optimally sparing the normal critical tissues surrounding the target to be irradiated [7], like SBRT (Stereotactic Body Radiation Therapy) for small lesions, in order to increase the delivered radiation dose in selected cases. SBRT has also been proposed to preoperatively irradiate the posterior margin area, in an attempt to increase the R0 resection rate in this difficult to resect area [8]. All these aspects will be described and precisely discussed at the time of the conference.

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Thursday, 22 March, 16:00−17:30

Session IV. Different Cancer Types in the Oesophagus and Stomach

PG 5.01 SPEAKER ABSTRACT

Modern endoscopic imaging of gastrooesophageal lesions: Different techniques for different locations

R. Kiesslich. *Interdisziplinäre Endoskopie, Uniklinik Mainz, Mainz, Germany*

The prognosis of esophageal neoplasia is closely related on the stage of the disease at the time of detection. Early neoplastic lesions have an excellent prognosis in contrast to more advanced stages that usually have a dismal prognosis. Therefore, the early detection of these lesions is of great importance.
Several new endoscopic techniques have been introduced to improve the endoscopic detection of early lesions. The most important improvement, in general, has been the introduction of high-resolution/high-definition endoscopy into daily clinical practice. The value of superimposing techniques such as chromoendoscopy, narrow band imaging and computed virtual chromoendoscopy onto high-resolution/high-definition endoscopy do further refine the characterization of lesions and guide endoscopic therapy.
Furthermore endomicroscopy enables in vivo histology during ongoing endoscopy at subcellular resolution. This leads to immediate histological diagnosis of Barrett's esophagus and associated neoplasia. Endomicroscopy will also open the door for functional and molecular imaging as initial studies have shown.
Standardized teaching of the new diagnostic possibilities will be of fundamental importance to provide the affected patients with the best standard of care.

Reference(s)

Kiesslich R, Goetz M, Hoffman A, Galle PR. New imaging techniques and opportunities in endoscopy. Nat Rev Gastroenterol Hepatol. 2011 Sep 6;8(10):547−53.
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Kiesslich R, Canto MI. Confocal laser endomicroscopy. Gastrointest Endosc Clin N Am. 2009 Apr;19(2):261−72.
Curvers WL, Kiesslich R, Bergman JJ. Novel imaging modalities in the detection of oesophageal neoplasia. Best Pract Res Clin Gastroenterol. 2008;22(4):687–720.

PG 5.02 SPEAKER ABSTRACT

Adenocarcinoma of the GEJ: gastric or oesophageal cancer?

J. Rüschoff. *Pathologie Nordhessen, Kassel, Germany*

Adenocarcinomas of the Oesophagus (OC) and Stomach (SC) are two different types of tumors that showed marked changes of incidence with constant rise of OC and decrease of SC during the last three decades in the Western World. Both types arise by two different etiological mechanisms − reflux and Barrett metaplasia in OC and H. pylori infection in SC. However, much confusion exists about the tumors at the distal third of oesophagus and gastric cardia. Adenocarcinomas at the gastrooesophageal junction (GOJ) have recently been defined as a third tumor type (GOJ cancer) by WHO Classification of Tumours of the Digestive System (2010). This classification is replacing the anatomic system of Siewert suggesting that type I (distal oesophageal) is different from type II (cardiac) and type III (subcardiac) adenocarcinoma. These tumors have now been included within the oesophageal category by UICC Cancer Staging Manual (7th ed., 2010). Besides morphologic heterogeneity (WHO 2010) there is strong evidence that at least two main different molecular pathways of carcinogenesis exist. Based on differences in the immunohistological-phenotype (intestinal vs gastric type cancer) and the accompanying background mucosa (Barrett vs cardiac type) about 70% of GOJ adenocarcinomas turned out to be associated with Barrett's metaplasia (*intestinal-type neoplastic pathway*) and about 1/3 with metaplastic columnar epithelium (*non-intestinal neoplastic pathway*). These pathways reflect distinct clinical and prognostic groups being significantly different with respect to the prevalence of potential therapy target such as EGFR and Her2/neu (Demicco et al., Mod Pathol 2011). In the latter anti-Her2 therapy recently turned out to be effective (Bang et al., Lancet 2011) which was based on specifically developed Her2 testing guidelines (Rüschoff et al., Mod Pathol 2012). Finally, the impact of the intestinal and non-intestinal pathway concept on current recommendations of AGA to not perform endoscopic surveillance in patients solely with (non-intestinal) columnar-type epithelium in the esophagus (http://www.gastrojournal.org/article/S0016-5085%2811%2900084-9/fulltext#sec2) will be challenged.

PG 5.03 SPEAKER ABSTRACT

Molecular mechanisms in gastric cancer: Basis for therapy?

R. Seruca. *IPATIMUP, Porto, Portugal*

Gastric cancer (GC) is one of the leading causes of cancer-related death worldwide, even though its incidence and mortality rates have been declining in recent decades. At initial diagnosis, most GC patients present an advanced disease stage with a high risk of relapse after surgical treatment. Various multimodal therapy regimens are used to improve the patient prognosis, with limited success. The high prevalence of incurable disease produces a heavy burden on patients' care which has a huge effect on healthcare resources. E-cadherin alterations/deregulation is a frequent event in gastric carcinogenesis, as an initiation event in more than 50% of diffuse GC, and as a progression event, by increasing epithelial cell invasion, in more than 70% of all gastric cancers. Recently, E-cadherin was suggested to act as a cell membrane receptor interacting with many signalling molecules. In this regard molecules interacting with E-cadherin became central targets for therapeutic intervention in gastric cancer. An increasing number of genetic and epigenetic alterations have also been associated with distinct histological types of gastric cancer. We will discuss the involvement of E-cadherin, EGFR, ERBB2, MMR genes, KRAS, and PIK3CA in the development and progression of gastric cancer and their role as biomarkers or as novel putative targets for therapy. We will also pay special attention to define the subset of gastric carcinoma which may benefit from EGFR and Notch inhibitors.

PG 5.04 SPEAKER ABSTRACT

Why is there a change in patterns of GE cancer?

J. Jankowski. *Centre for Digestive Diseases, Barts and the London School of Medicine and Dentistry, London, UK*

Abstract not available.

Friday, 23 March, 08:30−10:00

Session V. Choosing the Best Treatment for Oesophageal Cancer

PG 6.01 SPEAKER ABSTRACT

Who is a candidate for endoscopic surgery?

T. Oyama. *Gastroenterology, Saku Central Hospital, Nagano, Japan*

The incidence of lymph node metastasis is correlated with some pathological findings such as invasion depth, histological type and lymphatic or venous permeation [1,2]. However, these pathological findings could not be learned