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Abdominal Adhesions: From Formation to Prevention - Part One

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Abstract

Abdominal adhesions are still a major surgical problem for their incidence, clinical consequences, social costs as well as medico-legal implications. Most of the adhesions are postoperative, especially after colon-rectal surgery. Given the increasing number of surgical procedures performed and the increase in the average age of patients undergoing surgery, adhesions are also a problem in term of rehospitalizations directly or probably related. Readmissions mean the use of health care system resources. The peritoneal damage during surgery is the first pathophysiologic step that unleashes an acute local inflammation resulting in activation of a complex cytochemistry cascade. Adhesion development is secondary to an imbalance between fibrin formation, part of the healing process, and fibrinolysis: this is caused by increased levels of molecules inhibiting plasminogen and contemporaneous reduction of those activating it. Video laparoscopic surgery is also associated to this modified balance that determinates a pro-fibrotic state. Finally, we analyze adhesions classification systems.

Introduction

Abdominal adhesions are still a major problem, given their incidence, clinical consequences, the increasing population average age and the possible need for further surgery, not to mention the economic and medical-legal problems. The adhesions can be responsible of intestinal mechanical obstruction, chronic abdominal pain, especially pelvic pain, and furthermore infertility. Knowledge of adhesions related disorder dates back to 1889 (1) when Dembrowski published the first data on the intra-peritoneal adhesions induction in an animal model. Intra-abdominal adhesions may be congenital or acquired. The first arise during the organogenesis secondary to an altered embryologic development of abdominal cavity (such as the sigmoid colon attachment to the left abdominal wall) and are usually asymptomatic (2), whereas the latter are primarily a consequence of surgery. Acquired adhesions may be the result of different kind of peritoneal injuries, also: peritonitis, other intra-abdominal inflammatory disease such as endometriosis and Pelvic Inflammatory Disease (PID), radiotherapy, foreign body reaction and long-term peritoneal dialysis (2, 3, 4). The mechanism underlying post-radiotherapy adhesions is unknown, whilst the radiation effect on already existing adhesions has been clearly stated: they are more vascularized, wider and more tenacious (5).

Epidemiology

A post-mortem study of patients who had not undergone surgery showed a prevalence of post-inflammatory adhesions in 28% of cases (6). The incidence of non-surgical related adhesions tends to increase with age, being more frequent over 60 years (6). Post-operative adhesions, however, rise in 50-100% of cases after abdominal-pelvic surgery, including gynecological surgery, whether the surgical approach is laparotomic or laparoscopic (7, 8, 9, 10). Total abdominal hysterectomy with bilateral oophorectomy is among gynecological interventions at greatest risk for adhesions formation, followed by laparotomic adnexal surgery, even though adhesions can also be found in 40-60% of

