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Editorial

Benign Colonic Protrusions-Hyperplastic Polyp

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Colon polyps are defined as protuberances occurring within the colonic lumen. Localized epithelial proliferation typically enunciating a superficial serrated architecture, variably elongated crypts with epithelial proliferation confined to basal segment of crypts accompanied by a lack of epithelial dysplasia is cogitated as a polyp. Colonic polyps frequently appear as sporadic neoplasm although can arise as a component of specific syndromes.

Cogent classification

Polyps are variously categorized pertaining to the dimension:

- As diminutive if the diameter is beneath < 5 millimetres.
- Miniature or small for polyps demonstrating a dimension betwixt 6 millimetre to 9 millimetre.
- Large polyps in instances delineating a magnitude exceeding > one centimetre [1,2].

Polyps can be exemplified as excavated, flattened, sessile or pedunculated. A certain proportion of polyps arise within the submucosa and can be constituted of lipomas, carcinoid tumours and lymphoid aggregates. Frequently, polyps are engendered from the mucosa, are denominated as adenomatous and display distinctive architecture as with tubular polyps exceeding 80%, villous polyps in 5% to15% and tubulo-villous polyps in 5% to 15% instances, serrated polyps appearing as sessile or traditional forms besides non neoplastic polyps configuring hyperplastic and juvenile articulations [1,2].

Juvenile polyps are benign hamartomas, devoid of premalignant manifestations, frequent in the recto-sigmoid junction and commonly emerge within the childhood.

Sessile serrated adenomas/polyps (SSA/Ps) are frequently observed within the proximal colon and delineate minimal malignant conversion in the absence of dysplasia and proportionate malignant transformation in dysplastic polyps. The uncommon, typical serrated adenoma is generally observed in distal colon and manifests a significant potential of malignant metamorphoses.

Non-serrated polyps are comprised of hyperplastic polyps, juvenile polyps, intestinal hamartomas and inflammatory pseudopolyps [1,2].

Disease characteristics

Hyperplastic polyps as a frequent category are predominantly encountered within distal colon and demonstrate a minimal possibility of malignant transformation.

Factors associated with enhanced emergence of hyperplastic polyps recapitulate pertinent factors encountered with adenomatous polyps such as smoking, alcohol abuse, folate deficiency, obesity, individuals of advancing age, male subjects, intake of lipid rich diet with minimal fibre, consumption of tobacco and excessive quantities of alcohol. A cogent family history, emergence of colorectal cancer and intestinal polyposis syndromes are accompanied by an elevated probability of occurrence of colonic polyps. In contrast, inflammatory bowel disease is associated with decline in polyp prevalence [3,4].

Hyperplastic polyps are generally multiple, a frequent category of colonic polyps demonstrated within 75% to 90% subjects, typically delineate 1 millimetre to 5 millimetres magnitude and exceptionally a dimension of one centimetre or beyond.

Endoscopic evaluation of subjects beyond 50 years can incidentally exemplify hyperplastic polyps and the proportion rises with enhancing age although younger population can be incriminated. Majority of hyperplastic polyps incidentally discovered during en-

doscopy configure as a pale-staining, nodular bumps. However, endoscopic appearance is not confirmatory and lesions can resemble adenomatous polyps. Thus a tissue evaluation and polyp fulguration is mandated [3,4].

Incidence of hyperplastic colorectal polyps enhances with advancing age, depicts a prevalence of around 35% in asymptomatic subjects exceeding 50 years and is enunciated predominantly in the Caucasian population. Colorectal polyps are exemplified in an estimated 6% children whereas around 12% younger age groups exhibiting lower gastrointestinal haemorrhage can be incriminated. Non-white male subjects can predominantly exhibit colorectal polyps [4,5].

Occurrence of colonic polyps can be reduced by adhering to a lipid-restricted diet with enhanced fibre intake, decline in alcohol consumption with cessation in smoking.

Non-resected polyps can engender haemorrhage, intussusception or a malignant transformation. However, colonic polyps usually progress gradually and configuration of a miniature polyp can extend to up to ten years.

