Opinion

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Cancer Stem Cells Facilitated Chemoresistance In Gastric Cancer: Problems and Opening

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ABSTRACT

Abbreviation

TIC: Tumour-Initiating Cells; CSC: Cancer Stem Cells; GCC: Gastric Cancer Chemoresistance; MDR: Multi Drug Resistance; ABC: ATP-Binding Cassette; NSC: Normal Stem Cells; ALDH: Aldehyde Dehydrogenase; TKF: Tyrosine Kinase Family; PDFGR: Platelet-Derived Growth Factor Receptor

Opinion

Dear Editor, I have a particular interest in the current therapeutic challenges related to Gastric cancer chemoresistance (GCC). I appreciate the Author for the quality of information about GC biomarker and therapeutic target [1]. In this opinion letter, I would like to get your attention to the mysteries' chemoresistance machinery in gastric cancer. Cancer stem cells (CSCs) known as tumour-initiating cells (TICs) are heterogeneous and differentiated cells [2]. Recently demonstrated that CSCs are responsible for tumour progression and metastasis lately drug resistance as well as tumour relapse due to their ability of self-renewal, asymmetrical division, and differentiation into diverse lineages in different cancer cells including GC [3-4]. Advanced gastric cancer patients required frequent chemotherapy, and chemoresistance is the main obstacle in the treatment of GC. Anticancer drugs are effective against proliferating progenitor/ differentiated tumour cells, but CSCs through the induction of a quiescent state can survive to chemotherapy [5].

There are several molecular mechanisms involved in multidrug resistance (MDR) such as the alteration in the activity of membrane transporters belong to the ATP-binding cassette (ABC) drug transporter family. ABC transporters are located in the plasma membrane and protect cells from toxin protein and xenobiotic. This transporter can reveal stem-like properties of CSCs to escape the cytotoxic effects of anticancer drugs and support chemotherapeutic resistance [6 -7]. CSCs share several normal stem cells (NSCs) like properties that provide long-term stability inside the tumour, including DNA damage repair capacity and resistance to apoptosis [8-9].

Additionally, researchers noted that aldehyde dehydrogenase (ALDH) is a functional marker in CSCs, higher ALDH activity is en route for chemoresistance to 5-fluorouracil and cisplatin drugs via Notch1 and Shh signalling pathway and confirmed poor survival in GC [10]. Further, Wang et al. mentioned that Axitinib a small molecule of the tyrosine kinase family (TKF), a derivative of benzamide and indazole that works as an inhibitor against the VEGF receptor and platelet-derived growth factor receptor (PDGFR). It acts on CSCs increasing the efficacy of chemotherapeutic drugs via inhibiting the ABCG2 drug transporters in vivo [11]. Hence, another report also suggested that inhibition of the ABC transporters family might be helpful in combination with chemotherapy treatment, in the eradication or removal of MDR cancer cells [12].

However, in recent few years, several molecular agents (trastuzumab, fluoropyrimidine, cisplatin, ramucirumab and paclitaxel) have been studied in various combinations with predictable treatment against GC. However, the success rate of targeting agents for GC has restricted. Targeting the molecular signalling pathways and

interference molecules among CSCs might be a promising therapeutic strategy. Further Growing attention is being paid to be the around clear elucidation of the molecular mechanisms of CSCs regulation, it may lead to the development of novel treatment strategies that target gastric cancer stem cells and use as a new weapon to Beat cancer.

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