

To Suture or Not The Gallbladder Bed After Open Cholecyctectomy

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ABSTRACT

Our study was conducted at Al-Khadhimiya Teaching Hospital, Al-ThuraghamPrivate Hospital, and Al-Shifa'a Private Hospital from February 2003 to August2006.

Sixty four patients had undergone elective open cholecyctectomy for uncomplicated gallstones. They were randomly divided into groups, each consisting of thirty two patients.

In the first group of patients the gallbladder bed was sutured, while in the second group the gallbladder bed was left raw, i.e. uncovered with peritoneum.

The drainage material of each patient in both groups was collected for theamount and the presence of bile to detect bile leakage. Postoperative follow up was done to detect any subhepatic collection or occurrence of postoperative adhesions and how these are influenced by the surgical technique of suturing the gallbladder bed ornot. We found that the difference in the amount of drainage between the two groups is not significant. Neither bile leakage nor subhepatic collections nor postoperative adhesions are related to surgical technique. In addition, postoperative complications are minimal and unrelated to surgical technique.

Aim:

To compare between whether to suture or not gallbladder bed after cholecystectomy in relation to drainage material and post operative complication.

Introduction:

Although cholecystectomy is the second commonest major operation performed today, the ideal ancillary procedures have not been settled.

In 1882 Carl Langenbuch devised the technique of cholecystectomy by careful cadaver dissection, and performed the first such procedure on a living person. He opened the abdomen through a vertical lateral rectus incision with a subcostal T-extension, after removing the gallbladder he inserted o peritoneal drain before closing the abdomen (2).

In 1913 Spivac was the first surgeon to record cholecystectomy without drainage "Ideal cholecystectomy"(1,2).

In 1927 Halpert was well described Luschka's ducts in the gallbladder wall, as duct like structures which are responsible for transient seepage of bile postoperatively (3). In 1942 Boys stated that there is no evidence to indicate that reperitonealization prevents the development of postoperative adhesions (5). In 1959 some authorities stated that seepage of bile from the gallbladder bed stops spontaneously (3).

In 1966 Farquharson recommended that when the divided duct in the gallbladder bed can be demonstrated it should be ligated or coagulated with the diathermy (6).

Manigot said in regard to suturing the gall bladder bed or not, it is not my practice to reperitonealize the gallbladder bed since it is known from both clinical and experimental studies that denuded serosa undergoes rapid regeneration (14).

Reperitonealization of the gallbladder bed after cholecystectomy is a standard procedure described in many textbooks of surgery.

Most surgeons who advocate non-drainage after cholecystectomy also routinely obliterate the gallbladder bed to reduce the amount of postoperative drainage and avoid adhesions to the liver (1, 2, 13, 15, 16, 18).

Those surgeons, who reconstitute the peritoneum over the gallbladder bed, think by so doing, they can achieve better haemostasis and reduce bile leakage

from small biliary radicals in the gallbladder bed (18).

Leakage of bile from the gallbladder bed is believed to come from Luschka's ducts. These ducts, unlike the cholecytohepatic ducts which are larger, may easily be missed during removal of gallbladder from its bed and leading to seepage of bile postoperatively. This seepage will stop spontaneously due to diversion of bile drainage through these ducts toward the intrahepatic biliary tree (10, 15, 18)

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The leakage of bile from these ducts will cause no clinical significance since it is discharged via the intraperitoneal drain, otherwise in non-drainage procedures, the accumulation of bile leads to a transient increase in the serum bilirubin due to resorption of bile through the peritoneum in the subhepatic region (2, 18).

Despite all of these theoretic advantages many surgeons used to leave the raw surface of the liver uncovered without complications.

The biliary system and the liver are of the same embryological origin. The liver primordium appears in the middle of the third week in 3mm embryo as an outgrowth of the endodermal epithelium at the distal end of the foregut, this outgrowth known as the hepatic diverticulum consists of rapidly proliferating cell strands which penetrate the septum transversum that is the mesodermal plate between the pericardial cavity and the yolk sac.

While the hepatic strands continue to penetrate the septum, the connection between the hepatic diverticulum and the foregut (duodenum) narrows, thus forming the common bile duct, its Y-shaped bifurcation produces the right and left hepatic ducts.

A small ventral outgrowth is formed by the common bile duct and this blind diverticulum gives rise to gallbladder and cystic duct.

During the last two months of intra-uterine life, the gallbladder and cystic duct have fully developed and the cystic duct has joined the hepatic duct. (11, 9, 12) The biliary apparatus collects bile from the liver, stores it in the gallbladder, and transmits it to the second part of duodenum.