Probability of malignant conversion of polyps is amplified in hereditary non polyposis syndrome. Proportionate malignant transformation of a sporadic polyp of one centimetre magnitude is around 8% at 10 years and roughly 24% at 20years. Malignant conversion is also contingent to the variety of polyp, morphological description and associations with diverse polyposis syndrome [5,6].

Nevertheless, consumption of multivitamins, folate, calcium, statins, azathioprine or 6-mercaptopurine may not display a decimation in emergence of colorectal carcinoma. Proportionate elevation of carcinoma ovary and uterus is also enunciated. Hyperplastic polyps can depict complications such as bleeding, diarrhoea, bowel obstruction and exceptionally, a malignant conversion [4,6].

Disease pathogenesis

Majority (> 95%) of colonic adenocarcinomas originates from pre-existing colorectal polyps. Genomic aberrations are preponderant in emerging malignancies. Polyps can progress into frank carcinoma following inactivation of tumour suppressor gene as enunciated with adenomatous polyposis coli (APC) or mismatch repair gene (MLH1). Familial adenomatous polyposis (FAP) is autosomal dominant condition and majority of subjects demonstrate colorectal carcinoma within 40 years [4,6].

Juvenile polyposis syndrome (JPS) is an autosomal dominant condition wherein 50% to 60% subjects delineate a chromosomal mutation within SMAD4 or BMPR1A gene. An estimated 20% individuals evolve into a colorectal carcinoma by 35 years.

Adjunctive variants of genomic inheritance arises in the form of MUTYH- associated polyposis (MAP) engendered as an autosomal recessive condition arising on account of bi-allelic mutation of MUTYH gene. Administration of non-steroidal anti-inflammatory drugs (NSAIDs) and calcium in familial adenomatous polyposis (FAP) and MUTYH- associated polyposis (MAP) can decrease incidence and reoccurrence of polyps [5,6].

BRAF or KRAS genetic mutation can ensue along with DNA hyper-methylation with hyperplastic polyps [6].

Clinical elucidation

Hyperplastic polyps are essentially asymptomatic and devoid of malignant potential. Asymptomatic colorectal polyps are usually discerned upon screening colonoscopy for detection of colorectal carcinoma. Painless rectal haemorrhage with fresh or altered blood, diarrhoea, constipation, blood or mucus mixed stools, abdominal pain and signs and symptoms of iron deficiency anaemia secondary to chronic bleeding can be observed. Majority (90%) of hyperplastic polyps occur in the left colon, particularly within the rectum. Emerging as singular or multiple lesions, hyperplastic polyps confined to the proximal colon are enlarged, in contrast to distally located polyps. Also, micro-vesicular hyperplastic polyps are minimally exemplified within distal colon [5,6].

Infrequently, hyperplastic polyps can occur as a component of MUTYH- associated polyposis, Peutz-Jeghers syndrome, juvenile polyposis syndrome, hyperplastic polyposis syndrome, PTEN hamartoma syndrome, Birt-Hogg-Dube syndrome or hereditary haemorrhagic telangiectasia syndrome [5,6].

Histological elucidation

On gross examination hyperplastic polyps are generally beneath < 5 millimetres magnitude, situated atop a mucosal fold. Multiple hyperplastic polyps can be enunciated which delineate a colour identical to the encompassing colonic mucosa.

Hyperplastic polyps are categorized into micro-vesicular, goblet cell and mucin poor subtypes contingent to mucin quantification and occurrence or absence of dystrophic goblet cells, a classification which lacks clinical concurrence or significance [6,7].

Epithelial hyper-proliferation within basal portion of crypts engenders hyperplastic polyps. Colonocytes are usually generated rapidly in contrast to decimation of colonocytes with a consequently serrated appearance.

Morphological expression of a micro-vesicular hyperplastic polyp demonstrates an admixture of goblet cells and columnar cells along with the occurrence of micro-vesicular mucin and inconspicuous nuclei with prominent luminal serrations. Surface epithelium of the polyp is configured of mature, columnar cells and mitosis is confined to basal glandular region [6,7].

