Fears of cancer survivors about relapse

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Abstract

When people are treated for cancer, it is common for them to fear it could come back, either in the same place or another part of the body. In the last decade, medical researchers have increasingly come to recognize the big breadth of these fears and to their possible serious consequences. The patients who are most afraid of cancer recurrence say they have a poorer quality of life, are more likely to develop depression and use more health services. They tend to have thoughts about their cancer returning with very little triggering as a sensation from some area of the body. All this may convince them that they have a new tumor.

Keywords

Cancer survivors, cancer relapse, metastasis, quality of life, depression, health services, new tumor

Introduction

Estimates vary about how fearful patients are about cancer recurrence, but it appears a serious concern. Several research show that about half of cancer survivors are between moderately and very worried about recurrence, and slightly fewer than one in ten are very worried. We also know, for example, that over 60% of breast cancer patients say they wish to speak to their specialist about their worries. The research confirms also another part of the puzzle: patients who have received chemotherapy tend to fear cancer recurrence more than those who only have a surgery. Research combined results of 40 studies over a number of years using a tried and tested statistical approach known as metaanalysis found the same thing to be true with radiotherapy.

Fears and uncertainty

It might be not surprising that patients receiving more extensive treatment are more fearful. Some commentators have previously argued that more varied and extensive treatments could signal to the patient that every possible weapon in the armoury has been used to eradicate the disease. This logic should therefore strengthen their belief that the cancer has been “cured”. But if this way of thinking seems confounded, there are indications that the correlation between cancer fears and receiving chemotherapy has been weakening over time. This may mean that in the future, the relationship won't be there anymore.

Maybe, the clinicians are explaining chemotherapy treatment better than they used to. It may be that they are providing greater reassurance or possibly that patients are coming round to the way of thinking of the other school of thought, increasingly believing that extra treatment over and above surgery is important to reduce the risks of recurrence. When it comes to radiotherapy, they found no equivalent decline in fears. It could be that patients see radiotherapy more as a risk factor for a new cancer than a “cure” compared to how they see chemotherapy.

Of the many different types of patients that have been investigated, breast cancer patients show the strongest correlation between chemotherapy and greater fears of recurrence. Psychologists are now focusing on how they can assist patients in making sense of the experience of diagnosis and treatment for this life-threatening disease. We know that once patients develop the fear of recurrence, it tends to endure and is difficult to reduce. It is therefore important to try and prevent these fears from taking hold. (Humphris, 2017)

Supplementary theory of tumors metastasizing

Scientists at the California NanoSystems Institute at UCLA have taken a major step toward confirming an unusual theory of how cancer cells metastasize. Their findings may lead to new strategies for keeping melanoma from spreading.

A commonly held theory about how cancer cells spreads is that

tumor cells break off from the primary tumor and travel through the blood stream to reach other organs, where they attach and grow into new tumors. Questions about that model have remained because circulating tumor cells in the blood sometimes have a short lifespan, and because of lack of knowledge about how the cells leave the bloodstream and attach to organs.

The research team was lead by Laurent Bentolila, director of UCLA's Advanced Light Microscopy/Spectroscopy lab, and included Clair Lugassy and Raymond Barnhill (France's Institut Curie). They theorized that, in addition to the prevailing theory about cancer spreads, tumor cells also could spread through the body by a mechanism called angiotropism. This means that they could travel along the outside of blood vessels, without entering the bloodstream.

Over the past decade, Lugassy and Barnhill gathered proof that tumor cells, especially those of the deadly skin cancer melanoma, creep along the outside of blood vessels like tiny spiders to spread cancer. They also found that the migrating cancer cells mimicked pericytes cells that line the capillary blood vessels, which prevented the cancer cells from being killed by the human immune system. The images showed the cells begin to grow as a primary tumor at the injection site. The researchers observed the green cells spreading from the tumor and migrating along the outer surfaces of the red-dyed blood vessels. Angiotropism has questioned the assumption that all metastatic tumor cells break off and flow through the bloodstream to spread disease. If tumor cells can spread by continuous migration along the surfaces of blood vessels and other anatomical structures such as nerves, they now have an escape route outside the bloodstream.

If tumor cells are found circulating in the bloodstream, doctors prescribe chemotherapy. But if the metastasizing cells are on the outside of the blood vessels, they escape exposure to the treatment and continue to spread cancer. These findings may enable researchers to seek new targets for deadly cancers such as glioma, glioblastoma, pancreatic cancer, prostate cancer and gynecological carcinomas. UV lights aids cancer cells that creep along the outside of blood vessels. (Bentolila et al., 2016)

Protein play key role in the spread of pancreatic cancer

Researchers from the University of Liverpool have found an explanation for how pancreatic cancer spreads to the liver. These findings potentially hold the key to stopping this disease from spreading.

