Polyacrylamide hydrogel injection for breast augmentation: Another injectable failure

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Summary

Background: Increasing complications of polyacrylamide hydrogel (PAAG) augmentation mammoplasty, such as chronic persistent infection, have recently caught the attention of both the medical field and the general public.

Material/Methods: A total of 96 patients with severe chronic infection following PAAG augmentation mammoplasty were treated in the present study including 63 cases with infection confined to the breast and 33 with systemic infection. Endoscopy and surgery were performed to completely remove the materials and clear the infected tissues followed by drug-irrigation and vacuum-assisted closure for several days.

Results: In patients with severe infection there were large amounts of PAAG, fibers and infiltration of numerous neutrophils and macrophages. The infection-inducing materials were extensively dispersed in the mammary and subcutaneous tissues, pectoral fascia and intermuscular space. In addition, there was scattered distribution of PAAG materials in the armpit, chest wall and abdominal wall, which were mixed with necrotic tissues and surrounded by lymphocytes, giant cells, macrophages and other inflammatory cells, forming chronic granulomatous and fibrous lesions. Infection was controlled following surgical intervention. No residual infectious foci or recurrent infections were noted among these patients. Although the severe infection did not result in mastectomy, patients had breast atrophy and various degrees of deformation.

Conclusions: Chronic infection following PAAG augmentation mammoplasty usually causes systemic infection and other devastating adverse reactions. This study confirms PAAG augmentation mammoplasty is another failed attempt. More attention should be paid to the injection of large doses of liquid filler.

key words: polyacrylamide hydrogel • augmentation mammoplasty • chronic infection
**Background**

Polyacrylamide hydrogel (PAAG) injection was developed in 1980 as a minimally invasive technique for breast augmentation; it has been used in many women and is widely applied in more than 30 countries in Europe, Canada, USA, New Zealand, Australia, South America and Asia [1,2]. In Ukraine, Russia, China and Iran, as many as 300,000 women have received PAAG injection for augmentation mammoplasty. PAAG is applied as a colorless and transparent jelly-like soft-tissue filler. PAAG is a non-resorbable sterile watery gel consisting of approximately 2.5% cross-linked polyacrylamide and non-pyrogenic water. Aquamid (Conformite Europeene) is a representative PAAG. The hydrogel monomer of Aquamid at <0.0064 µg/ml is atoxic for humans and other animals and has been regarded as a non-cytotoxic, non-pyrogenic, non-biodegradable, non-carcinogenic and biocompatible material. However, the acrylamide monomer possesses neurotoxicity and teratogenicity [3]. Sometimes there are residual acrylamide monomers during the synthesis of PAAG, which may cause toxicity to nerve and muscle function [4]. Thus, the toxicity of this material is closely related to its purity and degradation. Polyacrylamide entering the circulation may cause embolism or even portal hypertension. Other adverse effects include edema, transient erythema, ecchymosis and pain [5].

PAAG was initially considered to be a compatible, atoxic, non-biodegradable and tolerable material with favorable safety due to absence of severe fibrosis, pain and capsule contracture [6–9]. Over time, increasing complications after PAAG injection have been reported, for which many women have suffered greatly. Incidence of complications following PAAG injection has been reported to be 6.74% [10]. Complications including hyaline degeneration and necrosis of muscle fibers as well as sclerosis and hyperplasia of surrounding muscle fibers are frequently observed. In addition, inflammation and granulomatous reaction are also reported as complications of PAAG injection [10]. Although PAAG has been prohibited from production and clinical application in plastic surgery, the complications following PAAG injection are not rare in clinical practice. The majority of women receiving this operation have local symptoms and adverse effects to different extents. The complete removal of this material is very difficult and may cause severe adverse effects [11–13].