Goblet cell rich subtype is preponderantly composed of goblet cells with mild, superficial serrations.

Mucin poor subcategory principally demonstrates a micro-papillary architecture, mucin depletion and absence of goblet cells and is often associated with a regenerative appearance.

Villous, tubular and tubulo-villous polyps are diverse morphological categories of colorectal polyps. Adenomatous polyps can eventually depict dysplastic modifications, a feature of segregation from hyperplastic polyps. Generally, colonic polyps are benign. However, polyps demonstrating high grade dysplasia eventually emerge as malignant. Immune histochemical reactivity to Ki- 67 depicts an epithelial proliferation predominantly confined to basal portion of hyperplastic polyp [6,7].

Differential diagnosis

Hyperplastic polyps require a demarcation from pseudo-polyps, juvenile polyposis, hyperplastic polyposis, familial polyposis coli, Turcot's syndrome, Cowden syndrome and Peutz-Jeghers syndrome.

Hyperplastic polyps require a distinction from sessile serrated adenoma or polyps (SSA/P) which demonstrate identical histopathological features and can undergo malignant transformation. Also, enlarged, right sided hyperplastic colonic polyps can delineate a possible malignant conversion and could be reclassified as sessile serrated adenoma/polyp (SSA/Ps) [6,7].

Diminutive or cauterized hyperplastic polyps can be suitably distinguished with Ki-67 immune stains. Sessile serrated adenoma/polyp are predominantly right sided and display aberrant crypt architecture with serration of basal crypts [7].

Screening recommendations

Benign and asymptomatic hyperplastic polyps are devoid of an appropriate endoscopic distinction from adenomatous polyps.

Cogent screening procedures for emergence of colorectal carcinoma recommend the adoption of a colonoscopy commencing at 50 years for normal population and an antecedent screening for possible malignant emergence. Screening can be discontinued in instances where the life expectancy is beneath < ten years [7,8].

Probability of malignant conversion of colonic polyps is encountered with cogent pathologies such as adenomatous polyp, serrated polyp, polyp depicting high grade dysplasia, polyps enunciating > 25% villous histological features, polyp exceeding one centimetre diameter, polyp situated within the proximal colon and quantifiable polyps exceeding three.

Appropriate monitoring with follow up colonoscopy is recommended at an interval of 10 years in the absence of additional polyps or the appearance of distal, miniature hyperplastic polyps, at five year interval with the occurrence of miniature, sessile serrated polyps devoid of dysplasia, within three years in concurrence with large, sessile serrated polyps or polyps delineating dysplasia or a traditional serrated adenoma [7,8].

Specific guidelines for singular or two, miniature tubular adenomas mandate adequate screening in five to ten years, within three years when associated with minimally three to ten adenomas or a singular adenoma beyond ten millimetre dimension, in subjects delineating the occurrence of hyperplastic polyps within three years and in instances exceeding quantifiably ten adenomas.

Alternatively, screening with colonoscopy is mandated within three years for a singular or multiple adenomas demonstrating villous features or high grade dysplasia [7,8].

Subjects with a family history of polyps can benefit from a screening colposcopy. Hereditary nonpolyposis colon cancer mandates a screening colposcopy on an annual basis or once in two years [8].

Investigative assay

Physical examination is unremarkable although prolapsed rectal polyps can be palpated on a digital rectal exam. Faecal occult blood test (FOBT) as obtained and examined by a digital rectal exam (DRE) is not as conclusive as is the presence of occult blood discerned on spontaneous stool examination.

A preliminary complete blood count to rule out anaemia due to chronic bleeding and basal metabolic profile to exclude electrolyte disturbances as cogitated with hyper-secretory adenomas is required [8,9].

Guaiac (g FOBT) faecal occult blood employs a chemical indicator of haemorrhage to produce a colour modification.

Immune histochemical (i FOBT) faecal occult blood or faecal immune histochemical assay adopts the detection of specific antibodies towards human haemoglobin.

