Natural Component Use in Ovarian Cancer

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Abstract- Among all female reproductive cancers, ovarian cancer is the most common cause of mortality. The seventh most frequent cancer among women worldwide in 2018 was ovarian cancer. The many kinds of ovarian cancer exhibit a wide range of molecular and genetic abnormalities, as well as varying responses to treatments, which makes it difficult to develop a universal therapeutic approach. Additionally, ovarian cancer cells have a propensity to develop resistance to standard cancer treatments via a variety of ways. To find an effective cancer treatment, a number of components have been investigated, including cytokines, growth factors, proteases, adhesion molecules, coagulation factors, hormones, and apoptotic agents. According to research, phytochemicals offer strong potential anti-cancer capabilities against a variety of malignancies. One of the phytochemicals that is widely present in daily life is quercetin foods. Several forms of cancer, including breast, lung, nasopharyngeal, kidney, colorectal, prostate, pancreatic, and ovarian cancer, have been shown to be inhibited by quercetin, according to extensive research. There have been numerous in vitro and in vivo studies done to assess the cytotoxic potential of quercetin against ovarian cancer. Researchers believe quercetin might be a good candidate for ovarian cancer treatment because it is cytotoxic to cancer cells through a variety of mechanisms and does not harm healthy cells. It may also work well as an adjuvant agent when combined with other anti-cancer medications. As a result, we concentrated on the chemo-preventive and curative effects of quercetin for ovarian cancer in this review and outlined some of the most recent research into the potential molecular processes by which this natural substance inhibits this cancer.

INTRODUCTION

There is evidence of cancer found in ancient human remains and in medical literature since ancient times, dating back to the time of the pharaohs in ancient Egypt and the classical world. Although it is difficult to interpret the diagnosis of doctors who lived so many centuries ago, we can assume that many of their descriptions are related to cases of cancer. [1] Ovarian cancer (OC) is one of the deadliest gynecological cancers in the world, causing about 14,000 deaths in the US in 2020. Computed tomography (CT) is part of the standard pre-treatment evaluation of patients with OC to evaluate the spread of the disease. In the last few years, personalized and precision medicine has begun to pave the way for tailored and individualized treatment, based on the growing knowledge of the tumour microenvironment, at the microbiological and molecular levels. Radiomics is a recently established translational field of research that aims to make connections between quantitative information obtained from imaging studies and clinical data to support evidence-based clinical decision-making. [2] The genesis of ovariectomy for ovarian tumours was when Jane Todd Crawford became the first person to he underwent such an operation on Christmas Day 1809 in Danville, Kentucky. Surgeon Ephraim McDowell removed 22lbs of flesh under an oral opium "aesthetic" with a line of nurses holding her still during the 25-minute operation! Released in January 1810, she lived for another 32 years. The short operative time and her survival suggest that the tumour was either benign or borderline or, if malignant, confined to one ovary. Since then, surgery has been at the fore in the treatment of ovarian malignancies: medical or radiation oncologists are unlikely to receive a referral without at least a biopsy-proven diagnosis. At the beginning of the last century, it became clear that most ovarian cancers cannot be cured by surgery alone, and this conclusion remains valid to this day. Treatment would consist of laparotomy, biopsy with possibly an attempt at more extensive intervention, then radiotherapy. Despite improvements in surgical and radiotherapy techniques, the outcome for the patient was disappointing; the five-year survival rate improved from only 29 percent (1950–1959) to 32 percent (1965–1969). At the same time, there was an interest in postoperative chemotherapy. A palliative effect was indicated by a reduction in the incidence of recurrent ascites; moreover, expectations of the potential for a "cure" arose from an early study with

the alkylating agent melphalan in which 13 patients "had such an unusually good response that a laparotomy was performed to determine whether the inoperable tumor had become respectable to assess the need for further therapy. In each of the 13 patients, no tumor was found, and chemotherapy was discontinued'. At a later follow-up, only two patients had developed a recurrence. [3]

Cognitive Status of Ovarian Cancer

Ovarian cancer is the seventh most common malignancy and the fifth leading cause of death among female reproductive diseases. The number of diagnosed cases continues to increase with higher life expectancy, while it is wildly recognized.