In the last decade, some women undergoing PAAG injection have presented complications such as scleroma, ectopic breast tissue, plasticine-like changes in the breasts, pain, infection, ulceration, milk leakage, and formation of lactating mammary glands, among which post-operative infection is the most common and severe complication [4]. In the early stage the improper treatment of complications usually leads to new damage to tissues and causes bleeding and infection. In addition, infusion of normal saline into the scleroma may rupture the capsule, and squeezing the scleroma during the treatment may spread the material. Improper management of this condition frequently results in sepsis or even mastectomy [14]. Studies have shown that persistent local infection and systemic infection are often encountered in clinical practice. PAAG-induced infection in the breast is complex and difficult to treat. Delayed healing, recurrence and incomplete removal of this material are the main complications [15]. Therefore, it is imperative to develop formal and standard procedures for the treatment of complications following PAAG injection for augmentation mammoplasty, aiming to avoid iatrogenic complications.

The aim of the present study was to report the local and systemic symptoms and pathological findings after PAAG injection, attempting to find the cause of complications and the influence of the involved materials on human health. Our results may be beneficial for early identification of symptoms and signs of complications following PAAG injection and for early treatment of these complications to minimize their influence. In the present study, a total of 96 patients receiving PAAG injection with recurrent infections were recruited. Endoscopy and repeated surgical treatment were performed to completely remove the materials and infected tissues followed by drug irrigation and vacuum-assisted closure for several days. Infection was controlled within 1–8 weeks, achieving favorable outcome.

**Material and Methods**

**Clinical data and methods**

Patients (n=96) who were diagnosed with chronic infection following PAAG (Aquamid) injection were recruited from our department from January 2004 to December 2010. The local and systemic symptoms and the pathological findings were analyzed to investigate the influence of PAAG injection on human health. Informed consent was obtained before the study began.

**Inclusion criteria**

Patients with recurrent infection were treated in local hospitals by conservative intervention (no surgery or partly drainage) and the infection had still been uncontrolled for more than 2 months. The infection spread to the armpit, chest wall and/or abdominal wall, and sinus tract was found in several sites. Pus was observed in the infectious focus that were systemic. Patients were admitted to our hospital by the outpatient service. According to the extent of the infection, patients were divided into a local infection group and a systemic infection group.

**Chart review**

The clinical information of these patients was retrospectively reviewed. The operation record, anesthesia record, preoperative and postoperative notes and clinic record were reviewed and the age, sex, cause of infection, operation site, surgical methods, post-operative complications and corresponding interventions were also analyzed.

**Surgical procedures**

Pre-operative ultrasonography was performed to detect the infection extent, which was then marked. For patients with local infection, an incision (2–3 cm) was made at the lower edge of the areola or at the fistula orifice. The lower pole of the breast was separated along the gland surface. Then, the mammary glands were elevated and the retromammary space was exposed, showing a large number of PAAG. For
patients with systemic infection, an incision of different sizes was made at the low part of the fistula or pus-filled cavity.

A detacher was used to separate the individual cysts (under the guidance of an endoscope if necessary). The pus and the materials in the cysts were completely removed and the cyst wall was separated for removal of necrotic tissues. A specifically designed curette was applied to thoroughly clear the necrotic tissues. Under the premise of ensuring optimal shape and function, the visible PAAG was removed as much as possible. The cyst was irrigated with 3% hydrogen peroxide once and then with normal saline and antibiotics solution. Subsequently, a drainage tube was put into the lower part, followed by vacuum-assisted closure and pressure dressing. Post-operatively, antibiotics solution and normal saline were applied to irrigate the cysts intermittently in the presence of vacuum-assisted closure until the residual cavity was closed.

Pathological examination

During the operation, tissues (2×3×3 mm³) adjacent to the lesions were collected for pathological examination. For patients without complications within 12 months, biopsy of the breast (2×3×3 mm³) was performed. Tissues were embedded in paraffin, followed by sectioning, HE staining and observation under a light microscope.