cases after a laparoscopic intervention for ectopic pregnancy (11). The gender and the Body Mass Index (BMI) are not related to post-operative adhesions developing (6). Surgery at greatest risk for developing adhesions is the colon-rectal surgery, as it has been demonstrated by the SCAR-3 study (Surgical and Clinical Research Adhesions) which is based on analysis of 12756 patients (12): hospital readmission rate directly related to adhesion disease was 5% at 5 years follow-up after surgery. Procedures most related to adhesion formation process are proctocolectomy (15.4%), total colectomy (8.8%) and ileostomy formation (10.6%). The appendectomy, per se, was associated with a low readmission rate (0.9%) but it still contributes to more than 7% of all adhesions-related readmissions, given the high incidence of this procedure. Adhesions related to appendectomy lead to intestinal obstruction needing surgical revision with a cumulated risk of 0.41% after 4 weeks, 0.63% after one year and 1.3% after 30 years. The risk increases in case of perforated appendix (adjusted hazard ratio 3.5), and it is still higher in case of operation for mesenteric lymphadenitis (adjusted hazard ratio 2.4), or for non-specific abdominal pain (adjusted hazard ratio 2.6) compared with operation for non-perforated appendicitis (13). These figures probably reflect a greater manipulation of bowel loops and a more extensive exploration of the abdominal cavity when the appendix is "negative". The appendectomy in children causes hospital admission for adhesion related disorder in the 0.3% of cases compared to 0.9% in the general population (12). Adhesions are deemed responsible for 40% of all intestinal obstruction, particularly small bowel obstruction (65-75% of cases) (8). Large bowel obstruction is mainly caused by neoplasms and only a small percentage of cases is secondary to adhesions (14). Most of the patients affected by bowel obstruction have a history of colon-rectal surgery (24%), gynecological surgery (22%), hernia repair (15%) or appendectomy (14%) (15). Mortality following surgery for bowel obstruction related to adhesions may be up to 10% (16). Intra-abdominal adhesions are also the first cause (42%) of injuries secondary to trocar or Verres needle placement (0.22% out of 29532 patients) (17): in case of accidental bowel injury conversion rate to laparotomy is almost 100%. Adhesions are in fact the most frequent cause of conversion in case of laparoscopic surgery (20-22%) (18, 19, 20, 21). This shows that, even if asymptomatic, adhesions may still represent a problem in patient undergoing surgery for a different reason: there is a significant risk of accidental enterotomy especially secondary to adhesions to the anterior abdominal wall which have to be taken into account in case of reoperation, whether with a laparotomic or laparoscopic approach, to reduce bowel injuries. In case of surgical intervention they can lead to a prolonged operative time and consequently a prolonged general anesthesia, hospital stay and finally higher costs; not to mention for a considerable risk of further adhesions formations (22, 23, 24). In addition to accidental enterotomy, the presence of adhesions increases the risk of intraoperative bleeding, injury to parenchymal organs (e.g. liver injury during right hemicolectomy or splenic injury during left hemicolectomy) and bladder and ureteral injuries (ureteric stents placed preoperatively may reduce the risk of injury and at least allow a prompt recognition of possible iatrogenic lesions); unfortunately there are limited data in literature regarding adhesion related injuries, perhaps for the reluctance to publish data about surgical accidents. Adhesions increase also the chance of other several complications such as formation of entero-enteric fistula, enterocutaneus fistula, enterovescical and enterovaginal fistulas and even short bowel syndrome secondary to repeated or wide bowel resections (25). One patient out of 3 undergoing surgery for adhesion related bowel obstruction has the risk of accidental enterotomy, whereas the risk of bowel injury in patients undergoing surgery for different pathology ranges from 4.7% to 6% (21, 26). The patient's age and the number of previous laparotomies are risk factors for accidental enterotomy: patients with history of 3 or even more previous laparotomies have a 10 times greater risk than patients who have undergone one or two laparotomies (27). The accidental bowel injury can often go unnoticed in course of laparoscopic surgery (up to 70% of cases in a urological study) (28), and its diagnostic delay significantly increases the mortality of patients (29, 30). The accidental enterotomy rate during cholecystectomy is 0.39%, 0.8% in course of intestinal resection, and 1.9% during laparoscopic hernia repair according a study of Binenbaum (31). Adhesions can involve any intraperitoneal organ and the damaged serosa but those involving the omentum and the surgical incision are the most common. The greater omentum is involved in 80% of cases of post-surgical adhesions while the bowel in approximately 50% (32, 33). Adhesions are less frequent with transverse skin incisions or Pfannenstiel incisions (25%) than with median

ones (70%) (33, 34) and less frequent after obstetrical surgery than after gynecological procedure. (35) Adhesions formation is more common after damage to the parietal peritoneum rather than visceral one, as is more likely when both peritoneal surfaces which come into contact are damaged (36). Adhesions are responsible of around 15-40% of infertility cases (2, 37). In fact adhesiolysis increases more than 50% the pregnancy rate in women infertile after laparotomic surgery (2). Ovarian adhesions can be proven in up to 90% of patients who had undergone adnexal surgery (38). Pelvic adhesion can alter the anatomy of the pelvis, complicate diagnostic ultrasound of endopelvic organs (uterus and adnexal), make difficult some assisted conception treatment (e.g. IVF) and also reduce the performance of medical procedures such as intraperitoneal dialysis and hyperthermic intraperitoneal chemotherapy. Even when are thin and avascular they are still capable to modify the pelvic anatomy limiting Fallopian tubal motility and in particular fimbria ability of retrieving the oocyte. This mechanism determines also a higher risk of ectopic pregnancy (37, 39).

