How to Manage Complications in Breast Reconstruction

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Francesca De Lorenzi

Breast surgery is associated with a very low postoperative complication rate and even lower mortality rate. The reported morbidity rates range between 2% and 49% and are increased in cases of axillary surgery and immediate breast reconstruction [1, 2]. In case of reconstruction, the complication rate is still low and does not result in higher readmission rates compared with mastectomy only, therefore supporting the broadened access to reconstruction [3]. Furthermore, immediate reconstruction has been shown to be safe in terms of surveillance for recurrent cancer [3] and timing of systemic treatment delivery [4].

Even though complication rate is low, it represents an important indicator of the quality of surgical care, and, in an era of heightened patient awareness, healthcare providers are striving to identify and implement methods to reduce postoperative complications and ensure patient safety. An accurate preoperative planning is crucial to reduce surgeon's mistakes that can lead to complications. Preoperative evaluation includes drawings, measurements and pictures with the patient in the standing position and the assessment of comorbidities, smoking status and other potential risk factors for complications. These criteria lead surgeons to a tailored reconstructive option for every patient.

When complications occur, their quick identification and an appropriate management are mandatory to lower patient morbidity and improve final outcomes. Not all complications lead to surgical failure if adequately treated. Finally, a strict follow-up of patients with surgical complications is recommended, including an effective communication to patients and a good relationship, necessary to avoid legal litigations.

In this chapter we focus on the main complications after reconstruction and their management. Unsatisfactory results due to surgical mistakes or changing local situations, as volume and shape asymmetries, symmastia, dislocations of the inframammary fold and double-bubble deformities are not investigated.

41.1 Bleeding

It is the most common cause of reoperation in the early postoperative period, both in case of mastectomy and reconstruction or oncoplastic procedures, varying from 0.4% to 1.9% of the patients and generally within the first 4 days from the initial procedure [1].

Although in the vast majority of cases early postoperative bleeding requires surgical revision and haematoma drainage, in few cases a conservative approach may be sufficient, including compressive bandage and single intravenous administration of antifibrinolytic drug (tranexamic acid is the most used medication to prevent fibrinolysis). When fibrinolysis exceeds coagulation, unwanted surgical bleeding may occur despite adequate haemostasis. Because of uncertainty about the effect of tranexamic acid, particularly on vascular occlusive events, it is not recommended for routine use during most surgical procedures [5]. On the contrary, topical application of dilute tranexamic acid at the site of the wound reduces bleeding (Figs. 41.1, 41.2, 41.3) [6].

In case of implant reconstruction, haematoma formation enhances the risk of capsular contracture; therefore, surgical revision may be justified even in case of non-massive bleeding.

Patients with a bleeding disorder are at higher risk of return to operative room for haemorrhagic event, even though the vast majority of them can undergo surgery safely without complications. Balancing the timing of stopping or resuming anticoagulants, risk of embolic or thrombotic events and surgery can be challenging at times and often requires a multidisciplinary approach.

Late haematomas are also described both in case of implant reconstruction [7, 8] and flap donor site [9], presenting several months/years after the reconstructive procedure. Patients with implant present breast swelling and asymmetry generally associated to pain and no bleeding disorders. Many of these

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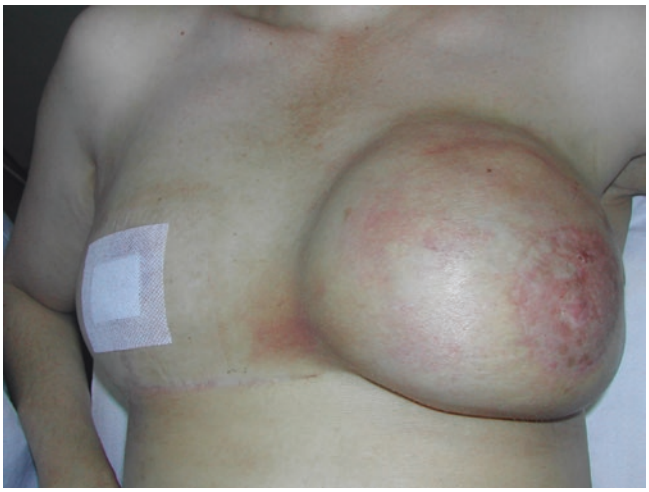

Fig. 41.1 Late haematoma of the left breast presenting 4 months after left reconstruction with implant. The patient described left breast swelling and progressive expansion. It required surgical revision

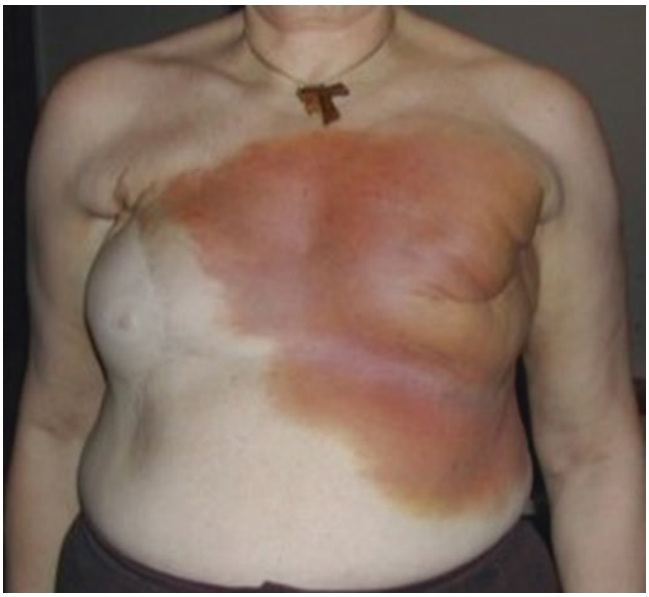

Fig. 41.2 Late haematoma of the left breast, no breast swelling is present, not requiring surgical revision

cases do not have a definitive mechanism of injury or develop symptoms immediately after the triggering event. Sudden enlargement onset or progressive expansions are both described. Ultrasound is an appropriate and cost-effective tool for differentiating between fluid collection and haematoma. Moreover, percutaneous ultrasound-guided needle drainage of fluid collection can confirm fluid nature, and the aspirate can be sent to pathology to rule out malignancy or infection. However, definitive treatment of late haematomas involves surgical drainage and capsulectomy and implant change (implant reconstructions) or sac excision (flap donor sites).

