ORIGINAL RESEARCH PAPER

Oncology

PLATELET COUNTING AND CANCER RELAPSE IN PATIENTS WITH SOLID TUMORS IN SOLCA-QUITO HOSPITAL

KEY WORDS:

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ABSTRACT

INTRODUCTION: The tumor cell undergoes a series of injuries on their way through the bloodstream to the development of a metastatic implant, for which activates platelets that allow you to move with ease by this means, the objective of this study is to relate the platelet count to a tumor relapse.

METHODS:A retrospective observational study was realized in June 2016, in which there were taken into consideration the last 100 patients who had died in this institution. 52 subjects were excluded, by what there were analyzed the information of 48 remaining individuals, the value of the platelet count at the start of cancer treatment, to the first tumor relapsing and its value premortem, it is considered to be a normal referential value between 150-450 K/uL.

RESULTS: Patients who presented progression hematogenous had elevation of the counting of thrombocytes to the relapsing with regard to the basal value of beginning of treatment, however we must stress that this value becomes significant when it rises above 17%

ANALYSIS: Given the results we conclude that the platelet count would help us as a tumor marker follow-up of a solid tumor, without forgetting that a percentage of patients present progression with platelet count within the normal range or lower, this group is due to lymphatic progression or Transcoelomic.

INTRODUCTION

The microenvironment in the bloodstream is hostile to the circulation of the tumor cell and its survival before extravasating and developing metastases, so models in mice suggest the recruitment of macrophages, neutrophils and platelets to protect tumor cells. 1.2

The tumor cell can add and activate platelets leading the initiation of a thrombus through a process known as Tumor cell-induced platelet aggregation (TCIPA). 2.5–10

During metastasis, the tumor cell enters the bloodstream giving rise to a metastatic niche.7

Platelets are small anucleated cell fragments, originating from precursor cells known as megakaryocytes in the bone marrow, that circulate in the blood in about 10 days. 7.8

In the blood the circulating tumor cell can activate platelets by direct contact or through the release of agonist mediators such as Adenosine di-phosphate (ADP), thrombin, thromboxane A2 (TXA2) or tumor associated proteins. 2,5,7,9-12

There are two main signaling pathways in platelet activation: One through glycoproteins (GP) VI and the other through type 2 lectin receptors (CLEC-2) that envelop the tyrosine phosphorylation cascade all this in contrast to soluble agonists such as thrombin, ADP and TXA2 that stimulate heterotrimeric G protein and induce signaling cascade. 5,9–11

GPIba forms a complex known as GPIba-IX-V being the largest ligand to vWF (von Wilebrand factor) at the vascular level, with respect to the tumor cell the role of the GPIba-IX-V complex in TCIPA indicates promoting metastasis, while that in other experiments this association is not observed.8,10,11 Platelets activated towards tumor cells by interaction of p-selectins are added thanks to GPIIb / IIIa - fibrinogen, encapsulating the tumor cell and protecting it from the action of NK cells. 2,7–11

The objective of the present study is to correlate the number of platelets in individuals diagnosed with solid tumors at the

beginning of their cancer-chemotherapy treatment, regardless of being adjuvant or neo-adjuvant therapy, in their relapse and premortem, considering the premortem condition to the last taking platelet count being this value days or weeks before death, during your stay in uci or prior to the palliative care service, in this way to be able to establish an oncological parameter for monitoring it and be able to use the platelet count as a tumor tracking marker.

MATERIALS AND METHODS

A retrospective, observational study was carried out, which was at the General Solón Espinosa Ayala Hospital "SOLCA NUCLEO DE QUITO" in June 2016, for which the list of the last 100 patients who had died in these institution was requested . From this number of patients, those with hematological pathology, pediatric patients (under 18 years of age), those who were with anemia or receiving colony stimulating factors were excluded, as well as those who did not have chemotherapeutic cancer treatment thus excluding 52 subjects

Of the remaining 48 individuals, the value of the platelet count was taken at the beginning of cancer treatment, at the first tumor relapse and its premortem value, these values were processed in the SYSMEX XN-1000 year 2015, whose reference range of platelet count is found between 150-450 K / uL.

