Breast Augmentation and Risk of Cancer [A Review]

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Abstract: In this modern and fashionable era, trends for cosmetic breast augmentation and uplifting have considerably increased which not only increase the aesthetic look, psychological satisfaction but also attract men. Women from as early as 300 BC up to date have used various methods to augment and uplift breast. In recent era, technological advances have made possible to achieve desired breast size, curve and uplifting. In most of these methods, various chemicals and tools are used which either directly or indirectly induced or promoted breast carcinogenesis. This review briefly discussed link between various breast augmentation procedures and risk of breast carcinogenesis.

Key words: Aesthetic - Augmentation - Carcinogenesis

INTRODUCTION

Women, for centuries have attempted to create the look of a voluptuous and full bosom through modification of clothes. Minoan women in 300 BC use primitive corsets and brassieres to raise the bosom to attract men [1] as large breast size represents a major deal of beauty. Large breast also remained a part of fashion as, models with lean, muscular body and large breast size are highly appreciated and adopted [1]. Now, for the sake of abrupt results, women underwent painful and disfiguring procedures in which such materials as ivory, glass, metal and rubber were implanted into their chests which causes serious medical problems. To achieve her idealized female image some women injected olive oil, petroleum and paraffin jelly which do not only cause medical complications but often horrific aesthetic results [1].

For several decades, prosthetic breast reconstruction and augmentation has enjoyed worldwide acceptance and has been considered to be safe [2]. But, recently due to the use of polyurethane sponges, carcinogenic concerns have emerged, as the break down product of polyurethane (toluene 2, 6- diisocyanatediamines and toluene 2, 4- diisocyanate) have been reported in the urine which can cause sarcoma in rats [1-2]. Further, it has also reported that, breast augmentation can cause lesions which can delay cancer diagnosis [3]. In 2001, more than 215,000 USA females achieved breast augmentation procedures and this number is continuously increasing. This is an underestimate of the actual number of breast augmentations performed annually, as increasing numbers of non-surgeon physicians are now performing cosmetic surgery [4]. Therefore, there has been concern not simply to breast augmentation which have resulted a rising in the occurrence of breast cancer but the breast cancers identification must be further highly developed [4-8]. The aim of this review was to briefly discuss various breast augmentation methods and its association with cancer.

Breast Size and Cancer: Breast size is usually not included on exhaustive lists of risk factors for breast cancer [9]. Breast size was generally estimated based on self-reported questionnaires using brassiere size (current or in early adulthood), for example using the classifications “infantile, small, average, or large” [10] or “small, medium or large” [11]. One study measured brassiere size based on chest circumference [12]. Other papers calculated breast size based on mammograms, with results validated by comparisons with tissue from mastectomy patients [13]. Brassiere use was assessed as a risk factor in one paper [14]. The authors
found that premenopausal women who did not wear brassieres had half the risk. This relationship was hypothesized to be due to breast size or other factors such as the thermal effects of brassiere use. In 2013, Williams studied a sample of 79,124 women and suggested that cup size was a strong predictor of breast cancer mortality, even when adjusted for BMI and other covariates [15]. Another recent study by Eriksson and colleagues [16] addressed the ongoing uncertainty regarding the link between breast size and risk of cancer. Noting the heritable nature of breast size, they identified genetic variants that affect both size and risk, using data from a consumer genetics company, supplemented by online questionnaire data on brassiere size. These results are encouraging for helping understand links between breast size and cancer risk and offer potential additional tools for screening.

Silicone Breast Implants: Silicone breast implants have been broadly used in the United States and all over the planet for more than 40 years. Huge bodies of scientific literature have been available dealing with a promising connection among silicone implants with cancer [17-22]. Overwhelming epidemiologic confirmation that women during implants are not on increased risk for primary breast cancer 23-27) but, several studies recommend an inferior rate of breast cancer in augmented women 28-32]. Apprehension concerning cancer threat following implantation of silicone procedure within the individual body contains information of sarcomas during silicone expose experimental animals as well as case information of breast plus further cancers of women by breast implants[33]. Presence of breast implant may obscure about 22 to 83% of breast tissues during mammography interfering visualization. Cancer in these augmented breasts can only be diagnosed in a more advance stage with a very poor prognosis [34, 35].