Costs

Evaluating hospital admissions related to adhesion it should be differentiate between "*admissions directly linkable to post-surgical adhesions*" (hospital admission for adhesiolysis or for female reproductive tract adhesiolysis, rehospitalization related to adhesions with no surgical patient management) versus "*admissions just probably linkable to adhesions*" (rehospitalization for gynecological interventions or abdominal surgery, rehospitalization for abdominal/pelvic pain in patients previously operated). According to the SCAR study the overall percentage of "*readmissions directly or possibly related to adhesions*" is 34.6% in the 10 years following first operation with a mean of 2.1 admissions per patient and particularly the percentage of "*readmission directly related*" is 5.7% in 10 years (40). These figures are confirmed by the SCAR-2 study that evaluates the same cohort of patients analyzing only the lower intestinal tract surgery: 32.6% of readmissions for "*potential adhesion related problem* " with a mean of 2.2 admissions per patient in the subsequent 10 years and again 7.3% of hospitalizations were "*directly related*" to the adhesions (41). It is very interesting to note that 25% of rehospitalizations related to postoperative adhesions occur within the first year after surgery (41). The risk of readmission directly related to adhesions is not significantly different when comparing colonic to rectal surgery: 5% vs 5.2% (p = 0.681). Readmission is more likely in case of left hemicolectomy (4.9%) compared to right hemicolectomy (3.8% p = 0.0911) (table 1). Colonic surgery for diverticulitis does not increase the rate of rehospitalization compared to the same procedure performed for non-diverticular disease, although the number of cases of diverticulitis in the study is very low. The presence or absence of colorectal tumor does not affect the rehospitalization: readmission percentage was 6.2% in patients who underwent colonic surgery for benign disease while it was 3.8% in case of oncological surgery (p = 0.0002). The rehospitalization is slightly greater in patients who initially present with a clinical picture of peritonitis compared to those without; surprisingly Crohn's disease is not a risk factor for hospital readmission.

Type of Surgery	Readmissions	
	N°	%
Colonic Surgery	158/3176	5
Rectal Surgery	88/1690	5.2
Proctocolectomy	19/123	15.4
Total Colectomy	14/160	8.8
Right Hemicolectomy	47/1235	3.8
Left Hemicolectomy	50/1023	4.9
Sigmoidectomy	37/779	4.7
Colostomy	24/416	5.8

Tab 1: "Rehospitalizations directly related to adhesions in 5 years", modified from SCAR3 (12)

Furthermore twenty per cent of cases needed further abdominal or gynecological surgery to resolve the clinical problem (40). The SCAR2 study has also demonstrated that there has not been any improvement in the rehospitalization rate comparing patients who underwent colon-rectal surgery in 1986 to patients operated between 1996 and 1999; this means that despite the improvement of surgical techniques, the problem of adhesions related disorder remains constant over the

time. A study conducted in the USA has calculated 282,000 admissions secondary to adhesions in 1988 with a cost of 1.18 billion (42), and these data have been confirmed by other two studies in 1993 and in 1994, respectively with a cost of 1.3 and 1.33 billions of dollars, accounting for 1% of all admissions (43). The economic burden in UK for such admission in 10 years amounted to more than 500 million pounds (44). A British study in 2001 showed that hospitalization for postoperative adhesion related disorder has an average cost of 4677.41 pounds in case of operative management while the cost is 1606.15 pounds in case of conservative management (45). Using data of the SCAR study, the SCAR Steering Committee has calculated that the cost of surgical procedures related to adhesions in Scotland in 1994 was 6 million pounds which represents the 2% of entire Scottish health care cost (46). Applying a model of cost-effectiveness, Wilson (44) stated that the annual direct cost of hospitalizations directly linked to adhesions (and 75% of those probably related) would be superior to 24.2 million pounds in UK and over 95.2 million per year following 10 year after the "trigger" surgical procedure since most of the adhesion related disorders become symptomatic over the time. In Sweden Ivarsson (47) has showed that the overall medical care cost of adhesion related bowel obstruction is 13 million dollars per patient per year, without taking into account social costs, which would have obviously raised much more that figure. It has been shown that during a colorectal procedure for benign disease every 30 minutes spent for surgical adhesiolysis there is one day increase of hospitalization length; if a bowel injury occurs, hospitalization increases on average of 3 days (48). According to other studies, the operating time of any surgical procedure is protracted on average of 15 minutes in presence of adhesions, with an estimated cost increase of 330-690 dollars per operation (24, 49). Adhesion related disorders also become an important medical-legal issue, since they should be mentioned at the informed consent compilation time (27). In fact, according a survey of International Adhesions Society, only 10.4% of doctors have entered adhesions as possible complications in the informed consent, while only 14.4% mentioned them to the patient, however without adding such complication in the consensus (50). A similar analysis was conducted in UK in 2002: only 14% of patients undergoing laparotomy was made aware of the post-surgical adhesions risk (51). The figures published by Wiseman in 2003 are slightly better: 25% of patients was informed and in 10% of cases adhesions were mentioned as complications in the informed consent (52).