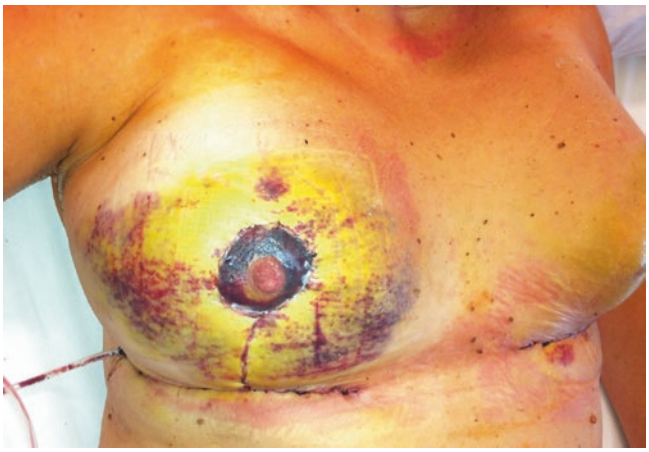

Fig. 41.3 Haematoma of the right breast after oncoplastic surgery conservatively managed

41.2 Surgical Site Infection

Surgical site infections at the donor or mastectomy site are the predominant cause of postoperative morbidity following mastectomy and immediate reconstruction [10]. Significant independent risk factors for infection were identified as BMI greater than or equal to 25 kg/m^2, chronic alcohol use, American Society of Anaesthesiologists classification of 3 to 5, flap failure in autologous or hybrid cases and operative time greater than 6 h [11]. The overall incidence rate of surgical site infection is 3.53% according to the US National Surgical Quality Improvement Program database (years 2005–2009). Patients within the dataset were divided in three groups according to the method of reconstruction: autologous procedures, prosthetic and hybrid type. Adjusting for confounding factors, there is no statistical difference in rates of surgical site infection among the three methods of reconstruction [12]. However, the gravity of surgical site infection is different among the three groups. In implant-based reconstructions, there is a risk of device loss and need for intravenous antibiotics. It generally requires reoperation with either removal of the implant or removal and replacement. Surgical site infection in autologous reconstructions typically does not result in loss of the flap but may result in deformity, the need for prolonged dressing changes and/or later reoperation (Fig. 41.4).

Focusing on infected implants, there is strong evidence that previous radiation therapy confers a significant risk of implant infection and suggestive evidence that simultaneous lymph node dissection increases the risk [13].

In the past, common practice was the immediate removal of the infected breast prostheses. However, the more recent literature has explored options for device salvage [14, 15, 16], changing surgical dogma that dictated foreign body removal in instances of infection. Methods for salvaging an

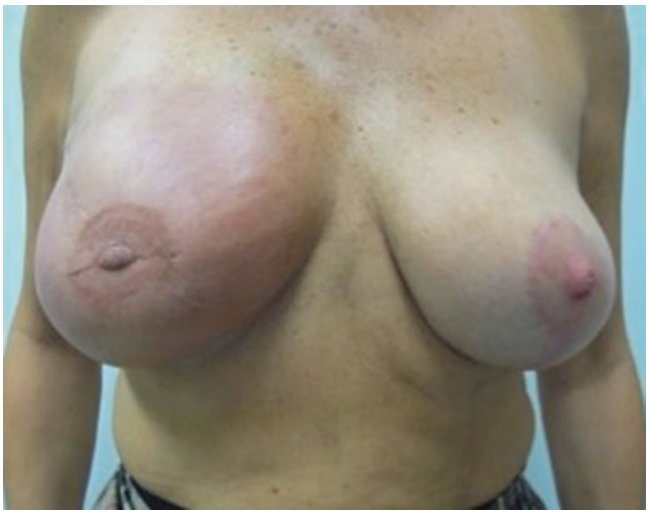

Fig. 41.4 Infection of the right breast after implant-based reconstruction

infected device have included systemic antibiotics combined with either conservative wound drainage or antibiotic lavage, capsulotomy/capsulectomy and device exchange, capsule curettage and continuous antibiotic irrigation. Treatment strategy is based on the response of the infection to initial antibiotic therapy and on the availability of soft tissue coverage (possible association to threatened or actual implant exposure). The severity of infection is an important factor in predicting the outcome of attempted salvage. The nature of infection may influence the ultimate outcome as well [17]. Organisms such as common skin flora may be treated with a high success rate, but organisms such as *Pseudomonas* species or Gram-negative rods may not be easily treated, and device removal is more likely indicated. Finally, if overwhelming localized infection or systemic sign of infection persists, further salvage attempts should be abandoned.

Finally, a further management strategy successfully indicated in case of mild infection and exposed implant is prosthesis explantation and immediate autologous reconstruction [18].

41.3 Necrosis and Wound Dehiscence

41.3.1 Managing Skin and Fat Necrosis After Oncoplastic Procedures

Glandular necrosis is the most challenging complication of oncoplastic procedures. Aggressive glandular undermining from both the skin and pectoralis muscle (dual-plane undermining) can lead to glandular necrosis in fatty breasts. Its incidence varies up to 13.4% [19, 20] in the literature. Imaging evaluation after oncoplastic surgery revealed fat necrosis in 18% of the cases on clinical examination, in 15% with ultrasound and 7% confirmed on pathology [21]. Non-healing wounds are recorded in 8.6% of the patients undergoing oncoplastic surgery [22]. These rates can be considerably reduced, incorporating the evaluation of breast density into the decision-making process [23]. In fact breast density predicts the fatty composition of the breast and determines the ability to perform extensive breast undermining and reshaping without complications. Breast density can be classified into four categories based on the Breast Imaging Reporting and Data System (BIRADS): fatty (1), scattered fibroglandular (2), heterogeneously dense (3) or extremely dense breast tissue (4) [24]. Low-density breast tissue with a major fatty composition (BIRADS 1/2) has a higher risk of fat necrosis after extensive undermining. Other risk factors for fat necrosis are diabetes, smoking habitus and previous irradiation of the breast.