Criteria for clinical evaluation

The efficacy of surgical intervention (material removal and infection control) was evaluated according to the patient’s subjective report and the wound healing and categorized into groups I, II, III and IV. In group I, non-effectiveness was defined, the infectious foci remained largely unchanged after surgical intervention, the PAAG was not completely removed, complications such as bleeding were observed in the treatment and the infection control required more than 2 months. In group II, the infectious foci were partially changed and PAAG partially removed; recurrent infection was noted post-operatively and required a second surgical intervention. In group III, the infectious foci were removed and the PAAG was macroscopically invisible; however, new infectious foci occurred post-operatively and required surgical intervention; recurrence of infection was not found. In group IV, the infectious foci were completely cleared and the PAAG was macroscopically invisible; the breast shape was acceptable, and visible defects were not found anywhere on the body; recurrent infection was absent.

Statistical analysis

Tests for treatment group differences in number of treatments and quantity of product were made using independent t tests. Groups were compared with Mann-Whitney U tests for improvements in observer-rated and investigator-rated infection control. The assessment scale scores and subject satisfaction ratings were recorded. Rater reliability for observer Surgery Efficient Assessment Scale ratings was evaluated using intraclass correlation.

RESULTS

Clinical information

A total of 96 women receiving PAAG injection for augmentation mammoplasty developed infection in unilateral breast (n=74) or bilateral breasts (n=22). The mean age of these women was 29 years (20–52 years). The time from PAAG injection to the occurrence of infection ranged from 3 months to 108 months. Patients developed local enlargement with bursting pain within 1 week to 1 month after occurrence of infection. In the local hospital, these patients were treated with antibiotics alone, and surgical intervention was not considered. Among these patients, 63 (local group) had only infection in the breasts characterized by progressive enlargement of the affected breast alternating with temporary improvement, dark red ecchymosis, blue or purple skin, bursting pain and increase of skin temperature. The tenderness was obvious and there was a sense of fluctuation on palpation. Oppressing the affected breast could cause cloudy liquid with unpleasant odor to flow out of the nipple. Several cystic nodules of various sizes could be found on palpation. Purulent fluid flowed out of the ulcer site, forming a sinus tract (Figure 1). The majority of patients
felt an increase of local skin temperature and fatigue. In the remaining 33 patients (systemic group), the infection spread to loose connective tissues and fat layer in the armpit, chest wall and abdominal wall along the subglandular space, resulting in systemic infection. In patients with severe infection, the infectious foci in the above tissues were connected with each other, forming a giant subcutaneous cavity (about 60×40 cm²). The infection compromised the immune function and impaired the wound healing. Thus, the infectious foci could not be immediately removed, resulting in delayed wound healing and chronic infection (Figure 2). The patients had poor health condition and felt weakness and fatigue. The symptoms related to fever were especially obvious.

On admission, ultrasonography of the affected breast showed the materials and infectious tissues located in the retromammary space and partially in the pectoralis major, and several cysts with different sizes were found in the breast and other infected sites. The structure of the affected breast was obscure and the internal echoes were enhanced (Figure 3A, B).

The infection-induced inflammatory symptoms were evident in the armpit, chest wall and abdominal wall, and the fat layer was severely damaged, forming a giant cavity with irregular borderline and involvement of the muscular layer (Figure 3C, D). MRI in certain patients revealed PAAG and the infectious foci under the chest and abdominal skin, in the breast tissues and retromammary space, under the pectoralis major fascia and in the pectoralis major and armpit. The infected tissues were mixed with excretions located in several spaces. The body temperature was slightly higher than or in the normal range. The inflammatory cells were normal.