Recognize Preventive measures or effective treatment have not yet kept pace. The disease is specifically diagnosed only at an advanced stage, which causes relatively high morbidity and mortality. Accordingly, symptoms progress insidiously like tumorigenesis, including pelvic and lower abdominal pain, vaginal bleeding during menstruation, irregular bleeding after menopause, bloating, and changes in urinary or bowel habits. In some cases, primary cancer cells invade adjacent tissue and spread to distant organs. Indeed, ovarian cancer cell metastasis in the peritoneal cavity moves for a long time without obvious symptoms, and the ways of spreading ovarian cancer include intracavitary implantation, hematogenous metastasis, and lymphatic metastasis. On the one hand, its threatening aggressiveness depends on the rapid spread of cancer cells to neighbouring tissues such as the peritoneum, tomentum and abdominal organs. On the other hand, the development of drug resistance to chemotherapeutic agents cannot be neglected. Histopathologic ally, nearly 90% of ovarian carcinoma originates from the ovarian surface epithelium (OSE), with the remainder classified as serous, mucinous, endometrioid, transitional, and clear cell carcinomas. It is a heterogeneous disease associated with inherited mutations in susceptibility genes such as p53 tumour suppressor gene, ERBB2 and PIK3CAOncogenes, etc. About 10% of ovarian cancers occur in women carrying BRCA1 or BRCA2 mutations, causing some ovarian cancers to cluster in families. In addition, various classical signalling pathways are involved in oncogenesis. For example, Wnt/β-catenin target genes mediate cancer initiation and progression. PI3K/AKT/mTOR plays a regulatory role in cell survival, growth and proliferation. The Notch signalling pathway is associated with cell proliferation, migration, cell stemness, and chemoresistance. Hyperactivation of Hedgehog signal transduction is also involved in the chemotherapy resistance phenotype, as overexpression of the transcription factor is associated with upregulation of ABCB1 and ABCG2 gene expression in ovarian cancer. [19]

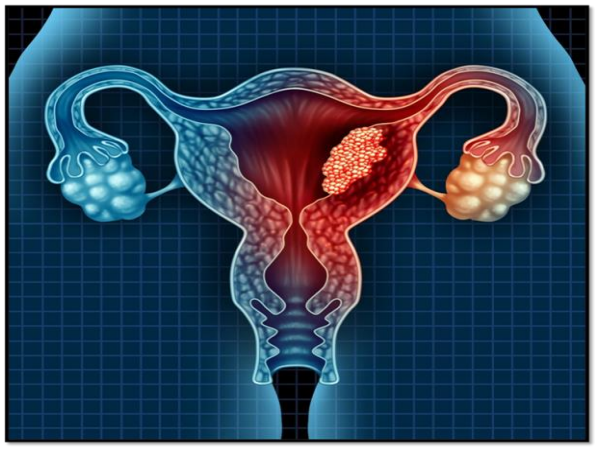

FIG NO-1 OVERIAN CANCER

Symptoms

When ovarian cancer first develops, it might not cause any noticeable symptoms. When ovarian cancer symptoms happen, they're usually attributed to other, more common conditions.

Signs and symptoms of ovarian cancer may include:

Abdominal bloating or swelling
Quickly feeling full when eating
Weight loss
Discomfort in the pelvic area
Fatigue
Back pain
Changes in bowel habits, such as constipation
A frequent need to urinate

When to see a doctor

Make an appointment with your doctor if you have any signs or symptoms that worry you.

Causes

It is not clear what causes ovarian cancer, although doctors have identified things that can increase the risk of the disease. Doctors know that ovarian cancer starts when cells in or near the ovaries develop changes (mutations) in their DNA. A cell's DNA contains instructions that tell the cell what to do. The changes

tell the cells to grow and multiply rapidly and form a mass (tumour) of cancer cells. Cancer cells continue to live when healthy cells die. They can invade nearby tissues and break away from the original tumour to spread (metastasize) to other parts of the body that can increase your risk of ovarian cancer include:

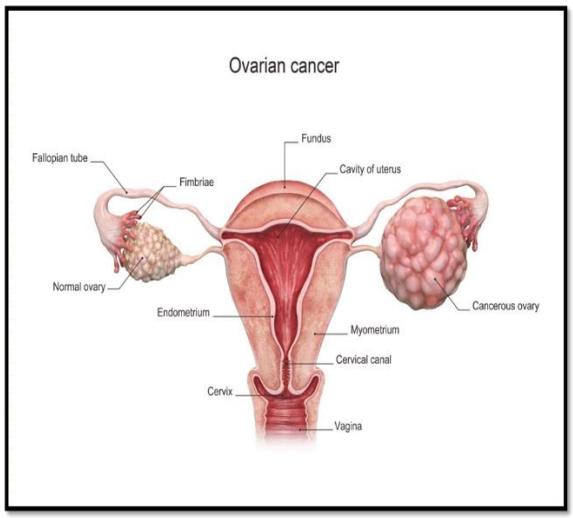

FIG NO-2 OVERIAN CANCER.

Factors:

Older age: The risk of ovarian cancer increases as you age. It's most often diagnosed in older adults.

Inherited gene changes: A small percentage of ovarian cancers are caused by changes in genes that yu inherit from your parents. Genes that increase the risk of ovarian cancer include BRCA1 and BRCA2. These genes also increase the risk of breast cancer.

Family history of ovarian cancer: If you have blood relatives who have been diagnosed with ovarian cancer, you may be at increased risk of the disease.

Being overweight or obese: Being overweight or obese increases the risk of ovarian cancer.

Postmenopausal hormone replacement therapy: Taking hormone replacement therapy to control menopause signs and symptoms may increase the risk of ovarian cancer.

Endometriosis: Endometriosis is and if you've never been pregnant, you may have an increased risk of ovarian cancer. often a painful disorder in which tissue like the tissue that lines the inside of your uterus grows outside your uterus.

Age when menstruation started and ended: Beginning menstruation at an early age or starting menopause at a later age, or both may increase the risk of ovarian cancer.

Types of ovarian cancer:

The type of cell where the cancer begins determines the type of ovarian cancer you have and helps your doctor determine which treatments are best for you. Ovarian cancer types include:

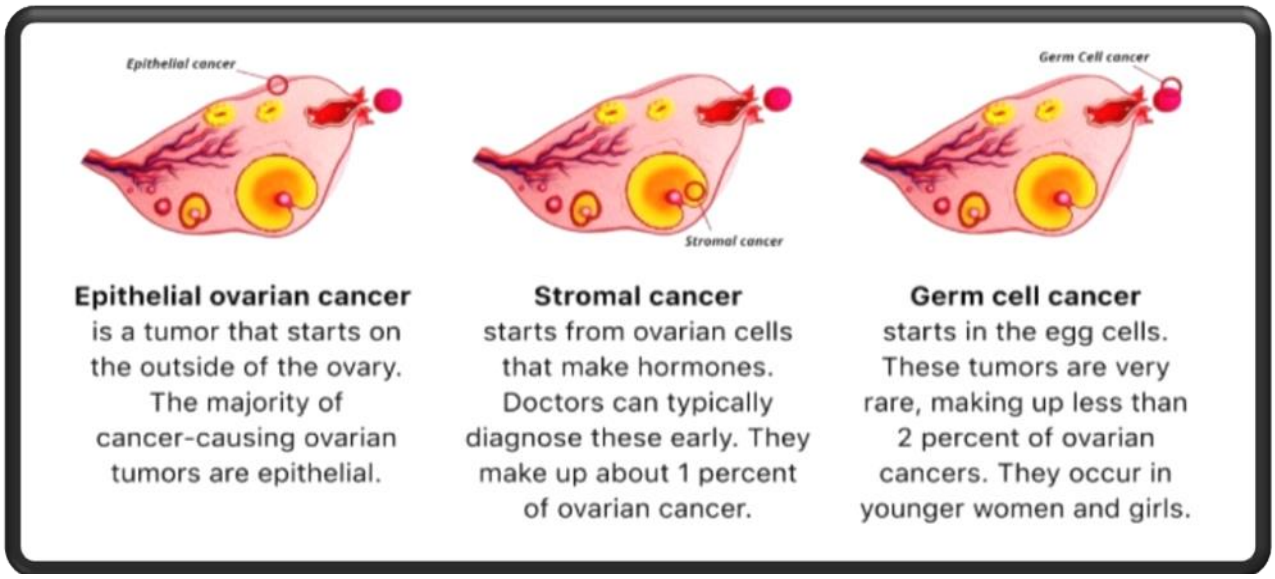

FIG NO-3 TYPES OF OVERIAN CANCER.

