including gastroenterologist, allergist, dietitian, psychologist and clinical nurse specialist. Of those, 32 children with eosinophilic oesophagitis (EoE) alone were excluded. Hence, 76 patients [61%, (n=46) non IgE mediated allergy, 4% (n=3) IgE mediated allergy, 27% (n=20) combined IgE, non-IgE and EoE, 10% (n=7) had other food triggered conditions) were retrospectively reviewed.

Results 97% (n=74) of children were following exclusion diets for ≥ 12 months, with a mean of 5 excluded foods (median 4, IQR 2, 6). At the follow-up, the mean number of foods excluded had reduced to 3 (median 2.5, IQR 1, 5); $p < 0.0001$. Milk (n = 59; 78%) was the most common excluded food, whilst fish/shellfish (n=18; 24%) was the least. Over 12 months 55% (n=42) of patients introduced at least 1 food into their diet and 16% (n=12) of patients reintroduced between 75–100% of excluded foods. The dietitians provided on average 3 contacts to patient, in the form of face-to-face appointments or telephone appointments (range 1–16).

Conclusion In children with GIFA, long-term unnecessary food exclusions should be avoided, due to the relationship with poor growth, feeding difficulties and nutritional deficiencies (Meyer, 2018) Despite the fact that the majority of them had been following long-term exclusion diets, over half of patients were able to reintroduce at least one food into their diet.

In this cohort, the MDT approach, which brings together professionals from different backgrounds to pave the most effective management plan for the patient, has shown to be highly beneficial in supporting patients and their families to reintroduce foods into their diets. However this requires ongoing support for many families' in-between medical appointments to achieve this goal.

This small centre outcome demonstrates some positive impacts of MDT approach, which should become the standard model of care in children with complex GIFA.

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P25

GUIDELINE VS CLINICAL PRACTICE, LOOKING BEYOND ESPGHAN POLYPOSIS WORKING GROUP RECOMMENDATIONS FOR FAP SCREENING

Suchandra Pande, Rajiv Mohan. *Leicester Royal Infirmary*

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Introduction/Background Current guideline for the initial endoscopic assessment for suspected polypoid disease of the colon have been created by professional bodies such as ESPGHAN.

In instances where there is a family history of FAP and genetics demonstrates a relevant mutation, in clinical practice, we refer to the above guideline to determine the age for initial endoscopic assessment and subsequent surveillance is further determined by the findings of the initial endoscopy.

This guideline recommends only a lower GI endoscopy for the initial test and do not recommend a concomitant UGI scope till age 25 years (Recommendation 5- ESPGHAN FAP Screening guideline).

We report two cases where an initial combined upper and lower GI endoscopies showed >100 gastric, 10 to 50 colonic

polyps and 50 to 100 Gastric polyps, >100 colonic polyps respectively and discuss the relevance of these guidelines for such instances.

Aim; To review the relevance of current guidelines with regard to the extent of initial endoscopy that is recommended in suspected cases of FAP in children.

Subjects and Methods Patient 1: 13 years old boy referred by geneticist as noted to have significant alteration in APC gene raising possibility of APC syndrome. At presentation, history of upper abdominal pain, dyspepsia. No bleeding PR, pallor. Mum had colectomy when 22 years old. Upper GI endoscopy done along with colonoscopy in view of upper abdominal pain and dyspepsia. Upper GI endoscopy revealed numerous (>100) small (<5 mm diameter) gastric polyps in body and antrum. 2 slightly larger polyps were noted at lower oesophagus near gastro oesophageal junction. Histology reported as gastric fundic gland polyp. H pylori not detected. Colonoscopy revealed 10 to 50 colonic polyps (some 2 mm, rest <2 mm diameter). Histology reported as adenomas with low grade dysplasia.

Patient 2: 12 years old boy referred by geneticist, father known to have FAP. Ophthalmology screening showed changes on the retina. Genetic test showed mutation in APC gene. First colonoscopy revealed scattered polyps throughout colon (<50). Histology reported as tubular adenoma of low dysplasia. No upper GI endoscopy done. Noted to have vague upper abdominal pain at the time of second colonoscopy 18 months later. Otherwise asymptomatic. Upper GI endoscopy done along with Colonoscopy in view of upper abdominal pain. Upper GI endoscopy revealed multiple (50 to 100) small gastric polyps (<5 mm diameter) in gastric body. Histology reported as fundic gland polyps. H pylori negative. Colonoscopy revealed >100 Colonic (2 mm) polyps. Histology reported as adenoma with low grade dysplasia.