**Findings in operation**

An incision was made in the affected breast or in the ulcerated skin, and light yellow or yellowish-white pus flowed out. The PAAG in the pus was roe-like and presented as milky-white granules. Cysts of various sizes were observed in the subcutaneous layer, glandular layer and muscular layer. The hyperplastic connective tissue composed the...
whole cyst wall. The material was mixed with the necrotic tissues. Some materials were adherent to the cyst wall, and the mammary glands and connective tissues were characterized by colloid degeneration (Figure 4A, F). Several infectious foci were located in the pectoralis major and formed sinus tracts in the upper outer quadrant and lower inner quadrant. After expanding the sinus tract, several cavities were observed in which a large amount of materials and pus were found. There were septums between 2 cavities. These changes obscured the structure and severely damaged the mammary glands (Figure 4D). In those with dispensation infection, the nodular infectious foci and cysts were found in the subglandular plane and the breast tissues. In addition, PAAG and necrotic tissues flowed into the armpit and abdominal wall, which led to the spread of infection in the loose connective tissues and fat layer, forming chronic infectious foci. The chronic inflammation stimulation leads to the formation of granuloma and chronic fibrosis, which were common in these patients (Figure 4B, E). The inflammation was obvious in the armpit, chest wall and abdominal wall. The fat tissues were damaged and absorbed, forming a giant cavity. The PAAG granules were also observed to be scattered. The materials were difficult to completely remove from the infected tissues. At the late stage, the dehydration promoted the capsulation of materials, with surrounding degenerated tissues forming scleroma similar to breast fibrosis (Figure 4C).

Pathological features

Under light microscope, the PAAG presented as indigo foreign bodies. In patients with severe infection, there were a large number of indigo foreign bodies mixed with neutrophils, macrophages and some fibers. The cells were hard to identify and almost destroyed (Figure 5A, B). The connective tissues were hyperplastic and fibrotic, forming the cyst wall. The PAAG was mixed with pus and necrotic tissues or even the cyst wall, and the connective tissues experienced porridge-like degeneration (Figure 5C). There were a variety of inflammatory cells surrounding the infectious foci, and the megakaryocytes, macrophages and fibrocytes formed foreign body granuloma characterized by a turbine-like structure (Figure 5E, F). The glandular tubes were involved and PAAG in the glandular tubes resulted in obstruction and oppression of glandular tubes and subsequent inflammatory degeneration (Figure 5D). In addition, infectious foci
were found in the breast tissues, under the skin, in the pectoral fascia and in the intramuscular space. The myocytes of pectoralis major were obscure and fused with each other (Figure 5H). In the armpit, chest wall and abdominal wall, PAAG was also dispersed and mixed with the pus and necrotic tissues surrounded by inflammatory cells including lymphocytes, giant cells and macrophages, forming chronic granuloma and fibrous lesions (Figure 5G, I).

**Therapeutic efficacy**

All these patients were regularly treated for 2~8 weeks, and the local and systemic infections were controlled and gradually resolved. Patients of the local group received surgical intervention once and the breast shape returned to nearly normal 2 weeks later (Figure 1E). Ultrasonography was carried out 3 months later. Only a small amount of materials were dispersed in the breast tissues and pectoralis major. The infectious space was absent and the infected tissues were completely cleared (Figure 6A).

Patients of the systemic group with infection in the chest and abdominal wall generally recovered within 8 weeks. On palpation, mass, cavity and fluctuation were not observed, and there were not excretions in the original sinus tract, nipple and incision. The pain resolved progressively. Post-operatively, the cavity was irrigated repeatedly and the excretions decreased 1~2 weeks later but the residual cavity was still present. Under laparoscopic guidance, the residual cavity was thoroughly irrigated and the degenerated tissues removed completely. Five to 6 weeks later, the cavity was irrigated again and the excretions decreased further and the residual cavity was still present. Under laparoscopic guidance, the cavity was irrigated thoroughly and the degenerated tissues were removed completely. Three months later, ultrasonography showed there were no PAAG in the breast tissues, no residual liquid accumulated in the lesions, and the infectious foci were absent. There were no cavities in the armpit, chest wall and abdominal wall (Figure 7).

**Follow-up**

Infection control was achieved in all patients. Residual infectious foci and recurrent infection were not observed. None of patients received mastectomy due to severe complications (Figure 8). However, the affected breasts were atrophic to different extents and the breast shape was unattractive in some patients (Figure 6C, D). In 1 case, the

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**Figure 6.** (A) Ultrasound showed absence of infectious foci and presence of residual small PAAG nodules; (B) Recovery of degenerated muscle but residual PAAG granules were found; (C, D) Breast atrophy following treatment.