Epithelial ovarian cancer: This type is the most common. It includes several subtypes, including serous carcinoma and mucinous carcinoma.

Stromal tumors: These rare tumors are usually diagnosed at an earlier stage than other ovarian cancers.

Germ cell tumors: These rare ovarian cancers tend to occur at a younger age.

PATHOLOGY OF OVARIAN TUMORS

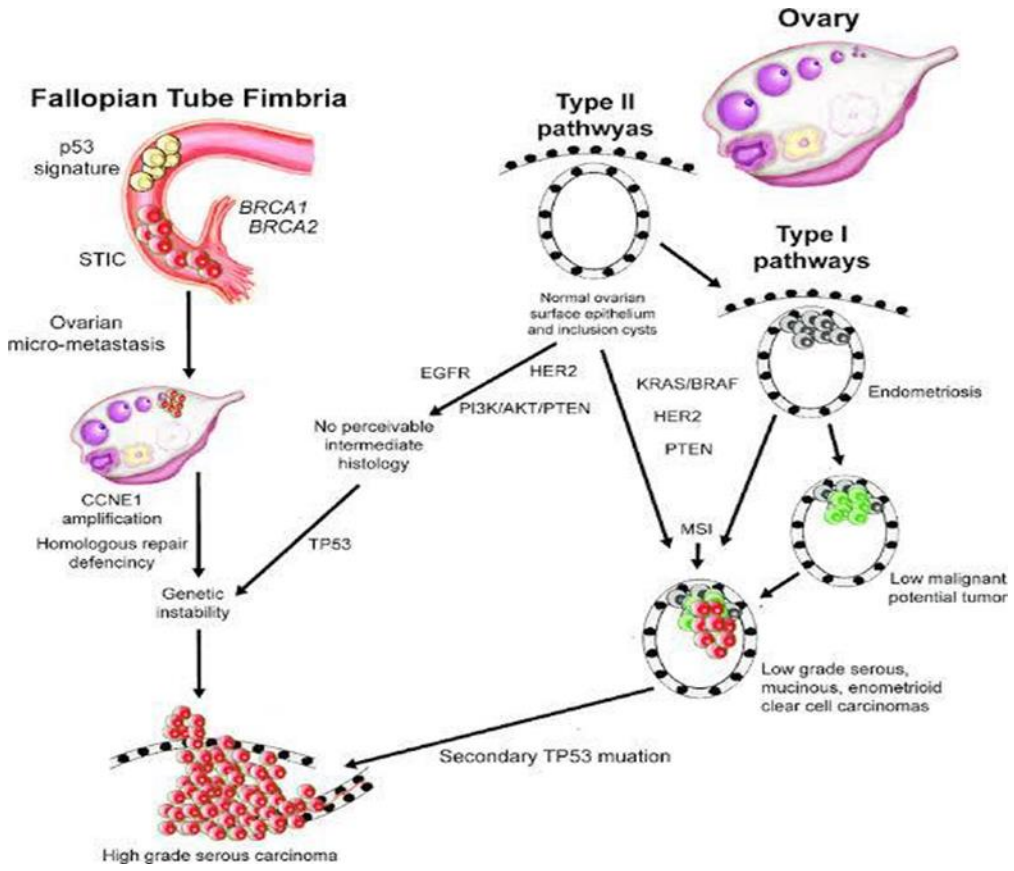

FIG NO -3 Pathology of ovarian tumour.

