(13):1049-1057.

[18] Bhat SK, McManus DT, Coleman HG et al. Oesophageal adenocarcinoma and prior diagnosis of Barrett's oesophagus: a population-based study. *Gut* 2015 Jan;64(1):20-25.

[19] van Putten M, Johnston BT, Murray LJ et al. 'Missed'oesophageal adenocarcinoma and high-grade dysplasia in Barrett's oesophagus patients: A large population-based study. *United European gastroenterology journal* 2018;6(4):519-528.

[20] Kearney T, Donnelly C, Kelly J, O'Callaghan E, Fox C, Gavin A. Validation of the completeness and accuracy of the Northern Ireland Cancer Registry. *Cancer epidemiology* 2015;39(3):401-404.

[21] Northern Ireland Statistics and Research Agency (NISRA). Northern Ireland Population Projections 2001 accessed at <https://www.nisra.gov.uk/statistics/population> on 5th November 2019.

[22] Asanuma K, Iijima K, Shimosegawa T. Gender difference in gastro-esophageal reflux diseases. *World j gastroenterol* 2016;22(5):1800.

[23] Beddow E, Wilcox D, Drake D, Pierro A, Kiely E, Spitz L. Surveillance of Barrett's esophagus in children. *J Pediatr Surg* 1999;34(1):88-91.

[24] Forssell L, Cnattingius S, Bottai M, Lagergren J, Ekblom A, Akre O. Risk of esophagitis among individuals born preterm or small for gestational age. *Clinical GastroenterolHepatol* 2012;10(16):1369-1375.

[25] Forssell L, Cnattingius S, Bottai M et al. Risk of oesophageal adenocarcinoma among individuals born preterm or small for gestational age. *Eur J Cancer* 2013;49(7):2207-2213.

[26] El-Serag, HB, Bailey, NR, Gilger, M, Rabeneck, L. Endoscopic Manifestations of Gastroesophageal Reflux Disease in Patients Between 18 months and 25 years with neurological deficits. *Am J Gastroenterol* 2002; 97(7);1635-1639.

[27] Andrici, J, Cox, MR, Eslick, GD. Cigarette smoking and risk of Barrett's esophagus: A systematic review and meta-analysis. *J Gastroenterol Hepatol* 2013; 28 (8);1258-1273.

Table 1. Comparison of demographic and clinical characteristics between pediatric and adult Barrett's esophagus patients.

	Age groupings (years)								
	<16 n=42 (%)	16-<35 n=749 (%)	P-value	35-<55 n=3,934 (%)	P-value	55-<75 n=5,932 (%)	P-value	≥75 n=2,679 (%)	P-value
Sex									
Female	6 (14.3)	227 (30.3)	0.03	1,298 (33.0)	0.01	2,600 (43.8)	<0.001	1,458 (54.4)	<0.001
Male	36 (85.7)	522 (69.7)		2,636 (67.0)		3,332 (56.2)		1,221 (45.6)	
Hiatus Hernia present									
No/Unknown	N/A*	673 (89.9)	0.10	3,503 (89.0)	0.08	5,318 (89.7)	0.09	2,438 (91.0)	0.14
Yes	N/A*	76 (10.1)		431 (11.0)		614 (10.3)		241 (9.0)	
Dysplasia									
No Dysplasia recorded	42 (100.0)	742 (99.1)	<0.001	3,814 (96.9)	<0.001	5,606 (94.5)	<0.001	2,513 (93.8)	<0.001
Dysplasia	0 (0)	7 (0.9)		120 (3.0)		326 (5.5)		166 (6.2)	

* Due to small number (<5) of patients aged less than 16 years with hiatus hernia present, numbers been suppressed to avoid disclosure of potentially identifiable patient data

Figure 1. Age-adjusted incidence of pediatric Barrett's esophagus per 100,000 population in Northern Ireland, 1993-2010.

Figure 2. The distribution of incident pediatric Barrett's esophagus patients in Northern Ireland by age and sex groupings.