INTISARI

Gastrointestinal stromal tumor (GIST) merupakan neoplasma non-epitel paling umum pada saluran pencernaan yang disebabkan oleh mutasi aktivasi pada proto-onkogen KIT atau PDGFR (*platelet derived growth factor receptor alpha*) yang mengode reseptor tirosin kinase. Imatinib mesilat, suatu *tyrosine kinase inhibitor* (TKI) adalah terapi lini pertama pada GIST yang dipakai dalam jangka panjang. Imatinib dapat menyebabkan terjadinya hiperpigmentasi mukosa palatum. Tujuan studi ini adalah untuk menganalisis profil maturasi epitel mukosa palatum keras pada penyintas *Gastrointestinal stromal tumor* yang diterapi imatinib berdasarkan durasi terapinya.

Penelitian observasional dengan pendekatan *cross-sectional* ini melibatkan tujuh penyintas GIST. Maturasi epitel mukosa palatum keras dievaluasi melalui sitologi eksfoliatif menggunakan metode *liquid-based cytology* (LBC). Preparat sitologi diamati menggunakan mikroskop cahaya, kemudian jumlah sel epitel dianalisis menggunakan *maturation index* (MI) yang menggambarkan rasio persentase sel parabasal, intermediet, dan superfisial serta *maturation value* (MV).

Hasil menunjukan pada umumnya peningkatan proporsi sel intermediet disertai penurunan sel superfisial yang mencerminkan pergeseran pola maturasi epitel ke arah kiri. Analisis korelasi *Spearman* menunjukkan hubungan positif lemah antara durasi terapi imatinib dan nilai maturasi epitel mukosa palatum keras ($r = 0,38$; $p = 0,40$). Kesimpulan dari penelitian ini adalah durasi terapi imatinib tidak berhubungan dengan profil maturasi epitel mukosa palatum keras pada penyintas GIST, meskipun sebagian besar menunjukkan pergeseran maturasi epitel ke arah kiri.

Kata kunci: Tumor stroma gastrointestinal, imatinib, epitel mukosa palatum keras, maturasi, indeks maturasi, nilai maturasi

ABSTRACT

Gastrointestinal stromal tumor (GIST) is the most common non-epithelial neoplasm of the gastrointestinal tract, largely caused by activating mutations in the proto-oncogene KIT or PDGFRA (platelet-derived growth factor receptor alpha), which encodes the tyrosine kinase receptor. Imatinib mesylate, a tyrosine kinase inhibitor (TKI), is the first-line therapy for GIST and is used long-term. Imatinib can cause hyperpigmentation of the palatal mucosa. The aim of this study was to analyze the maturation profile of the hard palate mucosal epithelium in gastrointestinal stromal tumor survivors treated with imatinib based on the duration of therapy.

This observational study with a cross-sectional approach involved seven GIST survivors. Maturation of the hard palate mucosal epithelium was evaluated through exfoliative cytology using the liquid-based cytology (LBC) method. Cytology preparations were observed using a light microscope, then the number of epithelial cells was analyzed using a maturation index (MI) that describes the percentage ratio of parabasal, intermediate, and superficial cells, as well as the maturation value (MV).

The results show a general increase in the proportion of intermediate cells accompanied by a decrease in superficial cells, reflecting a shift in the epithelial maturation pattern to the left. Spearman's correlation analysis showed a weak positive correlation between the duration of imatinib therapy and the epithelial maturation score of the hard palate mucosa ($r = 0.38$; $p = 0.40$). The conclusion of this study is that duration of imatinib therapy was not associated with the maturation profile of the hard palate mucosal epithelium in GIST survivors, although most showed a shift in epithelial maturation to the left.

Keywords: Gastrointestinal stromal tumor, imatinib, hard palate mucosal epithelium, maturation, maturation index, maturation value