Most ovarian tumors can be classified into one of three main categories—superficial epithelial-stromal tumors, genital umbilical-stromal tumors, and germ cell tumors (Fig. 1)—according to the anatomic structures from which the tumors likely originate. Each category contains a number of subtypes. Combinations of different subtypes, either perfectly mixed or juxtaposed within a single tumor, are found with some frequency. Tumors that combine two or more subtypes are designated as mixed, with the contributing subtypes specified in the designation. By convention, tumor subtypes constituting 10% of the total tumor mass are ignored for classification purposes. The ovarian surface epithelium is histologically similar to the mesothelium, which is the epithelium that lines the interior of the pelvis and abdomen

Cavities. This similarity, as well as the close morphological similarity of ovarian epithelial-stromal tumors to some epithelial tumors arising elsewhere.

In the pelvis and abdomen, it can be explained by the common origin (i.e. primitive coelomic epithelium) of the ovarian surface epithelium and the mesothelium.The cord-stromal group includes tumours of mesenchymal and mesonephric origin. Some of these tumours, namely fibromas and thecomas, have a fi-Brous appearance, and some appear to be derived from granulosa cells or their testicular germ-cord counterparts, the Leydig and Sertoli cells. Ovarian germ cells are the origin of a number of tumours that are identical to testicular germ cell tumours. Germ cells that are stalled or lost during their migration between the yolk sac and the developing gonads can develop into germ cell tumours outside the gonads. The three main types of ovarian cancer and their subtypes are briefly discussed in this article, with particular attention to key aspects related to the registration and epidemiology of tumor treatment. A more comprehensive and detailed discussion of the pathology of ovarian tumors can be found in specialized publications8–11, which were used to prepare the current summary. [18]

- To prevent ovarian cancer:

Prevention

New information about how much BRCA1 and BRCA2 gene mutations increase the risk of ovarian cancer is helping women make practical decisions about prevention. For example, mathematical models have been developed to help estimate how many years of life the average woman with a BRCA mutation can gain by having her ovaries and fallopian tubes removed to prevent the development of cancer. Studies have shown that women with a BRCA gene mutation develop fallopian tube cancer more often than doctors previously thought. However, it is important to note that although doctors can predict the average outcome of a group of many women, it is still impossible to accurately predict the outcome for each individual woman. [19]

- Current Treatments for Ovarian Cancer:

Currently, clinical management favours debulking surgery along with platinum-based chemotherapy such as cyclophosphamide and doxorubicin hydrochloride. Standard chemotherapy regimens are taxanes including paclitaxel and docetaxel and platinum agents such as carboplatin and cisplatin.[20] In combination with paclitaxel and carboplatin, the humanized monoclonal antibody bevacizumab is administered to patients with advanced ovarian cancer. The harsh truth is that quite several patients who have received and responded to chemotherapy are prone to developing resistance to chemotherapy. Additionally, cumulative toxicities included but were not limited to, nephrotoxicity, neurotoxicity, ototoxicity with cisplatin, and myelosuppression with carboplatin. [21] Although early treatment is effective in most patients, advanced-stage patients are prone to relapse at a median of 16 months. In particular, genetic changes lead to increased repair of DNA lesions, impaired intracellular signal transduction, and metabolic inactivation of drugs. The success of complete remission relied on surgical skills accompanied by the gene signature of the tumour. [22] Olaparib as a poly ADP-ribose polymerase (PARP) inhibitor is recommended for patients who have been carriers of BRCA1 or BRCA2 germline mutations. [23] It is also used for relapsed and subsequent platinum-based chemotherapy. However, once a relapse occurs, the interval between subsequent treatments decreases steadily due to rapid tumour progression and resistance to chemotherapy. New options More than just traditional chemotherapy is needed to improve the quality of patient survival for systemic therapy. Among potential drug sources, botanicals are natural antioxidant components due to their long history in ethnopharmacology. Usually, natural products of plant origin are considered auxiliary nutritional supplements. Meanwhile, as part of initial treatment or maintenance therapy options, active ingredients from natural plants could improve multidrug sensitivity, further reduce the tumour and metastatic burden of ovarian cancer. In traditional Chinese medicine, triptolide is an antineoplastic agent derived from herbs that has been shown to selectively kill both p53 mutated and p53 wild-type ovarian cancer cells. [24]

- Differential Diagnosis:

✓ The differential diagnosis for ovarian cancer includes:

✓ Colon cancer
✓ Embryologic remnants
✓ Gastric adenocarcinoma
✓ Metastatic gastrointestinal carcinoma
✓ Ovarian torsion
✓ Peritoneal cyst
✓ Retroperitoneal mass
✓ Uterine fibroids
✓ Endometriosis
✓ Papillary adenocarcinoma
✓ Serous adenocarcinomas.