Areas of fat necrosis can become infected and cause wound dehiscence resulting in postoperative treatment delay. They are usually managed conservatively, with daily dressing and antibiotic therapy. Surgical debridement is sometimes necessary to accelerate the healing process.

Fat necrosis can lead to scar retraction and deformities in the long term, therefore requiring surgical correction of sequelae.

41.3.2 Managing Flap Necrosis

It is due to an insufficient blood supply or drainage of the flap. Different mechanisms are responsible in case of pedicled or microvascular flaps, and the final result is the loss of part or the whole flap.

In case of pedicled transfer, flap necrosis is generally associated to peripheral venous congestion rather than arterial ischaemia. Important flap loss (greater than 25% of the transfer) is exceptional, and it may be related to preoperative risky factors as pre-existing scarring, smoking, obese and diabetic patients or thrombotic disorders. Massive abdominal flap necrosis can be also related to technical errors during the flap harvesting (traction of the pedicle) or insetting (twisting or torsion of the pedicle). Moderate flap loss (between 5% and 25%) occurs more frequently, and it is often related to venous congestion that will be the cause of necrosis. In both situations early surgery is recommended as soon as the limits of cutaneous venous congestion are well defined (generally on second postop day) and before thrombosis spreads to a larger portion of the flap. These situations generally require also late revisional surgery to correct the sequelae of the necrosis and eventual asymmetries with the contralateral breast (Fig. 41.5).

In case of minimal skin necrosis of pedicled flaps (less than 5% of the transfer), reoperations are not necessary, and the wound spontaneously evolves. Postoperative care is simple and generally managed by patients themselves.

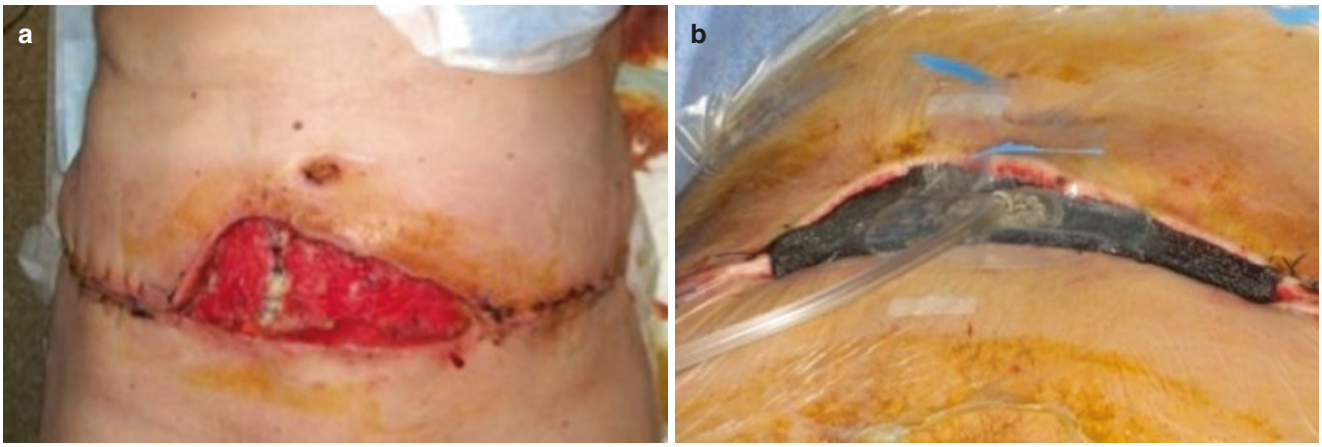

Fig. 41.5 Necrosis of the abdominal wall (**a**) after pedicled TRAM breast reconstruction. After debridement primary closure is not possible and negative-pressure wound therapy is a good solution (**b**)

In case of microvascular transfers, thrombotic complications are the major barrier to successful reconstruction, and they may be responsible of whole flap loss. For this reason, those patients candidate to microvascular transfers should be questioned about hypercoagulable history at the first consultation (family history of abnormal blood clotting, thrombosis in unusual sites, idiopathic or recurrent blood clots, a history of miscarriages, stroke at a young age). Unfortunately, thrombophilias remain a preoperatively silent and frequently undetected physiologic status against microvascular tissue transfer [30].

Routine postoperative care generally includes aspirin daily administration for coronary thrombosis risk reduction and potential enhancement of vascular patency as well as venous thromboembolism prophylaxis with subcutaneous heparin and sequential compression devices [31]. Microsurgical exploration is necessary for thrombotic events in about 7% of the cases [31]. It is due to arterial insufficiency (white and cold flaps, without turgor or Doppler signal) or venous insufficiency (blue and cold flaps, with fast capillary refill). Overall microvascular salvage rate is about 89% with a combination of intra-arterial, subcutaneous/intramuscular and intravenous tissue plasminogen activator, microvascular explorations and therapeutic multiagent anticoagulation/antiplatelet therapy.

41.3.3 Managing Mastectomy Flap Necrosis

It is a significant clinical morbidity after skin sparing and nipple and areola sparing mastectomies, related to the mastectomy itself and not to the reconstructive procedure. The incidence of native mastectomy skin necrosis after breast reconstruction ranges from 6% to 30% in retrospective series [25]. It is significantly higher in patients with higher mastectomy weight and body mass index, which correlates with breast size and subsequent mastectomy weight and in cases of diabetes. It is significantly more frequent in smokers, regardless of the type of reconstruction [26, 27]. Therefore, smokers undergoing reconstruction should be strongly urged to stop smoking at least 3 weeks before their surgery. Mastectomy incision type may influence its incidence [28].

