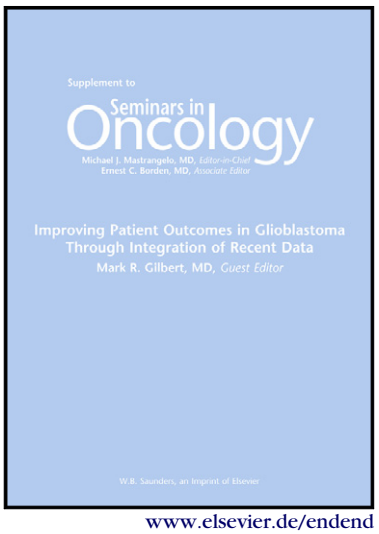

Guest Editorial: Platelets and Cancer

Brian I. Carr MD, FRCP, PhD.

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Guest Editorial: Platelets and Cancer

Abstract

Platelets have a well-studied role in coagulation and in thromboembolism. The latter was recognized to be a feature of occult cancer over 100 years ago. It has been increasingly appreciated, that as a component of the tumor microenvironment, platelets also have important functions in the mechanisms involved in carcinogenesis, tumor growth, tumor angiogenesis, tumor-related host organ inflammation and immune responses, tumor metastasis and in the modulation of tumor therapy. Therapeutic alteration of platelet numbers and function has increasingly gained attention for cancer prevention, survival prolongation and possibly for therapy. The idea that systemic response to the presence of cancer, including the platelet lymphocyte ratio (PLR) is an independent prognostic factor in many tumor types, has recently gained support. Platelets are thus seen as predictors of cancer prognosis, mediators of cancer biology and the subject of therapeutic intervention.

Article

Venous thromboembolism was first noted to be associated with cancer by Trousseau (1). It tends to be recurrent, migratory, involves multiple body sites and can be resistant to anticoagulation (2, 3). This cancer-associated thromboembolism was also found to be associated with thrombocytosis (4, 5). Platelets have come to be viewed, both as a systemic reaction to the presence of cancer (6-9) as well integral mediators of cancer biology (10-13). The actions of platelets on tumors may be direct (14, 15) or as part of the tumor microenvironment (16, 17), although these are not mutually exclusive.

Thrombocytosis can occur in association with many cancers, including those of ovary (18), GI tract (19) and liver (20, 21). Platelets typically derive from pro-platelet protrusions of megakaryocyte cytoplasm, by processes that depend on cell-cell interactions in the bone marrow microenvironment (22), as well as cytokines such as thrombopoietin, which is produced in the liver and by many tumors. Recent clinical evidence points to a feed-back loop involving interleukin-6, thrombopoietin and thrombocytosis, as described for ovarian cancer patients (18).

Several mechanisms have been suggested for the involvement of platelets in cancer development,