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Editorial

The Cannulated Amalgamation-Tubular Adenoma

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Tubular adenoma is denominated as a neoplastic polyp associated with low grade epithelial dysplasia. Preponderantly emerging within colon, tubular adenoma may configure as a precursor to invasive adenocarcinoma of colorectal region. Singular tubular adenoma is characteristically associated with an estimated < 25% villous component. Tumefaction may be incidentally discerned upon colonoscopy [1,2]. Enhanced consumption of fruits, dietary fibre or folate decimates proportionate metamorphosis of tubular adenoma or possible emergence of conventional colorectal adenoma [1,2]. Tubular adenoma is commonly encountered in individuals >50 years wherein disease incidence is enhanced in elderly individuals. A male predominance is observed. Generally, lesions are common within left colon comprising of left half of transverse colon, splenic flexure, descending colon and sigmoid colon along with rectum [1,2]. Tubular adenoma exhibits a distinctive spectrum of metamorphosis from adenoma to carcinoma comprised of genetic mutations within KRAS, TP53 and APC genes besides β-catenin. Genetic mutations of KRAS appear preponderant in tubular adenomas [1,2]. Activation of KRAS initiates downstream signalling mechanisms which influences cellular proliferation, anti-apoptosis and survival. Aforesaid phenomenon are typically associated with advanced tubular adenomas. Additionally, activation of Wnt pathway induces nuclear accumulation of β-catenin with subsequent transcription and cellular proliferation [1,2]. Chromosomal mutations are comprised of loss of function of APC gene or mutations of β-catenin, manifestations which circumvent degradation by APC gene [1,2]. Contingent to villous component, tubular adenoma is categorized into. TA1 lesions which demonstrate < 1% villous component, minimal overexpression of p53, mutations within KRAS gene and absence of MGMT [1,2]. TA2 lesions are comprised of up to ~ 20% villous component, enhanced proportionate mutations within TP53 and KRAS and decimated MGMT [1,2]. Generally, lesions < one centimetre magnitude are asymptomatic and may be discerned with screening colonoscopy. Tumefaction > one centimetre magnitude is prone to gastrointestinal haemorrhage or intestinal obstruction. Secondary iron deficiency anaemia or possible progression to colorectal carcinoma may be observed [1,2]. Upon gross examination, tubular adenoma appears intensely reddish and may be sessile or pedunculated [1,2]. Severe dysplasia or malignant metamorphosis of layering epithelium is indicated by villous architecture, magnitude > one-centimetre, superficial mucosal ulceration or friable lesions [1,2].

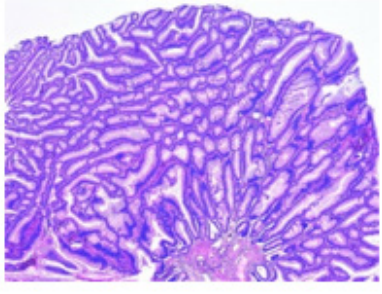

Figure 1: Tubular adenoma composed of crowded glandular articulations with basal, hyperchromatic nuclei, loss of polarity, minimal intervening lamina propria and variable mucin secretion [5].

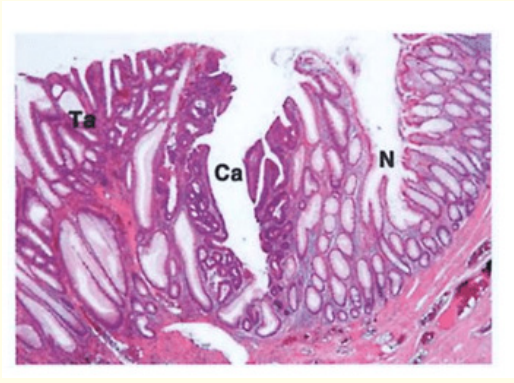

Figure 2: Tubular adenoma demonstrating focal dysplasia with nuclear crowding, hyperchromatic nuclei, glandular configurations and loss of polarity of basal epithelial cells with adjacent normal colonic mucosa [6].

Upon microscopy, colonic mucosa appears polypoid and exhibits dysplastic, stratified and pseudo-stratified superimposed epithelium wherein columnar epithelial cells are imbued with elongated, hyperchromatic nuclei with pseudo-stratification [3,4]. Characteristically, dysplasia is low grade. Nevertheless, high grade dysplasia may ensue which enunciates architectural modifications as a cribriform pattern, luminal necrosis and cytological features as decimated polarity of basal epithelial cell layer and vesicular nuclear chromatin with conspicuous nucleoli [3,4]. Commonly, distinctive, abrupt transformation from normal colonic mucosa into dysplastic mucosa can be observed. Mucin secretion is variable and decimated. Clear cells are exceptional [3,4]. Focal osseous or squamous metaplasia can be delineated along with occurrence of Paneth cells [3,4]. Benign, ectopic glandular articulations appear encompassed by lamina propria or admixed with hemosiderin pigment deposits. Pseudo-infiltration may simulate tumour progression into frank adenocarcinoma. Tubular adenoma is immune reactive to BCL2. Foci of dysplasia enunciate enhanced reactivity to CEA [3,4]. Tubular adenoma is immune non-reactive to p53 [3,4]. Tubular adenoma can exemplify aneuploidy [3,4]. Tubular adenoma requires segregation from lesions such as inflammatory polyp, tubule-villous adenoma, villous adenoma, familial juvenile polyposis, hyperplastic polyp, familial polyposis coli, Turcot syndrome, Peutz-Jeghers syndrome, Cowden syndrome and PTEN hamartoma, Cronkite- Canada syndrome, attenuated familial adenomatous polyposis or serrated polyps [3,4]. Cogent histological examination of tubular adenoma is confirmatory [3,4]. Colonoscopy is a diagnostic procedure wherein tubular adenoma may additionally be alleviated through polypectomy [3,4]. Severe mucosal dysplasia enlarged polyps and high-grade lesions are suitably treated with endoscopic mucosal resection or partial colectomy [3,4].

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5. Image 1 Courtesy: Webpathology.com.

6. Image 2 Courtesy: Research gate.