Physiopathology

Postoperative adhesions are the most frequent and multiple factors are involved in their development (table 2) (53). The peritoneum is a mesothelium which covers an area of about 2 square meters and consists of a single layer of cells anchored to a basement membrane. The basement membrane divides mesothelial cells from the underneath mesenchymal connective tissue that consists of an extracellular matrix (ECM) made up of various types of collagen, glycoproteins, glycosaminoglycans, proteoglycans fibroblasts, macrophages and mast cells.

Only 0.16-0.5% of peritoneal cells is in mitosis in basal conditions, since there is a slow cellular turnover. The rate of mitosis increases up to 30-60% after a peritoneal injury (63). Adhesions development recognizes as a first and fundamental input the surgical trauma to the peritoneum which establishes a consequent acute inflammation mechanism. Therefore the old concept that the adhesion was a simple inert scar has been largely supplanted by the idea of dynamic acute inflammation process (3, 64).

Risk Factors	Reference	Year
Type of surgery (higher risk with colo-rectal surgery)	12	2005
Complexity of the procedure, considering postoperative complications also (hematoma, intrabdominal collections, infections, re-operation, anastomotic leak….)	54	1997
Type of dissection (cold scalpel vs electric scalper vs harmonic scalpel vs CO^2 laser)	55	1992
Extent of peritoneal injury	56,57	1993, 2000
Patient comorbidities (e.g. diabetes) and medications taken	58	2001
Preoperative patient nutritional status	58	2001
Foreign bodies left at the time of operation (e.g. meshes, not-absorbable sutures, drainages and time of their permanence…)	58	2001
Exposition to foreign bodies during operation (latex gloves, tissue of surgical drapes and swab)	59,60	1996, 2002
Hemostasis not completed or excessive coagulation with necrotic tissue development	55	1992
Bacteria contamination and local or systemic infection	58	2001
Laparoscopy: tissue dehydration due to high-pressure gas insufflation, microcircle constriction, heat and artificial lightening; tissue-hypoxia due to CO^2	61,62	2008, 2001

Tab. 2 Risk factors

Adhesions microscopically are a tissue consisting of macrophages, eosinophils, fibrous tissue, mast cells and fibroblasts, which are variably present depending on the tissue remodeling phase and the final type of adhesion itself. Peritoneal damage causes bleeding from the microcircle with increased vascular permeability which lead to local concentration of inflammatory cells and exudation of fibrinogen which ultimately forms fibrin and finally adhesions. Patients who develop adhesions have a distorted balance between the fibrin formation, part of the physiological process of healing, and fibrinolysis: fibrinolytic activity of peritoneum is inhibited (table 3). Numerous studies, as it will be discussed later, have analyzed most of the cytokines involved in the inflammatory process, highlighting a very complex control system, which involves inhibitors and activators of fibrinolytic cascade.

Factors	Reference	Year
Damage of mesothelium surface integrity	65	1996
Local hypoxia / mesothelial ischemia	65,66,67	1996, 2008, 2007
Oxygen radicals formation	65	2008
Increase of profibrotyc systems	68	2002
Inhibition of fibrinolysis activators	69,70	2001, 1981
Bacterial contamination	66,71	2008, 1990

Tab. 3 Causes of altered peritoneal fibrinolysis, as amended by Bruggmann (53)

Following the initial mesothelial damage the first cells appearing on site are the polymorphonuclear neutrophils (PMNS) that persist for about 1-2 days. These are followed by monocytes that differentiate into macrophages and adhere to the damaged surface. Since the third day mesothelial cells begin to cover macrophages at the level of