**Figure 7.** Incidence of infection control following surgical intervention (control of local infection at 4 weeks after surgery was superior to that at 2 weeks ** P<0.01; control of local infection at 6 weeks was superior to that at 4 weeks * P<0.05. Control of systemic infection at 4 weeks was superior to that at 2 weeks † P<0.01; control of systemic infection at 6 weeks was superior to that at 4 weeks ‡ P<0.01).
severe infection resulted in ulceration in the areola and severely damaged the structure of the areola (Figure 4E). Six month after surgery, some patients received breast augmentation with breast implants and no complications, including infection, were found. One year after surgery, ultrasonography showed a few materials in the breast tissues and pectoralis major. However, the infection cavities were absent and the infectious foci and degenerated tissues had completely cleared (Figure 6B).

**DISCUSSION**

Congenital breast dysplasia and mastatrophy/small breast due to acquired causes significantly affect physical beauty in numerous women [16,17]. In the past century, many plastic surgeons and scientists in related fields made great efforts to develop atoxic materials to reconstruct a natural-looking breast [18–20]. Soft-tissue augmentation is a momentous procedure in aesthetic and reconstructive surgery. The demand for soft-tissue augmentation is growing due to increased interest in breast augmentation. In the quest for soft-tissue fillers, different biologic and nonbiologic materials have been introduced, but none have provided ideal results. Where there is exploration, there is failure. Inevitably, some women pay a heavy price for the breast augmentation due to the complications. Therefore, the prevention and treatment of complications following breast augmentation are of great importance. In as early as 1899, soft paraffin injection was introduced for nose and breast augmentation [18,20]. It was not until the 1950s that Vaseline injection was introduced for nose and breast augmentation. However, granuloma commonly developed within 6–8 years after augmentation, and has been a main complication of this technique [18,21]. In 1959, liquid silicone injection was introduced for breast augmentation. Similarly, inflammatory reactions (lumps, redness, pain, etc) would develop within 2 weeks to 2 years, and some patients even presented purulence, ulceration or tumor-like granuloma [22–28]. In the transplantion with free dermis-fat and fat-fascia tissues for breast augmentation due to flat chest or small breasts, the grafts are often partially absorbed and complications such as fibrous degeneration, tissue liquefaction and sclerosis frequently occur, which significantly limit the wide application of this technique [6,18,29]. These results suggest that simple, effective and minimally invasive techniques for breast augmentation using safe and durable materials are lacking [30,31]. The risk for complications of PAAG injection increases over time and the number of patients developing complication is still increasing. The effects of these complications are also impressive, which was demonstrated by a large number of patients with chronic infection in the present study. The infection following PAAG injection may be attributed to insufficient knowledge on the part of physicians of the physical characteristics of PAAG and the improper injection of PAAG. The breast represents a secondary sexual characteristic of women and is necessary for breast-feeding an infant. It is not isolated from the environment and remains in a semi-open state. Following PAAG injection, the PAAG can connect with the external environment through the mammary ducts, leading to opportunistic infection. Although PAAG is not degradable, this material is hydrophilic. Thus, it can absorb the body fluid and exudates, and inflammatory cells often infiltrate, forming gel-like materials rich in nutrients and resistant to flowing. Under this condition, the material can become a good medium for bacterial growth. PAAG is liquid and hydrophilic. After injection of PAAG and equal volume of normal saline, the materials may migrate under the influence of gravity. The PAAG entering the mammary glands may obstruct the glands, leading to mastitis and formation of potential infectious foci. In addition, the surgery may leave several cavities in the breast tissues. Although the tissues are tightly sutured, there are still cracks and tension. Thus, the materials may flow out, inevitably leading to wound non-healing or delayed wound healing, which finally results in infection [10–32]. In addition, breast-feeding and poor resisibility after augmentation mammoplasty may also attribute to the infection. The possible causes of infection may be the following. (1) Improper injection of materials or pressure applied during the operation that caused the material to flow along the potential space, which caused subsequent mastitis. (2) During pregnancy, the high estrogen level facilitates the proliferation of ductal epithelial cells, lumen expansion and edema of surrounding tissues. The progestational hormone promotes the atrophy of lobular tubular epithelial cells and interstitial edema, which aggravates the mastitis. (3) The materials may induce foreign body reaction, which may be another cause of chronic inflammation and adhesion. Thus, the mammary ducts are distorted, which deteriorates the milk stasis and facilitates the...
bacterial infection. (4) The improper breast-feeding may lead to nipple rhagades, which facilitates bacterial entry and subsequent infection. (5) In the present study, bacterial culture showed negative, which may be attributed to pre-operative treatment with antibiotics or the compromised immunity due to chronic infection [33–35].

