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Elucidative Intersects and Divergences Involving Cancer and Malaria

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Abstract

In a comparative appraisal of the courses and end points (terminal) taken by both ailments, we noticed the existence of intersects and divergences in malaria and cancer infections.

Examining areas of intersections connoting similar trends: Malaria is a disease resulting from infection by a Protozoan *Plasmodium* parasites (various species exist), and it has been found that one of the causes of cancer is through infection by a virus (Human Papilloma Virus HPV) leading to cancerous conditions of various types, a prominent one of which is cervical cancer. Invariably, both malaria and cervical cancer could be treated.

Malaria is a progressive disease, following initial infection and the disease could be very invasive if untreated or ineffectively treated. It is capable of progressing from a mild condition referred to as uncomplicated or mild malaria, into the severe form as the parasite multiplies, migrates and invade other tissues. As the parasite gain access to the tissues of the brain, they cause damages, leading to one of the deadly forms particularly in children, called cerebral malaria, which has the potentials of being severe. The parasite in other organs of the body such as the liver and spleen causes various health affecting conditions such as spleenomegaly. Invariably, commencing from its route of entry, principally through infectious bites – it is shed into the blood stream, move along with the blood, first into the liver where several cells of the parasite are replicated (likened to a place where there is a boost in the number of parasites) in preparation for its onwards dissemination or spread of its disease state, with capacity to afflict and cause pathologies to several other tissues, organs and various regions in the human or mammalian body.

As in malaria also, there are invasive attributes in cancer which is connected to the stage of cancer metastases (either caused by pathogenic HPV or non-pathogenic but carcinogenic agents) during which the initially formed tumor (tumor cells) from the primary site start to spread by several processes, of which include rapid proliferation and migration of new tumor cells, engagement of the blood vessels (during angiogenesis), engagement of the lymphatic vessels and the fluid blood and lymph to spread new tumor cells to other tissues and organs (in other sites of the body) in the progression of the disease state from the primary site to the secondary site(s), during which the disease may have advanced from benign to the malignant tumor state, usually referred to by some Oncologists at this stage as the real cancer in an all encompassing process of tumourigenesis.

In cancer, as new tumor cells are formed, some of the adjoining or adjacent normal cells of the body get affected as these normal cells loose part of their basic cell functions, as pathologies of associated damages set in and these cells die. During malaria, there is a destruction of the red blood cells (rbcs) as the parasite invades, metabolizes and damages components and gross structure of the red blood cells forming tissues in the organs that the parasite invades, through various technical processes or events the parasite undergoes in its quest to attain spread, dominate the blood system and entrench its invasive habitation in the human or animal host.

In area of disease diagnosis, malaria and cancer diagnosis for infection, cell derangement in cancer (appearance of tumor cells), and pathologies, both involve use of histological techniques, (examination stained blood or swab pap smears, as it appropriately applies, alongside microscopy), molecular based diagnosis engaging polymerase chain reaction (PCR) of various types, analysis for hematological parameters, sero-diagnosis including immune-assays for levels of antibodies, CT scans on organs for pathologies attributable to damages to invaded or disease pathogenesis related chemo-attack of organs, among others.

Several types of nuclear receptor responsiveness and cell surface – receptor responsiveness are vital for the active proliferation of the new tumor cells (daughter cells) that are

rapidly being formed – generally during tumourigenesis. In malaria, as it manifests in the patient, it involves the existence of a plethora of receptors, receptor molecules and ligands of cell surfaces of both the parasite and the red blood cells that are involved in several multi-faceted events embraced in the all encompassing progression of the disease state and invasive success of the parasite in its human or mammalian host in untreated or poorly treated forms in drug resistance involving cases. Tumour cells basically re-orientate and reconstitute their cell structure and content during existence and activities. This attribute of tumor cells might not be totally uneventful in malaria as red blood cells invaded by *Plasmodium* parasites undergo structural and content based transformations involving (degradation of its hemoglobin, its moieties) deposition of various products (of molecules and compounds) into the red blood cells which are metabolic products from the activities of the parasite, into the red blood cells and the blood stream, parasite sequestration, cyto0adherence and rosette formation, all these features have a pooled effect that enhances killing or dismembering of the *Plasmodium* parasite invaded red blood cells.

For malaria, it has capacity for recrudescence in which if all the parasites are not well cleared during treatment with drugs, the few counts of parasites can rapidly multiply and starts causing damages all over again if un-noticed, alongside the re-emergence of symptoms. Cancer behaves likewise, as a few cancerous cells left and un-destroyed during chemotherapy can start proliferating once more from the site of the body where that served as primary site of invasion, once more, and if un-noticed in time, they could upscale in abundance (multiplicity), gain access to the blood stream and by angiogenesis find their way to other sites of the body to create a secondary site of invasion and cause associated pathologies to the patient involved.

Examining areas of divergences connoting dissimilar trends: The current vaccine status indicates there is a vaccine for one of the forms of cancer (cervical cancer). Functional vaccines now exist for preventing cervical cancer, some of which are quadrivalent recombinant DNA technology based vaccines that target more than one specific HPV serotype, the typically pronounced sero-protective vaccines being those that target HPV 16 and 18 with broad protective capability against HPV serotypes 13, 45, 6, 11, 16 and 18. Some of these vaccines confer sero-protection for up to five years or more in the body system. However, for malaria, there is no confirmed functionally effective vaccine for malaria, the closest being RTS,S malaria vaccine (the first malaria vaccine to pass the phase 2 and 3 clinical trials for a malaria vaccine) which is now at the last phase of vaccine clinical trials.

There are specifically identified HPV viral proteins (oncogenic proteins such as E6 and E7 proteins (E6-7 oncogenic proteins) in relation to cervical cancer, whose activated status promote multiplicity of HPV pathogen (increases viral load). Then, some other proteins in activated status (non-oncogenic proteins) or downstream genes down-regulates HPV viral load and suppresses oncogenic E6-7 proteins expression involved in disease state; thereby supporting clearance of HPV load and with targets for design of immunotherapy against HPV associated cancer. As such, reduced E2 non-oncogenic protein load levels correlates with disease progression in HPV based cervical cancers. Observed specific roles of *Plasmodium* parasite proteins in disease progression has not been established nor clearly identified, nor such proteins involved in down-regulation of malaria disease progression been established as of current date. The major protein molecule related attributes that have been documented so far are those found in the host body. As such, elevated levels of host plasma proteins and chemo-molecules such as CRP C-reactive proteins, lipo-polysaccharide binding proteins, interleukins (such as IL-10), Interferon gamma, Tumor necrosis factor TNF, pro-inflammatory and inflammatory cytokine in-balance in the host system has been clearly associated with emergence of severity of malaria in humans and in animal models. However, some evidences have been recorded that associated *Plasmodium* gene diversity supports manifestation of clinical condition of observed severity of malaria disease.

Cancer appears to be more varied in terms of types compared to malaria. There are well over **200** different types of cancer, some of which have been associated with HPV pathogen, particularly cervical cancer, whereas there are just between 3 to 5 confirmed types of malaria based on species involved.

This comparative foray of both ailments is geared towards elucidating certain features in their disease progression courses, pathology and immunological characteristics, thereby contributing to body of knowledge in this area of science. Certain features in this can be identified and positively exploited in preventive and metabolic pathways that could be stage-specific for designing curative therapies that typify the concept of biomedical investigations from the bench side (biomedical) to the bedside (therapy), a tasking noble but useful mantra in therapeutic based translational medicine.