Metastatic pancreatic ductal adenocarcinoma (PDAC) is a very aggressive type of pancreatic cancer that kills around 8 000 people every year in the UK 330,000 worldwide. Current treatments are not very effective, thus new treatment strategies are urgently needed.

The study, led by Dr. Michael Schmid from the University's Institute of Translational Medicine, focuses on the role of host connective tissue cells in the pancreas, or stromal cells, as the cancer cells spread to the liver (metastasis).

Over the last few years it has become clear that non-malignant stromal cells and the formation of a tumor microenvironment strongly influences the course of cancer progression and metastasis.

The study found that stromal partners are critical for efficient metastatic growth of pancreatic cancer cells, and identified a protein granulin as a key regulator of pancreatic cancer metastasis.

Study provides evidence that pancreatic cancer metastasis critically depends on the support of stromal derived factors such as granulin and periostin, and that targeting these stromal factors may improve the outcome of this devastating disease. The expression of inflammatory white blood cells, or monocytes, from granulin plays a key role in pancreatic cancer metastasis. These findings suggests the management or disruption of the secretion of this protein holds the key to stop cancer from spreading from the pancreas to the liver. (Schmid, 2016)

Antibiotics fundamentally alter the gut microbiome

People who take antibiotics for a long time are more likely to develop growths on the bowel which can be a precursor to cancer. Researchers say this adds to emerging evidence that the diversity of bugs in the gut could have role in the development of tumors. Their paper appears in the journal Gut.

Bowel polyps (small growths on the lining of bowel) are common, affecting 15%-20% of the UK population. In the most cases, they do not cause any symptoms and do not become cancerous, but some go on to develop into cancers if left untreated. In this study, researchers looked at data from 16,600 nurses who were taking part in a long-term US trial called the Nurses'Health Study (NHS). They found that nurses who had taken antibiotics for two months or more, between the ages of 20 and 39, were more likely to be diagnosed with particular types of bowel polyps (adenomas) in the later life, compared with people who had not taken long-term antibiotics in their 20s and 30s. Women who had taken antibiotics for two months or more in their 40s and 50s, were even more likely to be diagnosed with an adenoma decades later.

But the study does not look at how many polyps went on to become cancerous. This data are showing that in some part of cases may antibiotics lead to the development of cancer. We are also registering that the bacteria, which the drugs are deployed to threat, might play an important role.

This might have a crucial role in the development of bowel cancer, and the bugs that that require antibiotics may induce inflammation, which is known risk for the development of bowel cancer. These findings suggest the potential need to limit the use of antibiotics and sources of inflammation that drive tumor formation.

Dr. Sheema Cruickshank, an immunology expert at the University of Manchester, said anything that disturbs our gut bacteria, such as changes in diet, inflammation, or antibiotic use, could potentially have an impact on our health.

And what increases the probability of getting bowel cancer ? A diet high in red or processed meats and low in fibre can increase the risk, according to NHS Choices. These experts also say bowel cancer is more common in people who are overweight or obese and people who are inactive.

Drinking a lot of alcohol and smoking also increase the chance of getting cancer of the bowel.

People who have bowel cancer in the family can also be at higher

risk.

Information from the above research is very interesting because that builds on the other studies showing how the microbes in the bowel affect our health. (The Nurses' Health Study, 2017)

Tumors recur after immunotherapy

Immunotherapy is a new and highly promising form of treatment of cancer. In many patients, however, tumors recur after immunotherapy. The members of a research team from Max Dehlbrück Center for Molecular Medicine (MDC) in Berlin explain in the Journal of Experimental Medicine why some tumors recur and how this can be prevented. Their findings may aid the selection of suitable targets for immunotherapy.

One form of immunotherapy for cancer is T-cell receptor gene therapy. It involves removing T-cells from the blood and altering them in the test tube to enable them to target cancer cells. The cells are then re-introduced into the patient's bloodstream, where they find and destroy the tumor cells. In clinical trials, this procedure has proved effective for some types of cancer, but it has often been found that new tumors recurred after treatment.

The tumors are not recognized by the T-cells, explains biologist Dr. Ana Textor, the lead author of the study. To achieve treatment, Textor focused on a particular molecule on the cell surface, the epitope. Epitopes are at the heart of the immune response. They are produced inside the cell by specialized enzymes, which split and trim proteins into short fragments and send them to the cell surface as epitopes.

In cancer, proteins are pathologically altered through mutation, they too appear on the cell surface, in this case as neo-epitopes. A cell with a neo-epitope probably can be recognized by T-cells, which then destroy the cell.