The data was analyzed in the IBM-SPSS 23 and Microsoft EXCEL 2015 program.

RESULTS

Data were collected from 48 individuals, for which the number of platelets at the beginning of cancer treatment, a second take at the time of tumor relapse and a third premortem value were taken as a reference value, the latter value was taken days to weeks before of dying, before the pass to palliative care service or during your stay in intensive care before dying.

Of this total of 48 individuals, 39 individuals with hemato genous progression was evalue while 9 individuals had lymphatic or transcellomic progression.

Of this number of patients who presented hematogenous progression, it was divided into percentiles and was considered as significant elevation of platelet count when its value with respect to baseline of treatment initiation was greater than 17% (25th percentile).

It is important to indicate that in the group of patients with hematogenous progression there was a significant increase in platelet count with respect to its baseline value of 71.8%.

It is striking the fact of this increase with respect to its baseline, in this group of patients with respect to its baseline value, so the fall in the important value after starting treatment is also striking, it is important to indicate that this drop in the Platelets are due to the initiation of cancer treatment or bone marrow infiltration, it was also seen that the elevation, fall and new elevation of platelet count in patients with 2 or 3 relapses appeared the presence of a carp, so this We could call this phenomenon "platelet carp, this phenomenon was observed in 2 individuals.

FIGURE 1.- Distribution of platelet value

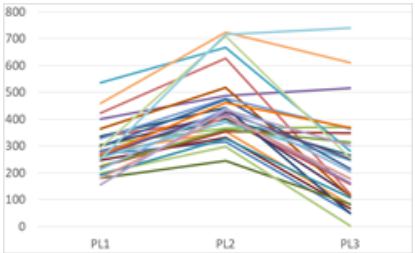


FIGURE 2.- Distribution of platelets over 17% in hematological group

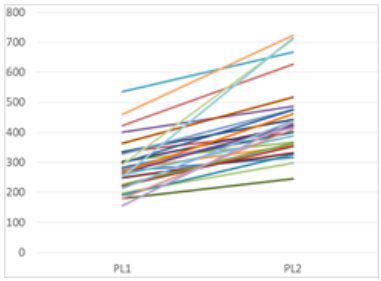
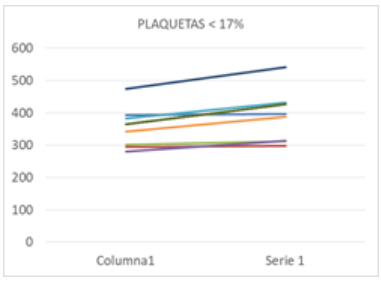


FIGURE 3.- Distribution of platelets less than 17% in hematological group



In the group of individuals who presented with lymphatic or transcellomic progression, none of them presented a platelet increase with the exception of one case, it should be noted that this single case is in the 17.03% percentile so it was not significant, we conclude that this phenomenon is due It does not depend on the platelet count at this level.

ANALYSIS

From the collected data, it can be analyzed that there are an increase in the platelet number of patients who presented tumor relapse, it is important to note that the increased value is within the normal laboratory ranges that are managed between 150-450 K in our institution / uL, however, it was observed during the investigation that the patients manage a basal range and during their first relapse they present an increase in it, we consider this value significant when it exceeds 17% of the baseline value.

In patients who followed for more relapses, an increase and fall in the platelet count similar to a carp was observed, a phenomenon that occurred as many relapses presented by the patient, said phenomenon we will baptize as "platelet carp".

The present study could represent a pattern of the use of platelet count as a low-cost follow-up tumor marker, alerting us to a possible tumor progression.

Let us not forget that there was a small percentage of patients who presented tumor relapse with a decrease in platelet count, or a non-significant elevation, which was reflected in lymphatic or transcellomic progression, for this reason their sensitivity would not be high, so we emphasize again which is important if you increase its value above 17% to its baseline value.

To conclude this finding, it could support a series of works that defend the use of antiaggregants as an oncological treatment, and perhaps their use delays the eventual tumor relapse.

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