Results Deviation from current guidelines has yielded a positive finding of multiple gastric polyps.

Summary and Conclusion Current guideline do not provide advice for further management and surveillance when gastric polyps are incidentally discovered on concomitant UGI endoscopy undertaken for other clinical reasons in patients with FAP.

P26

HELICOBACTER PYLORI CULTURE IN ROUTINE PRACTICE: A PAEDIATRIC RETROSPECTIVE STUDY

¹Rime Hicham, ²Mary Ann Ritchie, ³William Ennis, ³Margaret Murray, ⁴Pamela Saunders, ⁵Kamaljit Khalsa, ³Ashutosh Deshpande, ¹Andrew R Barclay, ¹Diana M Flynn, ¹Rachel Tayler, ¹Richard Hansen. ¹Department of Paediatric Gastroenterology, Royal Hospital for Children, Glasgow, UK; ²Department of Microbiology, Glasgow Royal Infirmary, Glasgow, UK; ³Department of Microbiology, Southern General Hospital, Glasgow, UK; ⁴Scottish Microbiology Reference Laboratory, Glasgow Royal Infirmary, Glasgow, UK; ⁵Department of Microbiology, Queen Elizabeth University Hospital, Glasgow, UK

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Introduction Helicobacter pylori is usually acquired in early childhood. The systematic use of standard eradication therapy regimes has resulted in a rising prevalence of antibiotic-resistant strains and a decreasing efficiency of H. pylori eradication therapy. The latest ESPGHAN guidelines (2016) suggest only investigating H. pylori in paediatric patients who would benefit from treatments and to base the eradication therapy on susceptibility testing.

Our local practice has been to test symptomatic children with H. pylori stool antigen and to treat with a standard

triple therapy as a first line. If a second attempt at eradication fails, children have oesophagogastroduodenoscopy (OGD) on a dedicated gastroenterology/microbiology *H. pylori* culture list. Subsequent antibiotic therapy is then based on antibiotic sensitivity.

Aims Our primary objective was to study the benefit of *H. pylori* eradication therapy based on culture and sensitivity in our population. Our secondary objective was to describe the sensitivities of the enumerated *H. pylori*.

Subjects and Methods We retrospectively included all paediatric patients who had undergone OGD for *H. pylori* culture in Royal Hospital for Children, Glasgow, between 2014 and 2020. We collected data from patient electronic records. *H. pylori* colonisation was based on the presence/absence of the organism on histopathology. Eradication was assessed by either *H. pylori* stool antigen or subsequent gastric biopsy histopathology.

Results In total, 20 patients were included with a median age of 10.1 [7.6–12.4] years. In keeping with our local practice they had a median of 2 attempts at eradication therapy before being referred for *H. pylori* culture. On these 20 patients, 15 patients (75%) had a confirmed colonisation by *H. pylori* on histopathology. 14 of these patients (93%) had successful culture of *H. pylori*, 1 (7%) had a failed culture.

On these 14 cultures, 1(7%) had initial growth but failed sensitivities. 3 *H. pylori* cultures (21% of positive culture) were fully sensitive to amoxicillin, metronidazole and clarithromycin. 4 (28%) were resistant to a single agent (50% to metronidazole and 50% to clarithromycin). 5 (35%) were resistant to both clarithromycin and another agent (80% to metronidazole and 20% to amoxicillin) and 1 (7%) was fully resistant to these 3 antibiotics.

After sensitivity-based eradication therapy, 7 patients (50% of positive cultures) had ongoing *H. pylori* colonisation (3 confirmed on repeated OGD, 4 confirmed on a positive stool antigen), 4 (28%) had confirmed eradication (3 confirmed on repeat OGD and 1 on negative stool antigen), and 2 results are still awaited. Out of the 7 patients who failed eradication, 6 (85%) patients had resistant organisms and the remaining patient had poor treatment compliance.

Conclusion Our dedicated *H. pylori* list has excellent culture recovery (93%), but its set-up can be challenging.

Our approach to restricting culture to two therapy failures may explain our high rates of targeted antibiotic failure (50%), but these patients may also be biased towards therapeutic non-compliance.