Skin flap necrosis can be defined as mild (no intervention needed), moderate (requiring at least office debridement) or severe (surgical debridement needed, implant loss or healing not complete at 8 weeks) [29]. A not healed wound by 8 weeks postoperatively indicates a severe degree of ischaemia or wound-healing problems, which may be associated with infection and increased risk of dehiscence and can potentially delay adjuvant chemotherapy or radiotherapy (Figs. 41.6, 41.7, 41.8).

A different management is recommended in case of autologous or implant reconstructions. The benefit of autologous reconstruction is the possibility of healing by secondary intention with a well-vascularized flap at the base of the wound. This is in contrast to implant-based reconstruction where mastectomy skin loss increases the risk of implant exposure, necessitating earlier and more aggressive intervention with debridement and closure.

41.3.4 Managing Necrosis at the Donor Site

Necrosis of the anterior abdominal wall is observed typically in the infraumbilical region, which occasionally involves the umbilicus as well, and it is more frequent in smokers and obese patients [26, 32]. In fact, abdominal flaps are widely undermined and depend on a random blood supply unlike the axial vascular source of TRAM and DIEP flaps.

Similarly, in case of latissimus dorsi reconstruction, marginal necrosis of the dorsal wound may occur especially in case of closure with tension (extended latissimus dorsi flap).

According to different degrees and the extent of necrosis, wound care treatment modalities include debridement, wet-

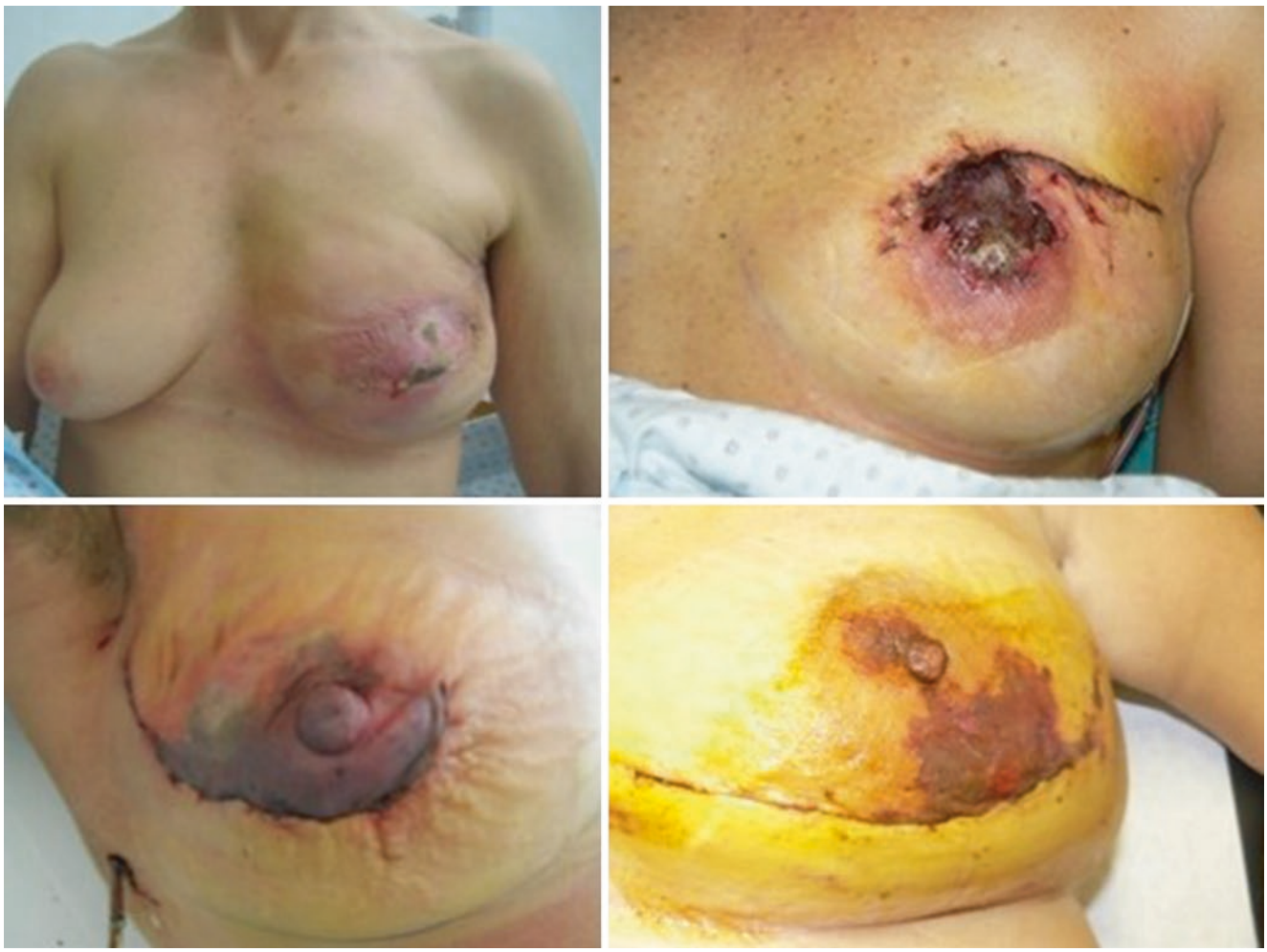

Fig. 41.6 Different degrees of mastectomy flap necrosis

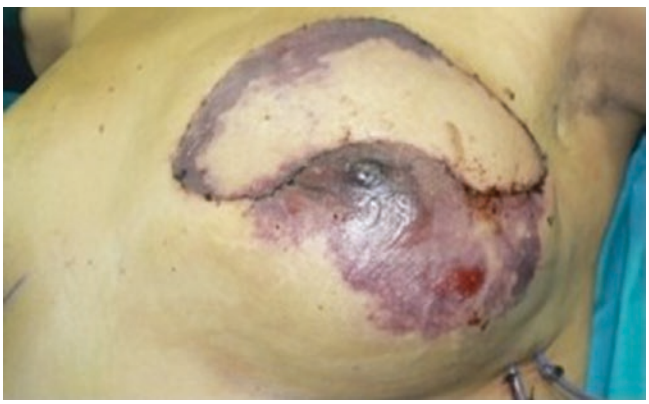

Fig. 41.7 Mastectomy skin flap necrosis and partial flap necrosis after left reconstruction with pedicled TRAM. Revisional surgery required