The researchers found that when the tumor recurred, the epitopes were no longer present on the cell surface in sufficient quantity. This was because the epitopes in these cancer cells were no longer correctly trimmed enzymatically, in this case by the enzyme ERAAP. It was not being properly activated until the cell is stimulated by the signal molecule interferon (gamma). The tumor cells, were intensive to interferon and could no longer be recognized by the T-cells because they were no longer producing the epitope. By contrast, the epitopes on the cells of the successfully treated tumor did not require processing by ERAAP and were therefore also not dependent on stimulation by interferon. Epitopes that do not need processing by the enzyme ERAAP are therefore likely be a better choice for immunotherapy. (Textor et al., 2016)

Living drug revolutionize cancer treatment

At the end of July 2017 the FDA panel recommended that the agency approve a treatment that genetically alters a patient's immune cells to fight leukemia from within. This kind of "living drug" called gene therapy, would be the first of its kind on the market in the United States if the FDA approves it.

The treatment has to be created from scratch for each individual patient, using their own T-cells (white blood cells used by the immune system). T-cells are removed from patient's bloodstream, then genetically engineered to recognized and kill cancer. The

weaponized cells are then multiplied and re-introduced into the patient.

Novartis, the company seeking approval for the first therapy, presented data from an 18-month study in which 52 out of 63 patients with B-cell acute lymphoblastic leukemia went into remission.

The products that are closest to approval focus on blood cancers like leukemia and lymphoma, which represent about 80,000 of the 1.7 million cancer diagnoses in the United States each year. As Denise Grady, reporting for The New York Times: This has been utterly transformative in blood cancers, said Dr. Stephan Grupp, director of the cancer immunotherapy program at the Children's Hospital of Philadelphia and a professor of pediatrics at the University of Pennsylvania. If it can start to work in solid tumors, it will be utterly transformative for the whole fields.

Solid tumors are highly resistant to the kinds of altered T-cells that have proven effective against blood cancers. Although researchers are working on approaches for solid tumors, the challenge they present means that gene therapies may be further off for breast, ovarian, prostate, lung and pancreatic cancers.

But T-cell approaches aren't the only thing out there. Researchers at the University of Texas MD Anderson Cancer Center are harnessing the power of so-called natural killer cells, another kind of immune cell that can be genetically altered to attack cancer. Unlike T-cells, natural killer cells can be taken from cord blood donated at childbirth, and modified cells can be safely given to multiple patients. They have a kind of drug-activated "off-switch", which can be flipped in the event that the treatment causes unmanageable side effects. After promising studies in mice, the researchers have opened a study for patients with relapsed or treatment-resistant leukemia and lymphoma. (Kazmier, 2017)

Circulating tumor cells predict prostate cancer treatments

Anatomy and Cell Biology professor Dr. Alison Allan is examining circulating tumor cells (CTCs), cancer cells that detach from a primary tumor, travel through the bloodstream and invade other parts of the body, as a possible guide for individualized prostate cancer treatment success.

With prostate cancer, 90 percent of the deaths occur because of metastasis to the vital organs, which can disrupt the making of immune cells. In order to get to these distant sites from the prostate, these cells have to get into the bloodstream, using it like the highway. Think of this blood test as the traffic cop, trying to determine who are the good cells and bad cells, and keeping the bad ones of the road, Allan said. She is a Lawson Health Research Institute scientist at the London Regional cancer Program as a national leader in the study of CTCs. Her lab developed a unique blood test to track metastasis by measuring the number of CTCs in a patient's bloodstream. As a result, they now offers these blood tests for prostate, breast and colorectal cancers.

It is known that cancer constantly changes, usually in response to treatment, and will become resistant. It will change its molecular characteristics. There is a need not only for finding those cells and counting them, but actually isolating them down to the single cell level. Researchers want to determine the genomic characteristics of them to figure out what particular gene and protein are express-

ing, and whether they can use information to determine the best type of treatment.

The team will use an emerging technology to determine whether CTC analysis can be used to indicate which patients will respond to chemotherapy versus other targeted therapies. This could lead to more effective and personalized management of prostate cancer with improved patient outcomes. (Allan, 2017)

Conclusions

These findings suggest the management or disruption of the secretion of this protein holds the key to stop cancer from spreading from the pancreas to the liver.

See also the role of so called “protein ropes” role in the eating habits, for example in the Alzheimer disease, etc. (Refer to the “Agriculture”..., 2017)

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Proteins may be the key factors of several types of cancer. Probably it may be a new potential for the treatment if we can study a program targeting special role of proteins in generating cancers. Proteins could be the Evil of Cancer behind the scene, but until now is not generally recognized its destructive function in full manner. It's an another goal for future cancer research initiative.

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