Estrogen: The links among breast cancer with estrogen have identified for more than 100 years. George beatson verified the mutual oophorectomy resulted during the diminution of breast cancer within premenopausal women [36]. Investigational facts strongly proposed that, estrogens have a role during the improvement as well as development of breast cancer [37]. The accurate mechanism by which estrogen induces breast cancer is not clear however, it is reported that estradiol, acting through estrogen receptor alpha (ERα), stimulates cell proliferation and initiates mutations arising from replicative errors occurring during pre-mitotic DNA synthesis. The promotional effects of E, then support the growth of cells harboring mutations. Over a period of time, sufficient numbers of mutations accumulate to induce neoplastic transformation. Laboratory and epidemiological data also suggest that non-receptor mediated mechanisms resulting from the genotoxic effects of estrogen metabolites are involved in breast cancer development [38-39]. Estrogens supports enhancement of mammary cancer within rodents and exerts both direct plus indirect proliferative effects on cultured breast-cancer cells from humans [37]. Direct tumor initiating effects might happen during the beginning of enzymes plus proteins concerned within nucleic acid synthesis as well as throughout the activation of oncogenes. Circumlocutory effects could happen during the motivation of prolactin secretion as well as the construction of growth factors, for example transforming growth factor α plus epidermal growth factor with non–growth-factor peptides for example, plasminogen activators. Tumor construction may also result from excessive hormonal stimulation of an organ in which normal growth and function are under endocrine control [40]. The reply of an organ to the proliferative effect of a hormone could be fruition from typical development to hyperplasia to neoplasia. During this model the threat of breast cancer might resolute through increasing contact of breast tissue to estrogen [41]. Differentiation in implement and dietary ingestion of assured nutrients may too influence contact to estrogen. Plants hold phytoestrogens which were structurally alike toward physiologic estrogens. Soybean is a rich resource of phytoestrogens in addition when ingested in moderately huge quantity, they contained mutually estrogen agonist as well as antagonist effects within humans plus animals [42]. Flaxseed is a rich dietary resource of equally mammalian lignans as well as α-linoleic acid which exert anti-estrogenic effects by mutually binding to the estrogen receptor plus reduce the formation of estrogen [43]. The occurrence of breast cancer is low in region wherever the intake of soy as well as flaxseed is soaring [43, 44]. Conversely it is unsure whether this opposite connection is a direct effect of phytoestrogen or flaxseed intake or whether it is an indicator of additional factors linked toward risk [45]. The connection among exogenous sex steroids plus the threat of breast cancer have been studied broadly. Primary epidemiologic study suggested little if any increase in the risk of breast cancer with the use of oral contraceptives [46]. The hormonal effect of oral contraceptives lying on the breast is complex. They often cause defensive an ovulation on the other hand; the combination of estrogen and progesterone may stimulate mitotic activity in breast tissue [47].
Prolactin: Prolactin, a polypeptide hormone is necessary for mammary gland improvement and lactation [48, 49]. Prolactin concerned into common breast maturity as well as lactation has put forward to be significant during the etiology of breast cancer. Experimental facts point out that, prolactin is able to encourage cell proliferation as well as increase cell motility and hold up tumor vascularization. It increase tumor growth rates, metastases as well as encourage both estrogen receptor + (ER) and ER- tumors in a transgenic mouse model in which ER+ tumors are unusual [50]. In 1984, Holtkamp et al. [51] reported 44% of patients with metastatic breast disease along with hyperprolactinemia. A number of cases of breast carcinoma in connection with prolactinoma have been reported [52]. In a separation of women at threat for familial breast cancer, basal serum PRL levels were extensively elevated [53, 54]. Similarly, circadian rhythm of PRL secretion from the pituitary differs between groups at high vs low threat of breast cancer [55] with no regular variation [56]. During a single study, hyperprolactinemia was establish toward significant pointer of adverse prognosis in node positive breast cancer patients equally when evaluated individually as well as in combination through steroid receptor status [57].

Macrolane Injection: Recently for breast augmentation, hyaluronic acid has been proposed as a new method [58]. Macrolane™ VRF20 and VRF30 are NASHA™ gels (non-animal stabilized hyaluronic acid) containing 98% water and approximately 2% of stabilized hyaluronic acid. Macrolane™ is biodegradable and the rate of degradation varies from individual to individual but, clinical studies have showed that complete degradation of hyaluronic acid takes 12-24 month [59, 60]. The use of Macrolane™ is very fast, requires local anesthesia, less invasive and low risk of allergic reactions and infections [61]. For breast augmentation usually 100-150 ml of Macrolane™ is required [62] which can last up to 12 month eliciting a breast tissue response [63]. The use of Macrolane™ is associated with several complications especially the formation of encapsulated lumps in breast tissues [60] and formation of visible and palpable nodules which makes the interpretation of a mammogram more difficult and could hinder the early diagnosis of breast cancer [62]. In cancer cell surface and the tumor of stroma, glycosaminoglycan is one of the main constituent. Further on cancer cell the hyaluronic acid receptor, CD44 is also located which can promote their migration and proliferation [62, 64]. Thus, high level of hyaluronic acid in breast with tumor cells is closely related with high tumor grade. In other words, even if Macrolane™ is not an initiating factor, it can possibly progress breast cancer. According to Crawford and Shrotria [65] Macrolane™ induces tumors, which delay breast cancer diagnosis.

Autologous Fat Grafting: For breast reconstruction and augmentation, the use of autologous fat grafting is increasing [66]. It was proposed in 1987 by the American Society of Plastic Surgeons (ASPS) that subsequently to autologous fat grafting, fat necrosis resulting calcification, compromises breast cancer detection [67]. However, in the recent years substantial improvement in the fat grafting technique and breast imaging modalities have been performed [68]. According to Delay et al. [69] if modern principles of fat transfers are used for lipo-modeling, it in no way hindered breast imaging. They studied 880 patients over ten years. In these patients after fat transfer, imaging was performed with MRI, mammography and ultrasonography. At the end of these ten years oncological follow-up study, the authors concluded that fat transfer did not reveal any increased risk of local recurrence or new cancer development. Similarly Zocchi and Zuliani [70], studied 181 patients who received 375 ml (average) of fat graft into one or both breast. All of these patients received both preoperative and serial postoperative ultrasound imaging and mammograms of breast. Only seven cases of micro-calcification and three cases of pseudo-cysts which resolved over six month simultaneously were observed. The authors concluded from their study that good communication between radiologists and surgeons can distinguish these artifacts from those associated with neoplasia.

CONCLUSIONS

To augment the breast, both physician and patient should select a safe method by accounting family history of breast cancer, hormonal profile and breast tissues sensitivity. Such type of women should also be informed about the possible complications. Further, there is a need for improving breast imaging technique in augmented breast for carcinogenesis.

REFERENCES