- History of natural cancer therapeutics

The use of plant substances - plants, herbs, mushrooms, seeds - as medicines predates historical history and represents the most significant direct predecessor of modern medicine. Recently, some of the most encouraging clinical evidence of the value of herbs in cancer treatment allows us to reconstruct the story of these plants and their possible use in these cases. Above all, it is important to realize that the modern concept of cancer is very different from the ancient one: the word cancer comes from the father of medicine, Hippocrates, who used the Greek word Karakinos to describe tumours, but the history of cancer actually begins much earlier. It is difficult to determine the diagnosis of cancer in ancient texts, only from the literary description. Progress in the understanding and treatment of cancer was slow and based on the development of pathological anatomy, beginning in the 18th century. The past 50 years have seen an explosion in our understanding of this most fundamental disease, with new discoveries emerging almost every week. For this reason, it is possible to find evidence of the relationship between plants and

cancer only recently. Some of the many plant compounds that have been shown to have positive effects in the treatment of cancer have a long history. For example, the green tea antioxidant EGCG (epigallocatechin-3-gallate) was recently shown to significantly slow the growth of breast cancer in female mice: its use is documented in ancient Japanese texts. Promising and selective anticancer effects have been observed in saffron (stigmata Crocus sativus L.) in vitro and in vivo, but not yet in clinical trials. The search for anti-cancer lead compounds has been the mainstream of marine chemistry. As a result, a number of natural marine products with unique mechanisms of action have been identified and recently entered into clinical trials. The use of Punica granatum juice, bark, and oil has also been shown to have anticancer activity, including interference with tumor cell proliferation, cell cycle, invasion, and angiogenesis. Modern scientific research has revealed that the wide range of dietary and medicinal functions of garlic can be attributed to sulfur compounds present in or generated from garlic, which can have a powerful anti-cancer effect. Myrrh is obtained from the dried resin of desert trees, Comephorid Myrrha and other species. Biblically speaking, it was chosen along with frankincense and gold as the gift of the three wise men to the newborn Christ. Celebrated for its anti-inflammatory and disinfectant properties, myrrh has historically been used for a variety of ailments such as stomach aches, indigestion, poor circulation, wound healing, certain skin conditions, and irregular menstrual cycles. What makes myrrh such an exciting player in the anti-cancer field is not just how well it kills cancer cells in general, but how well it kills those that are resistant to other anti-cancer drugs. It is thought to work by inactivating a protein called Bcl-2, a natural factor that is overproduced by cancer cells, particularly in breast and prostate. Although the myrrh compound does not appear to be as effective as other plant-based anticancer drugs — such as vincristine, vinblastine and paclitaxel — its advantage appears to be that it can damage cancer cells without harming healthy cells. other drugs do not. One of the most important plant compounds in the fight against cancer has been discovered in the bark and, in small amounts, in the needles of the relatively rare Pacific yew, Taxus brevifolia. [6]

- Histopathology

Ovarian cancer is a heterogeneous disease and contains several histological subtypes. EOCs account for more than 90% of primary ovarian tumors and can be classified into distinct morphologic categories: serous, mucinous, endometrioid, clear cell, transitional cell (Brenner tumors), mixed, and undifferentiated type (Table 2). Papillary serous histology accounts for 75% of ovarian cancers and its histological picture simulates the lining of the fallopian tube. High-grade, poorly differentiated tumors are the majority and are macroscopically indistinguishable from other epithelial tumors. This histological variant is often associated with concentric rings of calcification known as psammoma bodies.[7] Based on the observation of a high number of tubal intraepithelial changes (TICs) in high-risk women undergoing RRSO, it has been hypothesized that many apparent ovarian or primary peritoneal cancers may be of tubal origin. Recent studies have shown that up to 59% of high-grade pelvic (neuterine) serous carcinomas are associated with serous TICs. This is consistent with the hypothesis that the fallopian tube is the source of most of these tumors. There is increasing evidence that the pathogenesis of low-grade serous carcinomas and serous tumors of low malignant potential (LMP; discussed later in this section) involves similar genes and pathways and is distinct from that of high-grade serous carcinomas. Low-grade serous carcinomas and serous LMP tumours are characterized by a young age at the time of diagnosis and a prolonged natural history. The clinical behaviour of LMP tumours that recur as invasive low-grade serous carcinomas appears similar to newly diagnosed low-grade serous carcinomas. Other studies have shown that LMP tumours often coexist with low-grade serous carcinomas and, when they recur, often as low-grade serous carcinomas. These findings have led some to hypothesize that low-grade serous ovarian carcinomas represent the natural progression of an undetected LMP serous ovarian tumour. However, there is no definitive evidence that low-grade serous carcinomas always arise from LMP tumours, and whether these 2 entities represent a continuum of tumour progression remains unproven. Although there are ongoing studies specifically for low-grade ovarian carcinomas, low-grade and high-grade invasive serous ovarian tumours are currently treated similarly.[8]

