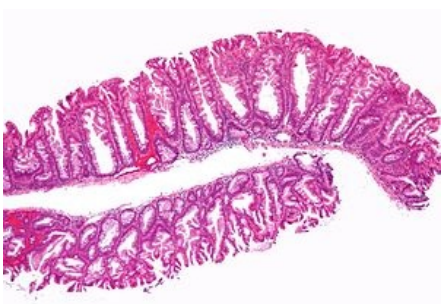


Serous lesions

Serate lesions (serrate lesions, serrate adenomas, serrate adenomas) are a group of adenomas of the large intestine, from which at least a third of all colorectal cancers arise (data in the literature vary). Serate lesions were first described by Longacre and Fenoglio-Preiser in 1990 as the result of an analysis of a group of colorectal polyps with mixed features of both hyperplastic polyp and adenoma. Serous lesions have several subtypes that differ in their risk of developing malignancy. Serate lesions owe their name to the fact that the crypt epithelium resembles a saw blade in its arrangement, more precisely, the protruding epithelial cells resemble the teeth of a saw. This characteristic arrangement is mainly caused by a disorder of apoptosis. According to the histological morphology, according to WHO (2010), serrate lesions are classified as follows:



Sessile serous adenoma, H&E. The arrangement of the epithelium in the wall of the crypts resembles the teeth of a saw, giving rise to the name of the group of lesions (eng. *serration* = serrations).

- hyperplastic polyp (HP, HPP)
- sessile serous adenoma/polyp 1 (SSA/P)
 - — without cytological dysplasias
 - — with cytological dysplasias (SAAD)
- traditional serous adenoma (TSA)

1 The terms *adenoma* and *polyp* are used, at least in English texts, as synonyms.

Sporadic hyperplastic polyps, which account for up to 90% of all serous lesions, are very unlikely to present a risk of progression to colorectal cancer. Both sessile serous adenomas and traditional serous adenomas represent a risk of developing colorectal cancer, which is probably higher than in the case of conventional adenomas.


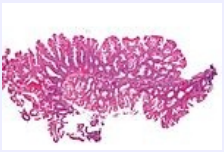
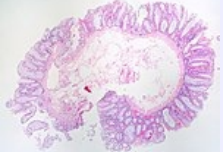
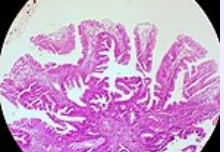
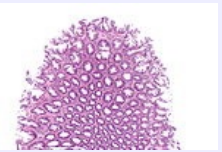
The molecular mechanism of the formation of serous lesions and the eventual malignant tumor is different from the traditional adenoma-carcinoma pathway, which involves the mutation of the APC gene , as well as from the direct mutation of the mutator genes , which is the cause of Lynch syndrome . There is talk of a **serous pathway** leading to carcinoma with a hypermethylation phenotype. A key step in the serate pathway is apparently mutations of KRAS or BRAF , protein kinases involved in the MAPK signaling pathway, followed by differently expressed hypermethylation of CpG islands in the regulatory regions of mutator genes, resulting in the so-called CIMP phenotype (CpG Island Methylated Phenotype). This results in suppression of the expression of repair proteins and the cell becomes more susceptible to the development of other somatic mutations. The exact sequence of mutations and disorders during the serate pathway is not known, it is likely that several partially interconnected pathways are involved. The following phenotypes of colorectal carcinomas are usually distinguished, their initiation was caused by a serous lesion:

1. BRAF mutation and significant CpC methylation (CIMP-H)
 1. associated with significant microsatellite instability (MSI-H)
 2. associated with microsatellite stability (MSS)
2. KRAS mutation and CpC low methylation (CIMP-L), microsatellite stability (MSS)

Although it is actually two pathways, one of which branches further, it is referred to as the serous pathway of colorectal cancer formation, in order to express its difference from the pathway of cancer formation in the conventional adenoma-carcinoma sequence and from the formation of tumors in Lynch syndrome.

Links

Virtual Preparations

				
{{Description1}}	{{Description2}}	{{Description3}}	{{Description4}}	{{Description5}}
source: [{{source_url1}}] [{{source_description1}}]	source: [{{source_url2}}] [{{source_description2}}]	source: [{{source_url3}}] [{{source_description3}}]	source: [{{source_url4}}] [{{source_description4}}]	source: [{{source_url5}}] [{{source_description5}}]

Related Articles

- Serous lesions/PGS
- Colorectal cancer
- Treatment of liver metastases in colorectal cancer
- Vienna Classification of Gastrointestinal Neoplasia (2002)

Literature

- REX, D. K. – AHNEN, D. J. – BARON, J. A.. Serrated lesions of the colorectum: review and recommendations from an expert panel. *Am J Gastroenterol* [online]. 2012, vol. 107, p. 1315-29; quiz 1314, 1330, Available from <<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3629844/?tool=pubmed>>. ISSN 1572-0241.
- FU, X. – QIU, Y. – ZHANG, Y.. Screening, management and surveillance for the sessile serrated adenomas/polyps. *Int J Clin Exp Pathol*. [online]. 2014, vol. 7, p. 1275-1285, Available from <<http://www.ijcep.com/files/ijcep1401068.pdf>>. ISSN 1936-2625.

External links

- PathologyOutlines.com. *Colon tumor > Polyps > Serrated adenoma/polyp* [online]. ©2011. [cit. 6/2014]. <<http://www.pathologyoutlines.com/topic/colontumorserrated.html>>.