In the present study, the proportion of patients with systemic infection was relatively high and this infection in all patients was secondary to local mammary infection. Thus, in the presence of local infection, improper management may result in delayed wound healing and subsequent chronic infection, which brings unwanted pain and damage to patients. In addition, PAAG is not biodegradable and thus cannot be completely removed. Moreover, PAAG is a hydrophilic liquid. Once PAAG absorbs water, it may promote the infection by bacteria and their reproduction. The watery PAAG mixed with pathogens may also flow under the influence of gravity, resulting in infection of surrounding tissues. Thus, the infection may invade the loose connective tissues and fat layer in the armpit, chest wall and abdominal wall along the submammary space, which is the main cause of systemic infection. Statistics show that the severe chronic infection following PAAG injection accounts for 2% of patients, which demonstrates the clinical importance of infection as a complication [1,2]. Moreover, some clinicians in primary care settings pay less attention to the complications of PAAG injection and then usually conduct improper treatment among these patients, which results in delayed wound healing and spread of infection or even more complex and severe complications.

Once infection occurs, the treatment is difficult. Conservative treatment is usually ineffective or requires a long course, and recurrence is frequently observed resulting in chronicity. Once infection is diagnosed, surgical intervention is preferred. The selection of surgical approaches depends on the location, size and extent of infection and the interrelation between infected tissues and surrounding tissues. The incision at inframammary fold and drainage at low site are often applied. In addition, the injected PAAG is scattered, foci usually have a capsule and septums are common between lesions; therefore, pre-operative ultrasonography is preferred for determining the location of infectious sites. The key to surgical procedures is to completely separate the infected tissues and cysts and thoroughly remove the materials and necrotic tissues, granulation tissues and fistula. Based on the hydrophilicity of PAAG, the wound is repeatedly irrigated with antibiotics in normal saline until the fluid in the drain is clear and PAAG granules and pus are not observed. These procedures are especially applicable for patients with a long history of augmentation mammoplasty because the capsule is thick and the fluid is difficult to extravasate. Our findings also indicated not all patients received PAAG injection in the retromammary space [1,18,36]. Thus, there were some nodules in the breast tissues and pectoralis major fascia, which are difficult to remove, and the removal of these nodules may cause damage to surrounding normal tissues. In addition, vacuum-assisted closure at the low part in combination with repeated irrigation with antibiotics in normal saline are also key steps in the surgical intervention. Apart from common bacterial culture, anaerobic and fungal culture is also needed. In the present study, the bacterial culture was mostly negative, which may be attributed to the early treatment with antibiotics [37]. If the procedures above can be performed properly, the infection may be effectively controlled in a short time.

Our results show that pathogens can cause infectious inflammation and that aseptic inflammation is also detrimental to tissues. The aseptic inflammation is usually characterized by bursting pain, tissue swelling, sometimes fever and increase of local skin temperature. At the early stage, the progression of infection is relatively slow and then is faster at the late stage. The affected breast usually presents redness, swelling, tenderness and sense of fluctuation. Of note, PAAG can also irritate the pectoralis major, resulting in unilateral ectopectoralis characterized by swelling and bursting pain of the affected breast when the upper arm exercises. However, few studies report this issue and more evidence is needed [34].