Primary tumours tend to be large and unilateral. First-line platinum-based chemotherapy appears to be less

effective in mucinous compared with no mucinous EOC. Current studies are investigating whether chemotherapy regimens including agents (such as capecitabine, oxaliplatin, and bevacizumab) with activity against GI cancers may be more effective in mucinous ovarian cancers. Endometrioid tumours closely resemble the components of endometrial carcinoma and appear to have a better survival compared with serous adenocarcinoma, regardless of disease stage or response to platinum-based therapy.50 Patients with clear cell carcinoma of the ovary often have a clinical history of endometriosis. [9]

TABLE 1. Pathology of Epithelial Ovarian Tumors.

Histiology type	Analogous cell type
Serous (75%	Endosalpingeal
Mucinous (10%	Endocervica
Endometrioid (10%	Endometrial
Clear cel	Mullerian
Jingyuan Wu 1 Tuoyu Zhou 2 Yinxue Wang 3 Yanbiao Jiang 1 and Yiqing Wang 1,4,* Transitional cell (Brenner tumor)	Transitional
Squamous cell tumor	Squamous
Mixed epithelial	Mixed
Undifferentiated	Anaplastic
Unclassified	Mesothelioma, etc

- Natural anticancer compounds

From plant sources

Other examples of plant-derived compounds currently under investigation are flavopiridol, homoharringtonine, -lapachone, combretastatin A4. Flavopiridol is a synthetic flavone derived from the plant alkaloid rohitukine, which was isolated from the leaves and stems of Amoora rohituka and later from Dysoxylum binectariferum. Flavopiridol is a cyclin-dependent kinase inhibitor. The substance is currently in phase I–II clinical trials. Available evidence suggests encouraging response rates in various solid and hematological malignancies and diarrhea as a dose-limiting toxicity. Based on the in vitro synergy of flavopiridol with several conventional cytotoxic agents, combination clinical trials are underway to evaluate flavopiridol with paclitaxel or cisplatin against advanced solid tumours. Homoharringtonine is an alkaloid isolated from the Chinese tree Cephalotaxus harringtonia; is characterized by efficacy against various leukaemia's and is currently in phase II–III. The main mechanism of action of homoharringtonine is the inhibition of protein synthesis, blocking the progression of the cell cycle. -lapachone is a quinone obtained from the bark of the lapacho tree (Tabebuia avellanedae). [10]

- From microbial sources

New compounds derived from microorganisms include rapamycin and geldanamycin. Rapamycin (sirolimus) is a macrolide compound obtained from Streptomyces hygroscopicus. Rapamycin is a potent immunosuppressant and also has antifungal and antineoplastic properties. Rapamycin acts as a specific inhibitor of m-TOR (mammalian target of rapamycin), which is a downstream mediator of PI3K/Akt. It selectively blocks the activation of transcription, which leads to the growth and division of tumour cells. Geldanamycin, a rapamycin analogy, is a benzoquine ansamycin natural fermentation product from the same microbial source that binds to and inhibits the 90 kDa heat shock protein HSP90. In this way, it is also able to suppress the protein kinase activity of m-TOR. Both substances are currently in phase I–II studies. The tumor inhibitory properties of the bacterial enzyme lasparaginase were discovered more than 50 years ago, and since then l-asparaginases have been used in treatment of various lymphoproliferative disorders and lymphomas, especially acute lymphoblastic leukaemia, in combination with other anticancer agents, in children and adults. [11]