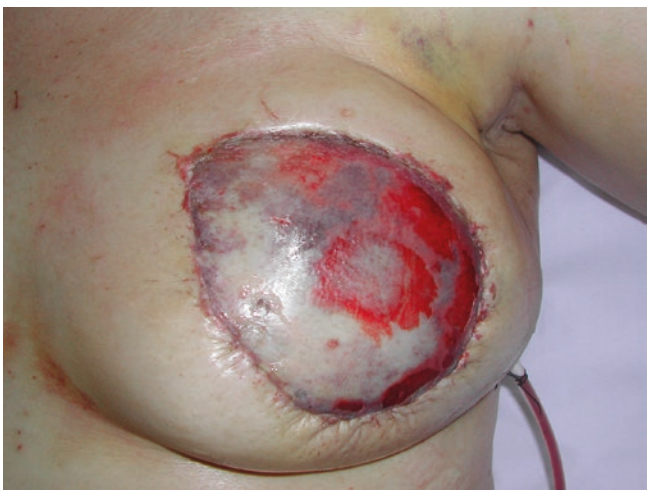

Fig. 41.8 Total flap necrosis after left reconstruction with microvascular transfer. Early microsurgical exploration failed

to-dry dressing changes and negative-pressure wound therapy. In case of massive necrosis, when primary closure is not possible after debridement, negative-pressure wound therapy may be an appropriate solution. The system generates negative pressure resulting in approximation of the wound edges, aspiration of infectious debris and exudates, reduction of oedema, increase in blood flow, promotion of granulation tissue generation and preservation of a wound-friendly moist environment [33].

In case of long-term chronic abdominal wound conservative treated, the risk of acute infection increases, and it may require the removal of the underlying abdominal mesh if present. If not, a chronic skin fistula may occur. If germs affect the mesh located behind the anterior rectus fascia, superficial wound debridement, even combined with adequate antibiotic therapy, is not adequate. The removal of the contaminated mesh is mandatory, which can weaken the abdominal wall.

In any case of healing problems at the donor site, revisional surgery may be necessary to correct scarring and deformities.

41.3.5 Pyoderma Gangrenosum

It is an inflammatory neutrophilic dermatosis characterized by painful, sterile ulcerations, bullae or pustules [34]. The aetiology is unclear, but it is thought to involve autoimmune dysfunction with dysregulation of the innate immune response. It is often associated with other autoimmune diseases as inflammatory bowel disease, rheumatoid arthritis, hematologic dyscrasias and connective tissue diseases. The so-called postoperative pyoderma gangrenosum is defined as the pathergic development of pathognomonic lesions in the surgical incision [35]. The typical presentation is an initial erythema of the recent surgical site, and afterwards the wound may dehisce or develop small punctate ulcerations that eventually coalesce, often associated with fever and leukocytosis. A wound infection is often diagnosed and antibiotics are initiated, as disproportional rapid wound breakdown is the typical presentation of necrotizing fasciitis. Even with negative wound cultures, the clinical presentation of postsurgical pyoderma gangrenosum still seems to favour the diagnosis of wound infection. Unfortunately, this misdiagnosis tends to result in prolonged courses of ineffectual antibiotics and surgical debridement that exacerbate and accelerate the problem by perpetuating the pathergic response in skin that was yet unaffected. Biopsies tend to show non-specific neutrophilic inflammation (Fig. 41.9) [36].