damaged peritoneum (3). Local trauma is followed by increase of vascular permeability with subsequent formation of an exudate, instead of the normal transudate.
Among the exudate substances there is also fibrinogen which will be activated to fibrin.
Fibrin will be progressively replaced by a tissue containing fibroblasts, macrophages and giant cells. Under normal circumstances fibrin bands that have been developed during the physiological process of healing are degraded by the phenomenon of fibrinolysis in smaller fragments called fibrin degradation products (FDP).
The main fibrinolytic factor is plasminogen which is converted to plasmin, its active form, by plasminogen activators: the tissue plasminogen activator (tPA) and the less powerful urokinase-like plasminogen activator (uPA) (72).
Fibrinolytic activity is balanced by the plasminogen activator inhibitors (PAI): PAI-1 and 2 are able to inhibit the activity of tPA and uPA (73, 74, 75, 76). The complex regulation system of fibrinolytic activity includes also proteases and protease inhibitors: the matrix metalloproteinases (MMP) and their inhibitors called tissue inhibitors of metalloproteinases (TIMP). MMP and TIMP play an important role in the remodeling process of the extracellular matrix (ECM) where the mesothelial cells are leaning. Another key molecule of adhesion formation process is the transforming growth factor (TGF-β).
The TGF-β interacts with the fibrinolytic system and contributes to the synthesis of ECM stimulating fibroblasts to produce collagen and fibronectin. Overexpression of TGF-β from the parietal and visceral peritoneum is associated with an increased incidence of adhesions development both in animal and human being. The TGF-β plays a key role as it is able to control the MMP/TIMP and PAI/PA systems at the transcriptional level increasing the production of ECM (tab. 4).

Molecule	Fibrinolytic activity	References
Urokinase-like-plasminogen activator (u-PA)	up	68
Tissue plasminogen activator (t-PA)	up	68
Matrix metalloproteinases (MMP)	up	3
Tissue-derived inhibitors (TIMP)	down	3
Plasminogen activation inhibitors (PAI 1, PAI 2)	down	69,70
Interleukins (IL1, IL6)	down	77
Neurokinin-1 receptor (NK-1)	down	67
Tumor necrosis factor alpha (TNF alpha)	down	78,79
Transforming growth factor beta (TGF beta)	down	78,80
Intracellular and Vascular cell adhesion molecule (ICAM-1, VCAM)	down	2,78
Bacterial lipopolysaccharide	down	66,71
Substance P (SP)	down	65,67

Tab. 4 Molecules involved in fibrin formation / fibrinolysis equilibrium, as amended by Bruggmann (53)

The injured peritoneum has plasminogen activators action significantly reduced and at the same time concentrations of PAI increased (table 5).
These observations are the basis of pathophysiologic mechanism underlying adhesions formation: on one hand a reduced activity of those systems which should activate plasminogen and on the other hand an increased response of plasminogen activity inhibitor; the whole process resulting in a reduced fibrinolysis.
It has been found an altered concentration of cytokines in patients with post-surgical adhesions as a possible result of the adhesion formation process or more likely it could be the inflammatory environment itself that predisposes to the formation of adhesions.
Changes of activity levels of the molecules involved in this delicate balance has been proven by numerous studies, both on human and animals (81, 82, 83), although the results presented are not comparable (75, 76.84). These differences may be related to the fact that these studies are based on molecules concentrations.
The concentration of a substance is influenced

by its synthesis, its degradation but also and above all from its dilution.
None of the studies consider the amount of total abdominal fluid in which molecules are analyzed and the results, sometimes discordant, could be explained in this way.

In addition, since the process of adhesion formation is dynamic, it is clear that the time of the sampling and subsequent measurement is essential, and multiple consecutive samples give more informations than a single sample.

Molecule	Modification	References
Plasminogen/plasmin	down	85
Urokinse-like-plasminogen activator (u-PA)	down	73,86,87,88
Tissue plasminogen activator (t-PA)	down	73,86,87,88
Matrix metalloproteinases (MMP)	down	89,90
Tissue-derived inhibitors (TIMP)	up	91,92
Plasminogen activation inhibitors (PAI 1, PAI 2)	up	88,93
Interleukin 1-6 (IL1, IL6)	up	94,95,96,97
Interleukin 10 (IL-10)	down	98
Interferon gamma (INF-gamma)	down	98
Tumor necrosis factor alpha (TNF alpha)	up	94,95,99
Transforming growth factor beta (TGF beta)	up	100-107, 72
Intracellular and Vascular cell adhesion molecule (ICAM-1, VCAM)	up	108
Vascular endothelial growth factor (VEGF)	up	109-111
Hypoxia inducible factor 1 alpha (HIF-1alpha)	up	109-111