- From marine sources

Marine compounds that have reached clinical research are trabectedin (or ET-743) isolated from Ecteinascidia turbinata, bryostatin, a macrolide lactone isolated from a species of alga, Bugula neritina, kahalalide F, a cyclodepsipeptide toxin isolated from the mollusk Elysia rubefescenscens isolated from the Caribbean anemone, and didemnin a second-generation aplidine isolated from Aplidium albicans. More recently, other compounds such as squalamine isolated from the dog shark Squalus acanthias, LAF389, a synthetic analogue of bengamide B (a compound isolated from the Jaspis sponge from coral reefs near the Fiji Islands and Australia), and neovastat, a derivative of shark cartilage extract, have been developed into clinical studies. Most of these compounds have been recognized by the FDA and the EMEA as "orphan drugs" for the treatment of various neoplasms. Among the previously mentioned compounds, trabectedin has undergone the most

extensive clinical research. It has demonstrated clinical activity in a wide range of solid tumors and was granted marketing authorization by the EMEA in September 2007 for the treatment of soft tissue sarcoma after failure of standard chemotherapy. In addition, positive results from a randomized phase III trial comparing trabectedin with pegylated liposomal doxorubicin vs. with pegylated liposomal doxorubicin alone in patients with ovarian cancer. These agents are characterized by different pharmacological properties. Although the exact mechanism of action of trabectedin is still not clearly defined, it is essentially an agent interacting with DNA and transcription. This complex mechanism of action is due to the chemical structure of the drug consisting of three fused tetrahydroisoquinoline rings. Two of these bind covalently to the minor groove of DNA and the third protrudes from the minor groove and may directly interact with transcription factors (e.g. SP-1) Different and conflicting reports have been published on whether trabectedin is a substrate for P-gp bryostatin acts as modulator of protein kinase C (PKC) activity and enhances the effect of chemotherapeutic agents such as the PKC inhibitor paclitaxel. [19, 20]. LAF389 was studied in a phase I trial. Squalamine and neovastat are currently being evaluated in phase II and III trials [13–14]

- Chemo preventive compounds from natural sources

Chemoprevention is a promising antitumor approach that aims to reduce the morbidity and mortality of cancer by delaying the process of carcinogenesis. Curcumin is one of the most studied chemo preventive agents. It is a natural compound extracted from the rhizome of Curcuma longa L. that allows the suppression, retardation or inversion of carcinogenesis. Curcumin has also been shown to have antitumor activity in various in vitro tumour models (cell lines from solid tumours and leukaemia) as well as in tumour animal models. Its special toxicological profile (doses up to 8000 mg/day are still safe) has allowed the development of a large number of phase II studies. As a chemo preventive agent, curcumin is currently in phase II trials in patients with colorectal cancer. Another candidate chemo preventive agent is resveratrol, a polyphenol found in many plant species, including mulberries, peanuts, and grapes. [15-16]

TABLE 2. Risk Factors for the Development of Ovarian cancer[17]

INCREASED RISK	DECREASED RISK
Age	Multiparity
Nulliparity	Lactation
Early menarche or late menopause	Hysterectomy
Menopausal hormone replacement therapy	Tubal ligation
Endometriosis	Oral contraceptive use
BRCA1/2 mutatio	
Lynch syndrome	

Synthetic drugs used in ovarian cancer:
Gemcitabine, doxorubicin and bevacizumab are drugs used to treat ovarian cancer resistant to cisplatin and carboplatin. The use of high-dose chemotherapy will lead to complications due to side effects and may lead to discontinuation of the treatment plan. [26]

CONCLUSION

This study demonstrates ovarian cancer treatment in the future. It illustrates how natural remedies affect ovarian malignancies. In this overview, information about many natural medications is provided. It provides information on the signs and symptoms of ovarian cancer, prevention, contemporary ovarian cancer treatments, and various diagnoses. It offers details on substances that are naturally anticancer.

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