Having a better knowledge of the positivity rate of stool antigens in our paediatric population and eradication rate after first-line therapy would be helpful to consciously consider our results.

P27

HYPOPHOSPHATEMIA AFTER TREATMENT OF IRON DEFICIENCY WITH FERRIC CARBOXYLMALTOSE (FCM) INFUSION IN PAEDIATRIC INFLAMMATORY BOWEL DISEASE IN A TERTIARY PAEDIATRIC GASTROENTEROLOGY CENTRE

Rajkumar Dhandayuthapani, Helen Doble, Fiona Cameron, Manjula Velayudhan Nair, Marcus Auth, Sarang Arun Tamhne, Stephen Allen, Jeng Cheng, Elizabeth Renji. *Alder Hey Children's Hospital*

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Background Anaemia is the most common extra intestinal manifestation in paediatric Inflammatory Bowel Disease (pIBD)

needing monitoring and supplementation. Intravenous iron is often needed in moderate to severe disease or intolerance to oral iron. Ferric Carboxymaltose (FCM) infusion has been used in our centre. While the risk of hypophosphatemia (HP) associated with FCM is known, (Medicines and Healthcare Products Regulation Agency alert), it is not been quantified in clinical practice.

Aims and Objectives Primary objectives' to report incidence and severity of HP after infusion of FCM in pIBD. Assess time to recovery from HP with interventions. Secondary objectives' to review patient characteristics and biochemical markers to identify risk factors for FCM infusions associated HP.

Design study We conducted a retrospective electronic records review of pIBD patients who received FCM infusion from November 19-November 20.

Results 24 patients (13 M) received 26 infusions in the period reviewed. The median age was 14.5(12.6–15.9). From all the children, 7(29.2%) were diagnosed with Ulcerative Colitis (UC), 16(66.7%) with Crohn's Disease and 1(4.2%) with IBD-Unclassified (IBDU). The timing of infusion coincided with: admitted with new diagnosis of pIBD, [10(41.7%)], flare of disease [9(37.5%)] and elective admission [5(20.8%)] for correction.

Median dose for the 26 infusions was 1000(500–1000)mg. Pre-infusion median (IQR) haemoglobin 107.5 (92.7–119.2), MCV 81(74.7–83.2), iron 3.4(2.1–5.5), ferritin 73.1(33.2–115.7) and transferrin saturation 4.85(3.6–10.0)%. All had normal renal function; median(IQR) urea 2.9(2.32–3.35) and creatinine, 50.5(37–61.7). Pre-transfusion Vitamin D (n=19) median was 38.5(27.5–53.5), with levels being deficient in 13 (68.3%) with no association to recovery time.

The biochemical markers are shown in table 1 below, with the change shown as delta change. The delta change was found to be statistically significant for serum phosphate levels. There were no statistically significant association, on univariate analysis, between the delta change in serum phosphate levels and the features of patient demographics or biochemical markers.

All 24 patients had reduction in phosphate level post FCM infusion. In 14/26(53%), phosphate levels dropped to moderate-severe range; 10/14(71.4%) HP was moderate (< 0.65 mmol/L) and in 4/14(28.5%) HP was severe (< 0.32 mmol/L). The median time of recovery from hypophosphatemia was 14(4–26) days. Those that took longer than 14 days to normalise phosphate (9,37.5%) had no features attributable to it.

No serious sequelae of hypophosphatemia were seen. Treatment included a combination of IV phosphate infusion (6,25%), oral phosphate supplements (12,50%), oral calcium supplements (15,62.5%) and vitamin D supplements (17,70.8%).

Conclusion HP is frequently seen with FCM infusion. The fall in phosphate post FCM infusion was found to be clinically

Abstract P27 Table 1

Biochemical markers	Baseline	Post-infusion	Delta change	p
Serum phosphate	1.19 ± 0.26	0.71 ± 0.40	-0.48 ± 0.35	p<0.0005*
Ionized calcium	2.35 ± 0.09	2.37 ± 0.11	0.02 ± 0.09	p<0.284
Serum potassium	4.03 ± 0.52	4.21 ± 0.37	0.17 ± 0.62	P<0.177
Serum magnesium	0.82 ± 0.08	0.77 ± 0.18	-0.01 ± 0.07	P<0.221