Tab. 5 Molecular modification during adhesion formation

Analyzing adhesion development process related to laparoscopic procedures it is clear that video laparoscopic surgery (VLS) for its less invasive nature reduces trauma to the peritoneal serosa, however the increased intra-abdominal pressure, the intensity of gas insufflation to induce pneumoperitoneum (PNP), changes in temperature and hypoxia are all conditions which modify and alter the peritoneum integrity and biology (table 6).
After VLS, due to its reduced invasiveness, the levels of IL-6 and PCR are lower than those in patients undergoing a laparotomy; furthermore during VLS there is a reduced inflammatory response caused by TNF-alpha and IL-1 compared to same surgical procedure conducted open (68, 112-115). The PNP with helium or CO^2 reduces the oxygen partial pressure at the level of peritoneal surface in the rat, whereas a mixture containing 80% of CO^2 and 20% of oxygen doesn't seem to affect the local concentration of oxygen. Inflation of heated and humidified CO^2 causes minor changes at the level of the peritoneum than those registered with cold and dry CO^2 (43). The adhesions incidence in a mouse model increases with increasing duration of the PNP and with higher inflation pressures. Conversely the incidence decreases when an oxygen mixture is added to the gas used for PNP.

Laparoscopy characteristics	Effects	References
Use of CO^2 to induce PNP	-Local hypoxia -Capillary flow reduction -Intercellular damage	-42 -116
Local hypoxia due to gas	-Reduction of T-PA, increase of PAI -Increase of HIF-1 alpha and VEGF -Metabolic acidosis	-110,117,118 -109-111 -111
Cold PNP	-Intra-abdominal temperature reduction -Increase of IL-1, TNF-alpha -Increase of PAI	-119 -80,102 -120

Tab. 6 Laparoscopy effects

The incidence reduction induced by oxygen is much more when it is added just only 2-3% of oxygen (69, 121). However the increase beyond this threshold of oxygen has deleterious effects, increasing adhesions (122). It has been shown that a constant perioperative oxygen supplementation determines an increase in partial pressure of oxygen in peritoneal surface also, reducing the adhesion severity (117). Peritoneal hypoxia induces metabolic acidosis with peritoneal pH reduction which remains stable at values around 6.4 during VLS. Once the procedure ends, the pH returns to normal values. Using helium to induce PNP the pH increases instead, stabilizing at 7.2 (123-125). During VLS intra-abdominal temperature decreases up to 27.7° C (119): hypothermia as well as acidosis leads to an alteration of fibrinolytic balance establishing a pro-fibrotic state. Very few human studies have compared VLS with open surgery in terms of adhesions formation. Bergstrom (126) has found an initial increase of PAI-1 concentration during VLS, as a possible consequence of CO^2 insufflation, but at the end of the procedure there were no statistically significant differences in patients undergoing laparoscopic cholecystectomy compared those undergoing to the same open procedure. Neudecker (127) showed a reduction in the activity of tPA both in the group of patients undergoing laparoscopic colonic resection and in that undergoing open resection: a bias of this study is secondary to the fact that in both study arms surgery was started with an exploratory laparoscopy which may have influenced the final results. The reduced fibrinolysis seems to start earlier with conventional surgery compared to the VLS, but the same effect during VLS becomes more marked if the intervention duration increases (> 90 min) (128). In conclusion VLS, despite being a minimally invasive technique, determines a peritoneal trauma resulting in reduced fibrinolysis. It should be noted that some studies have shown that using helium or air has less pro-fibrotic effects than CO^2, but further studies are needed to better explain these effects. Considering minimally invasive surgery, natural orifice transluminal endoscopic surgery (NOTES) seems to be superior both to laparotomic and laparoscopic surgery in terms of postoperative adhesions development, but a longer follow-up and bigger series are definitely needed to define properly the consequences (129).

Classifications

Many classification systems (table 7) have been proposed to score the severity, extent and type of adhesions. None of these has been accepted globally, thus leaving a considerable variability even within published studies. The ideal classification system should be simple and reproducible; a system with too many parameters that need to be assessed is not popular because it is difficult to apply, remember and analyze. Conversely a very simple system fails to highlight the differences between types of adhesions and therefore between the various arms of a possible study (108)

Morfologic classification of adhesions (130)
Type I: thin and avascularized
Type II: thick and avascolarized
Type III: thick and vascularized
Tipo IV: extended and condensed
Classification by POPA study (131)
0: absence of adhesions
1: filmy thickness, avascular
2: moderate thickness, limited vascularity
3: dense thickness, vascularized
Intraoperative adhesiolysis difficulty classification
Spontaneous
Traction lisys
Traumatic section
No clivage plan
Morfologic and dissection classification (132)
Filmy adhesions spontaneously separable
Filmy adhesions separable by gravity
Filmy adhesion separable by traction
Thick adhesion that need traumatic dissection

Tab. 7 Classification systems published in literature

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