Although the breast following PAAG injection is soft and natural-looking at the early stage, the PAAG may form palpable scleroma when the water in the PAAG is absorbed. In addition, PAAG has no fixed form and can migrate into the armpit, chest wall and abdominal wall, which makes the removal of infected tissues difficult. At the late stage, the degenerated tissues are encapsulated, forming scattered scleroma that is difficult to differentiate from breast cancer [4,38]. Moreover, the relationship between PAAG injection and hyperplasia of mammary glands or tumor formation needs further study. The occurrence of local scleroma may be attributed to tissue damage or foreign body reaction [30,33,36]. Tissue damage may be caused by the relatively large needle used in the PAAG injection, which may cause damage to tissues to different extents, leading to local adhesion. Foreign body reaction occurs when, following injection of PAAG, a large number of macrophages surround the stimulants and form large phagosomes, which are also known as foreign body giant cells. The fusion between macrophages can form multinucleated giant cells forming a structure that can isolate the host tissues and foreign bodies. In addition, these cells can secrete pathogen-killing substances, resulting in erosion of normal tissues and necrosis and adhesion of surrounding tissues. The incidence of scleroma following PAAG injection is about 74%. The cause of chronic inflammation is the persistent presence of inflammatory cytokines and their damage to tissues. The chronic inflammation can be not only transformed from acute inflammation but also is caused by intracellular infection (tubercle bacillus or virus infection). These pathogens have low pathogenicity but can cause immune response. The continuous stimulation by undegradable but potentially toxic substances and the auto-immune response (rheumatoid arthritis) may also result in chronic inflammation. The PAAG is composed of undegradable materials and is potentially toxic. Thus, it can induce the foreign body reaction once it is injected, resulting in local scleroma and pain. In addition, systemic inflammation may also induce systemic response, such as abnormal increase of body temperature, which can be severe under certain conditions [39].

In recent years, the use of several imaging modalities, such as ultrasound and magnetic resonance imaging in addition to mammography in the diagnosis of breast disease, has increased [40]. Few data are available on the use of these techniques in the presence of augmentation mammoplasty, although both have been described. Ultrasound of the breast
is of proven value in the diagnosis of augmentation mammoplasty, and has several important indications, including the assessment of breast implant integrity [41]. In this report, all the patients received ultrasonography of the breast and the borderline of injected materials was marked as the basis of surgical intervention. CT and MRI may be the effective auxiliary examinations for these cases [42], but they are too expensive for most Chinese. Ultrasonography can meet the requirement for clinical differentiation. Each cyst in the mammary glands and the layers with infection must be carefully separated, and dead cavity is not allowed. For physicians with clinical experience, ultrasonography has been effectively applied to detect the infection-induced abscess and foreign body cyst, and the cost of ultrasonography is far lower than that of MRI and CT. However, the performance of ultrasonography in the detection of residual PAAG is not acceptable.

After surgical intervention, the majority of PAAG has been removed from the breast and a second ultrasonography is performed to detect the presence of infection/abscess or PAAG-induced cysts. Patients of the local group received surgical intervention once and the breast shape nearly returned to normal 2 weeks later, but patients of the systemic group with infection in the chest and abdominal wall generally recovered by 8 weeks. On palpation, no mass, cavity or fluctuation was observed, and there were no excretions in the original sinuses tract, nipple and incision. Our results definitely confirmed that the infection/abscess and PAAG-induced cysts were completely cleared by surgical intervention.

Conclusions

Taken together, the causes of infection following PAAG injection for augmentation mammoplasty are complex and their treatment is also troublesome. Generally, conservative anti-inflammatory treatment is ineffective and may result in chronicity. The chronic infection may also lead to systemic infection and/or adverse effects due to improper treatment or compromised immunity, which are usually severe. Once the infection is confirmed, surgical intervention is preferred. Taking appropriate measures to completely remove infected tissues and PAAG is crucial for infection control and to reduce the destructiveness. The tumor formation, aseptic inflammation, sclerosis and pain following PAAG injection for augmentation mammoplasty need further study. Augmentation mammoplasty with PAAG is again confirmed to be an injectable failure, and caution should be taken in the injection of liquid filler.

Disclosure

The authors have no financial interest in any of the products, devices, or drugs mentioned in this article.

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