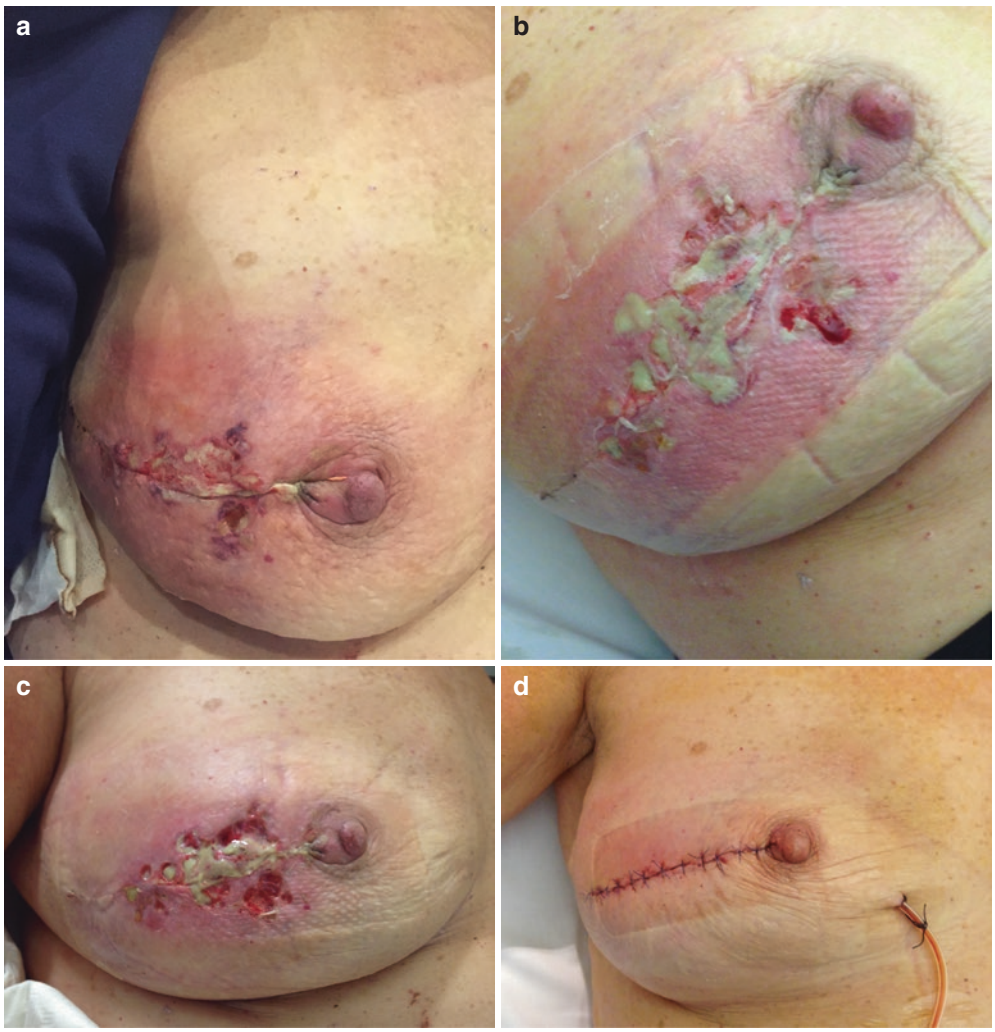

Fig. 41.9 Initial erythema and small punctate ulceration at the surgical site 6 days after right mastectomy and immediate reconstruction with implant (**a**). A wound infection is often diagnosed and antibiotics are initiated. The ulcerations coalesce (**b**—8 days postoperatively and **c**—9 days postoperatively). After systemic corticosteroids therapy, surgical debridement was necessary and successful (**d**—13 days postoperatively)

Awareness of postoperative pyoderma gangrenosum can help early diagnosis and an appropriate management to decrease patient morbidity. Systemic corticosteroids and immunomodulation agents are the first-line therapies; surgical wound debridement and reconstruction (skin grafts) are often necessary to expedite the healing process only if appropriate medical therapy has already initiated [37]. Postoperative pyoderma gangrenosum is a diagnosis of exclusion but should be considered in the differential diagnosis of postoperative wound dehiscence and infection.

41.4 Seroma Formation

Seroma is defined as a fluid collection that can occur both in case of flap reconstruction (at the donor site) and implant reconstruction (around the prostheses).

41.4.1 Managing Seromas at the Donor Site in Flap Reconstruction

Donor site seroma is the most common complication after latissimus dorsi reconstruction reported in the current literature with rates varying from 6% to 95% [38]. Postoperative seroma occurs in a lower percentage of breast reconstruction with abdominal flaps (2–13.5%) [39]. Seroma formation inevitably complicates any extensive surgical dissection and disruption of tissue planes that results in a dead space.

The consequences of developing a donor-site seroma are additional visits to the outpatient clinic for percutaneous aspiration as well as an increased risk of infection.

Several techniques have been described to prevent seroma formation. They minimize the "pocket" created at the donor site. Fibrin glue and quilting sutures are both performed for this purpose [40]. The principle of quilting is to promote flap apposition that facilitates healing. Finally, specially designed supportive garments may reduce seroma formation by applying external pressure on the donor site without jeopardizing the donor vessels.

41.4.2 Managing Seromas Around Breast Implants

Fluid collection around implants is a common event in the immediate postoperative period after drain removal. In case of small amount of serum, it could be asymptomatic, and the excess fluid spontaneously reabsorbs. In case of moderate or large seromas, breast swelling occurs, and percutaneous puncture and drainage are necessary. Ultrasound-guided drainage represents another solution.

The occurrence of late seromas, developing at least 12 months after the most recent breast implant surgery, is a rare event, reported in 0.6% of anatomical silicone form-stable implants [41]. It ranges from 0.4% in primary augmentation patients to 0.9% in reconstruction revision subjects. Late seromas appear as clinically symptomatic breast swelling. They are arousing increased interest in both cosmetic and reconstructive surgery since recent reports describe a possible rare connection between breast implants and anaplastic large cell lymphoma (ALCL), as these tumours often present as late seromas [42, 43, 44, 45].

Late seromas are often related to some sort of trauma and shearing forces between the capsule and textured implants (mechanical theory) [46] or low grade, subclinical infections (e.g. mycobacterium or biofilm—infectious aetiology) [47, 48]. In the vast majority of cases, they appear to be idiopathic, without a clear evidence of infection or malignancy [49].

There are a variety of recently described methods to manage late seromas [50]. Early acquisition of seroma fluid is recommended to rule out infection and malignancy with microbiology and cytology evaluation. The surgeon must decide whether to proceed with percutaneous (ultrasound-guided drainage) versus open therapeutic drainage of the fluid collection. If the decision is surgical drainage, the capsule needs to be inspected to determine whether local biopsy or total capsulectomy is necessary. Implant replacement also needs to be considered. A standard oncological evaluation is recommended to detect associated palpable or radiological masses in the breast or capsule or in the axilla. In fact, although the most common presenting sign of implant-related ALCL is late seroma, in some cases it presents as a mass adherent to the capsule, with or without associated fluid [45].

In our experience, the first approach is percutaneous puncture with culture and cytology. If no suspicious mass is present and fluid drainage resolves the problem, nothing further needs to be done. On the other hand, in case of recurrent seromas, the more definitive and reliable approach appears to be the surgical drainage associated to implant change and possible capsulectomy. In fact, implant-related ALCL tumour cells are usually found both in the fluid and the capsule, but occasionally they are found only in the capsule. In these cases, capsulectomy is essential for diagnosis [45].

