Types of Tumors Should be Tested?

Abnormal KIT Expression?

As mentioned above, we can also perform IHC for KIT. Aberrant expression of this gene has been detected include canine and feline mast cell tumors (MCTs) and canine gastrointestinal stromal tumors (GIST) and have been suspected in canine oral melanomas.

ITD mutations in exon 11 of c-Kit have been detected in about 20 to 30 percent of canine cutaneous MCTs. ITD mutations in exon 8 of c-Kit are less common and have been detected in 2 to 5 percent of canine cutaneous MCTs. Both of these PCR tests are included in our canine mast cell tumor prognostic panel, but can also be ordered as individual tests.

Most cutaneous MCTs in cats are benign and cured by complete surgical excision. Occasionally, more aggressive feline cutaneous, enteric, and visceral MCTs (generally affecting the spleen and/or liver) are seen. For these more aggressive forms, we also offer PCR to detect mutations in exon 8 of c-Kit that have been detected more commonly in non-cutaneous, aggressive tumors.

In addition, it is important to perform immunohistochemical (IHC) labeling for the protein encoded by c-Kit, a tyrosine kinase receptor called KIT, in all canine nonangiogenic gastrointestinal sarcomas. Those tumors that are positive for KIT are called GISTs and are a particularly aggressive type of gastrointestinal sarcoma. Many canine GISTs also have an activated mutation in exon 11 of c-Kit.

In What Other Ways Can c-Kit Be Evaluated?

As mentioned above, we can also perform IHC for KIT. Aberrant expression of this protein can be identified in cells based on the pattern of immunolabeling. For canine MCTs, there are three types of KIT patterns (1, 2, and 3). In feline MCTs KIT expression has been evaluated in some studies, but is of less prognostic value than in canine MCTs. Any positive immunolabeling for KIT in a nonangiogenic nonlymphogenic gastrointestinal sarcoma is consistent with a GIST.

Why is it Important to Know If a Tumor Has a c-Kit Mutation or Abnormal KIT Expression?

Detection of c-Kit mutations in exon 11 predicts a high rate of metastasis and mortality in canine MCTs, but both MCTs and GISTs with such mutations have been shown to respond well to tyrosine kinase-inhibiting (TKI) therapies, e.g. toceranib phosphate (Palladia) and masitinib mesylate (Kinavet-CA). While no prognostic value has been established for detecting mutations in exon 8 of c-Kit in canine and feline MCTs, those tumors are also expected to respond to TKIs. In addition, aberrant KIT expression patterns (patterns 2 and 3) in canine MCTs have been linked with decreased survival as has cytoplasmic localization of KIT in feline MCTs.

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