Evaluation of deoxy ribonucleic acid (DNA) within the stool is a sensitive indicator albeit minimally specific as compared to faecal immunochemical test for evaluating haemorrhagic stools [8,9].

Colonoscopy is the gold standard for detection of polyps wherein a polypectomy is performed and further management is contingent to histopathological assessment. However, detectable polyps can be missed on account of inadequate colonic preparation, miniature polyps, right sided colonic polyps and an inexperienced endoscopic surgeon. Subjects depicting ten colorectal adenomas mandate an assessment of MUTYH and APC genes [8,9].

Contemporarily, screening for colorectal polyps is achieved by a computed tomographic colonography (CTC), a procedure which necessitates excellent bowel preparation. Sensitivity of detection of flattened polyps is reduced with the employment of CTC.

Double contrast barium enema and colon capsule endoscopy can be adopted to discern colorectal polyps [8,9].

Enhanced optical technologies are a contemporary methodology of delineating colorectal polyps which are applicable in differentiating neoplastic from non-neoplastic colonic polyps and comprise of a narrowed spectrum endoscopy narrow band imaging (NBI), confocal laser endo-microscopy (CLE), Fujinon intelligent chromo-endoscopy (FICE) and image enhanced endoscopy (i-scan) [9].

Therapeutic options

Typically, hyperplastic polyp undergoes a tissue excision with fulguration.

Colonic polypectomy is a procedure which can be adopted during colonoscopy employed for diagnostic and therapeutic manoeuvers. Snare polypectomy with electrocautery is beneficial for excising pedunculated polyps or for mucosal resection of sessile polyps.

A comprehensive, total colectomy along with ileo-rectal anastomosis or procto-colectomy ileal pouch anal anastomosis is a prophylactic procedure which can be adopted for managing colorectal polyps [8,9].

Cogent colectomy is recommended for resected polyps delineating features of malignant transformation such as infiltration of lower one third of colonic mucosa, indeterminate or infiltrated surgical margins, invasive resection perimeter below one millimetre, lymphoid and vascular tumour invasion and appearance of a poorly differentiated carcinoma. Additionally, enhanced incidence of lymph node metastasis is exemplified [8,9].

Familial adenomatous polyposis (FAP) and MUTYH - associated polyposis (MAP) necessitate cogent therapy on account of persistent bleeding or when enunciating innumerable polyps. Surgical extermination of colonic polys is accompanied by an alleviation of the condition [9].

Figure 1: Hyperplastic polyp with an admixture of goblet cells and columnar cells, surface maturation and lack of atypia [10].

Figure 1: 2Hyperplastic polyp with columnar mucosa configuring villi and tubules along with surface maturation and minimal atypia [11].

Figure 3: Hyperplastic polyps with tubulo-villous architecture, minimal mitosis and unremarkable intervening stroma [12].

Figure 4: Hyperplastic polyps with tubular configuration, goblet cell and columnar epithelial lining and basal regeneration [13].

Figure 5: Hyperplastic polyp with tubulo-villous architecture, mucin-poor epithelial lining and uninvolved encompassing lamina propria [14].

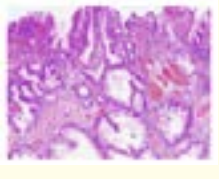

Figure 6: Hyperplastic polyp with tubulo-villous arrangement, columnar cell lining, absence of atypia and basal regeneration [15].

Figure 7: Hyperplastic polyp with mixed articulations, goblet cell and columnar epithelium admixture, absence of mitosis and normal lamina propria [16].

Figure 8: Hyperplastic polyp with surface serrations, maturation, columnar cell hyperplasia and tubulo-villous articulations [17].

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10. Image 1 Courtesy: Pathpedia.com.

11. Image 2 Courtesy: Basic medical key.

12. Image 3 Courtesy: Wikimedia commons.

13. Image 4 Courtesy: Research gate.

14. Image 5 Courtesy: Libre Pathology.

15. Image 6 Courtesy: Pathology outlines.

16. Image 7 Courtesy: Flickr.com.

17. Image 8 Courtesy: Webpathology.com.