41.5 Flap Reconstructions: Donor Side Morbidity

41.5.1 Abdominal Hernia and Bulging, Abdominal Asymmetries

After pedicled TRAM flap reconstruction, parietal complication may occur due to rectus muscle and fascia harvest and relaxation of the fascial suture. Even in cases of DIEP flaps,

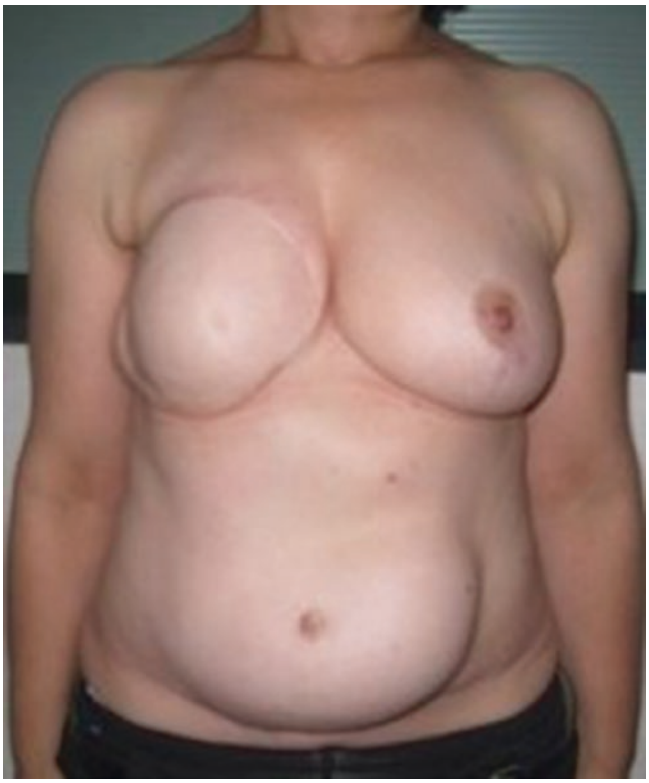

Fig. 41.10 Abdominal hernia after right breast reconstruction with a pedicled TRAM flap

sparing the muscle and the fascia, intramuscular perforator dissection may lead to morbidity [51]. Chang et al. [52] reported an overall hernia/bulge incidence of 5.9% after abdominal-based free flap reconstruction, varying from 3.3 to 9.9% depending on the type of free flap and if these were unilateral or bilateral reconstructions.

Weakness of the abdominal fascia is responsible of laxity and bulging in the infraumbilical region. It can be corrected by plication of the fascia (re-tension) and reinforcement with a preaponeurotic mesh (Fig. 41.10).

Most troublesome are abdominal hernias, which can be localized in the epigastric region (pivot point of the pedicled transfer) or below the umbilical region (anatomical fascia weakness below the arcuate line). They should be treated as if they are symptomatic. A reinforcing mesh is generally used.

41.5.2 Shoulder Function

Latissimus dorsi muscle primarily contributes to shoulder adduction, extension and internal rotation. There is a general consensus that after latissimus dorsi rotation or removal, the actions of synergistic muscles of the shoulder joint compensate for the missing muscle when it comes to the mobility of the shoulder and to carrying out daily activities [53, 54, 55]. Conversely, there are several reports of decreased shoulder strength, but the severity of this loss varies [56].

41.6 Implant Exposure and Extrusion

It is due to insufficient soft tissue or muscular coverage of the implant, and many reasons might be responsible: thin mastectomy flaps or necrosis of the mastectomy flaps and/or deficit in the muscular pocket or wound dehiscence.

It occurs in 0.25–8.3% of implant-based breast reconstructions [57]. In case of actual exposure, the assumption is made that contamination or mild infection is present even though there may be no clinical evidence of infection. Therefore, all exposed devices are treated accordingly. Salvage attempts and decision-making process is based on the severity of local infection and on the availability of soft tissue coverage. Thus, initially the patient is covered with antibiotics, the device is removed, a capsulectomy is performed, the pocket is curetted, a new device is placed and closure is performed with local or distant tissue [17]. If sufficient local tissue is present, local tissue rearrangement may be adequate. If local tissue is not adequate, a distant flap should be used for coverage. If a flap has been used in the initial procedure before implant exposure and this distant tissue is unavailable for a salvage procedure, the implant should be removed in favour of a delayed reinsertion (Fig. 41.11).

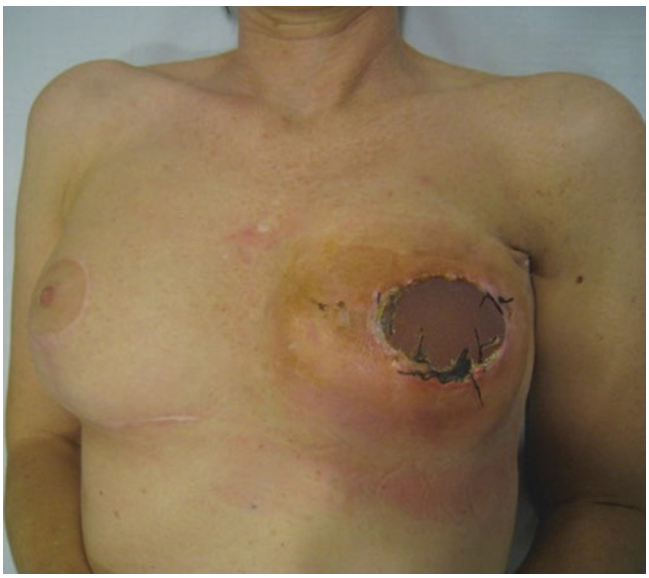

Fig. 41.11 Left implant exposure and extrusion after necrosis of the mastectomy flaps and wound dehiscence

41.7 Implant Rupture

It is defined as a gap in implant envelope leading to silicone gel or saline diffusion outside the implant itself. Saline prostheses are almost abandoned nowadays; in case of rupture, the implant deflates, and it is clinically evident. Conversely, the majority of implants on the market are silicone ones composed of a textured silicone elastomer shell and filled with cohesive silicone gel. Cohesive gel is formed by increasing the number of cross-links between gel molecules, with results in form-stable implant less likely to fold or collapse.

Rupture occurs as a result of biochemical degradation of silicone, physical trauma to the elastomer at the time of implantation or as a result of mechanical injuries during mammograms, closed capsulotomies or accidents. Intracapsular rupture is defined by the presence of silicone outside the implant shell and within the intact fibrous capsule. Extracapsular rupture is defined by the presence of silicone into surrounding tissues and lymph nodes.

