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EDITORIAL

In this issue

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This issue opens with a historical perspective on the clinicopathological conference, essential element in the communication between pathology and whichever clinical discipline. The issue furthermore contains a good harvest of interesting research papers.

Kreft et al. (DOI 10.1007/s00428-015-1785-9) take a close look at diagnostic criteria for graft-versus-host disease. When full-blown, this condition shows characteristic morphology but in an early phase, or when the case is complicated by viral infection or toxic side effects of medication, the diagnosis can be difficult. To get to consensus diagnostic criteria, five pathologists from different institutions independently evaluated a biopsy series. Reading of the biopsies prior to development of consensus criteria clearly left room for improvement in terms of diagnostic consensus. This significantly improved once consensus criteria had been agreed upon. Some discordancies remained, notably in delicate differential diagnostic situations. These can only be resolved by integrating histopathological findings with clinical information, typically in a clinicopathological conference.

Other papers report on interesting new tissue-based biomarkers. Jernman et al. (DOI 10.1007/s00428-015-1795-7) explore PROX1 as a marker in neuroendocrine tumors (NETs). PROX1is a homeobox transcription factor target of oncogenic Wnt signaling and is expressed in colorectal cancer. Given the ongoing discussions about malignant potential of gastroenteropancreatic NETs, the group decided to explore a role for PROX1 in malignant progression of rectal NETs. By immunohistochemistry, expression of PROX1 was stronger in metastasized cases and correlated with patient prognosis. The authors conclude that PROX1 may be involved in progression of rectal NETs as a part of the Wnt pathway. Whether or not PROX1 will turn out to be a useful diagnostc marker remains to be examined further.

The question which pleomorphic adenomas might progress to a carcinoma ex pleomorphic adenoma and which molecular mechanisms are involved is explored by Souza et al. (DOI 10.1007/s00428-015-1804-x). To this end, they investigated expression of cell cycle markers p16, cyclin D1, CDK4, E2F, and Rb by immunohistochemistry in cases of pleomorphic adenoma with or without recurrence and carcinoma ex pleomorphic adenoma. It turns out that recurrent pleomorphic adenomas and carcinomas ex pleomorphic adenoma show strong staining for p16, cyclinD1, and E2F, while in pleomorphic adenomas that did not recur, weak or no expression is found. Cell cycle proteins might therefore be involved in recurrence and malignant transformation of pleomorphic adenoma. Prospective studies will be necessary to validate clinical relevance of this marker in terms of recurrence prediction.

Finally, Krenacs et al. (DOI 10.1007/s00428-015-1796-6) studied myocyte enhancer binding factor 2 B (MEF2B), a member of the evolutionary conserved transcription family MEF2, as a new B cell lineage marker. In nonneoplastic lymphoid tissues, intense nuclear MEF2B immunostaining was confined to germinal center B cells while plasma cells showed weak nuclear staining. MEF2B staining was equally strong in follicular lymphoma even in bone marrow biopsies, in Burkitt lymphoma, nodular lymphocyte predominant Hodgkin lymphoma, a large majority of mantle cell lymphoma, and diffuse large cell B cell lymphoma cases but almost consistently negative in marginal zone lymphoma. They conclude that MEF2B specifically labels normal germinal center B cells and might be useful in the differential diagnosis of small B cell lymphomas.

The cover image is taken from this paper and shows MEF2B staining in a germinal center.

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