The incidence of implant rupture widely varies in the literature [58, 59], and its prevalence increases over time. It depends on the type and generation of implants, different detection methods, mean implant life span and different follow-up period. In 2015, 10-year results from the Natrelle 410 anatomical form-stable silicone implants have been published. The overall rupture rate (suspected and confirmed) in those patients who underwent bi-annual MRI to screen for silent implant rupture is 9.7% of implants at a 10-year follow-up [41]. Rupture rates are even lower using Mentor MemoryShape implants, but results at a 10-year follow-up are not yet available, and patients have been screened with MRI at 8 years [60, 61, 62].

The majority of silicone implant ruptures are asymptomatic and are detected during routine follow-up ultrasounds. In case of suspicious images with ultrasound, MRI is recommended. MRI is the most accurate technique to evaluate implants integrity. Its sensitivity for rupture is between 80% and 90% and its specificity between 90% and 98% [63].

Explantation is the gold standard, with the removal of the capsule to include eventual silicone residuals.

41.8 Capsular Contracture

The pathologic process of capsular contracture manifests from excessive peri-implant fibrosis or capsular formation beyond the normal state. Clinically, it can manifest as pain, hardening of the breast and distortion of the reconstructed breast. The rate and risk of capsular contracture remain controversial. It increases over time [64], and it is reported as baker III and IV in 14.5% of the patients at 10 years after reconstruction with the Natrelle 410 anatomical form-stable silicone breast implant [41]. A meta-analysis (level Ib evidence) demonstrated that textured implant shells clearly reduce the risk of contracture for subglandular implants [65]. Review of randomized controlled trials found a significantly increased risk of contracture for smooth subglandular implants [66]. Its incidence is significantly higher in cases of irradiated beasts [67].

Despite advances in breast implant surgery, it is the most frequent cause for implant revision, and capsulotomy and capsulectomy represent the standard treatments.

The exact cause for capsular contracture has yet to be determined. Several theories on the pathomechanism and origin of capsular contracture have been suggested. These theories underpin the pivotal role of an inflammatory reaction, which leads to induction of fibrosis and shrinking of the capsule. A non-specific inflammatory process directed against silicone and periprosthetic bacterial contamination is considered to be the primary pathogenic mechanism leading to excessive local inflammation [68]. Therefore, treatment strategies also include the use of targeted inhibitory molecules as the leukotriene inhibitor zafirlukast to affect capsule formation [69, 70]. These drugs are also used in the prophylaxis of contracture. Implant insertion with a funnel may also decrease capsular contracture reducing skin contact and potential contamination of the implant pocket with skin flora [71].

Finally, more recently, acellular dermal matrices have been proposed to decrease the incidence of capsular contracture in implant-based reconstruction. They are hypothesized to block the inflammatory process suspected to be the trigger in the pathogenesis of capsular contracture, and several animal models support this statement [72]. In the clinical setting, the higher level of evidence (level III) is represented by a study comparing capsular contracture rates in a cohort of women who had acellular matrix-assisted implant reconstruction against a cohort who underwent standard implant reconstruction [73]. This study concluded that acellular dermal matrix is associated with less capsular contracture.

41.9 Systemic Complications

41.9.1 Pneumothorax

Pneumothorax remains a serious although rare complication of breast reconstruction. Most cases in the plastic surgery literature relate to breast augmentation, but they are described also in case of tissue expander/prosthesis placement [74] and autologous reconstruction [75]. The American National Incidence of pneumothorax after expander reconstruction is 0.55% per patient [76].

When pneumothorax occurs, it may cause significant morbidity. Delay of diagnosis may be fatal as the patient can quickly become hemodynamically unstable.

Clinically it can be classified as spontaneous (no obvious precipitating cause present) or traumatic. In patients with breast implant, it may be a direct complication of surgery (pleural damage) or secondary to pulmonary blebs. The mechanism of damage to the parietal pleura during surgery may include intraoperative pleura laceration during capsulectomy or creating a new muscular pocket, needle puncture for anaesthetic infiltration and pleural rupture due to high anaesthetic ventilation pressure. Falling SpO_2 levels despite oxygen supplementation generally occur. Chest auscultation reveals reduced air entry on the affected side, and chest X-ray can confirm the diagnosis. Chest drain insertion is required for successful treatment, determining good reinflation of the lung seen on check chest X-ray.

41.9.2 Pulmonary Embolism

Incidence of symptomatic pulmonary thromboembolism postoperatively after breast reconstruction is low, and it is reported in 0.7% of the patients after pedicled TRAM reconstructions [39]. Unfortunately, however, this number underestimates the true value because asymptomatic events may not have been identified. These accidents may present a dangerous and dreaded complication, even towards mortal pulmonary embolism.

Both latissimus dorsi and pedicled TRAM are associated with risk factors for deep vein thrombosis and pulmonary embolism including underlying malignancy (immediate reconstructions), operation time and transient immobilization in the postoperative period [77]. Abdominal flaps, however, have been thought to decrease venous return of the pelvis and lower extremities in superficial veins, increasing the risk of deep vein thrombosis and subsequent pulmonary embolism. In addition, an abdominal flap generally has a longer period until full ambulation in comparison with latissimus dorsi reconstruction [78]. This further supports the importance of proper deep vein thrombosis prophylaxis. The combination of the type of reconstruction (immediate vs. delayed procedures, abdominal flaps, dermolipectomy) and the predisposing individual patient risk factors (age, obesity, varicose veins, venous thromboembolism history, coagulation disorders) defines the level of thromboembolic risk that can be mild, moderate or high [79]. Prevention is based on general guidelines (early mobilization) and, at every level of potential risk, on the use of low molecular weight heparin and/or wearing antithrombosis stockings. If thromboembolic complications occur, their surveillance with duplex venous scanning of lower limbs is recommended at an early stage as well as appropriate systemic treatment to avoid evolution towards pulmonary embolism.

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