The biliary apparatus consists of:

- 1. Right and left hepatic ducts
- 2. Common hepatic duct
- 3. Gallbladder
- 4. Cystic duct
- 5. Common bile duct

1.Hepatic ducts:

The right and left hepatic ducts emerge at the porta hepatis from the right and left lobes of the liver. The arrangement of structures at the porta hepatis, from behind forwards, is: the branches of the portal vein, the hepatic artery and the hepatic ducts.

2. Common hepatic duct:

It is formed by the union of two the hepatic ducts near the right end of porta hepatis. It runs downwards for about 3 cm to be joined on its right side at an acute

angle by the cystic duct to form the common bile duct. The cystic duct and the common hepatic duct with the quadrate lobe of the liver form the boundaries of Calot triangle in which the cystic artery passes.

3.Gallbladder:

It is a pear shaped organ, located in the bed of the liver in line with anatomic division of the liver into

right and left. It is divided into 4 parts: fundus, body, infundibulum and neck.

4. Cystic duct:

It is a three to four cm in length. It begins from the neck of the gallbladder, runs downwards, backwards and to the left where it lies against the porta hepatis to join the hepatic duct between the two layers of peritoneum that form the free edge of the lesser omentum (gastrohepatic omentum) forming the common bile duct. The cystic duct lies immediately in front of the right main branch of the hepatic artery. The mucous membrane of the cystic duct forms a series of 5-12 cresentic folds arranged spirally to form the so called spiral valve of Heister which is not a true valve.

5. Common bile duct:

It is approximately 8-11.5 cm in length and 6-9 mm in diameter. The upper portion is situated in the free edge of the lesser omentum to the right of the hepatic artery and anterior to the portal vein.

The middle third curves to the right, behind the first part of the duodenum where it diverges from the portal vein and the hepatic arteries.

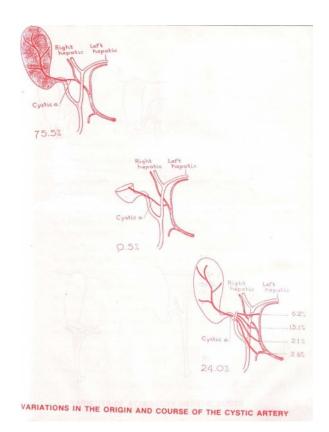
The lower third curves more to the right behind the head of the pancreas, which its grooves entering the duodenum at the ampulla of Vater, where it is frequently joined by the pancreatic duct.

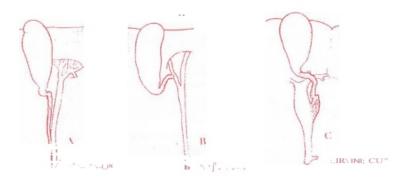
1-Ducts of Luschka:

Luschka's ducts in the gallbladder wall were well described by Halpert as ductlike structures found occasionally in the periphery of the gallbladder wall occurring most frequently on the hepatic surface of the viscus usually along the edges of fossa vesicae fellae, they have a wall of their own with the histological structures of intrahepatic bile ducts. The lumen is generally less than 0.3 mm in diameter. These ducts anastomose freely with each other and communicate with the intrahepatic biliary tree. No

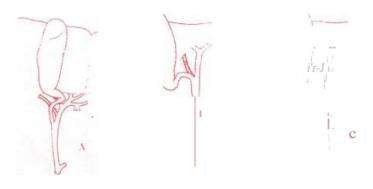
communicate with the intranepatic biliary tree. No communication with the gall bladder has ever been established.(12, 17, 19)

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VARIATIONS IN THE LENGTH AND COURSE OF THE CYSTIC DUCT



VARIETIES OF ACCESSORY HEPATIC DUCTS

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Sixty-four patients had undergone elective cholecystectomy (simple chronic cholecystitis), where the gallstones were confined to the gallbladder only. These patients were divided randomly into two groups, each group consisting of thirty-two patients in the first group the gallbladder bed had been sutured with 0-0 chromic cat gut and in the second group the bed was left unsutured.

The age of patients in the first group (sutured gallbladder bed group) ranged from 23 to 68 years with a mean age of 46 years. This group consists of 23 females and 9 males.

The age of the patients in the second group (nonsutured gallbladder bed group) ranged from 26 to 56 years, with a mean of 48 years. This group consists of 25 females and 7 males.

On admission to the hospital all patients in both groups were fully investigated, as they were going to undergo a major surgical procedure, so a complete blood picture and blood biochemistry (including blood urea, serum creatinine, total serum bilirubin,

serum GPT, and serum alkaline phosphatase) were done.

Ultrasonography of the abdomen was done twice to every patient by two different ultrasonographers to confirm the presence of gall stones in the gallbladder only. Elective cholecystectomy was arranged for, after giving the patient sedation I.M. as a premedication at the morning of the operation day.

A prophylactic antibiotic e.g. cephalosporin was given to the patient as 500mg keflin I.V. or garamycin 80mg for patients who were allergic to keflin at the time of induction of anesthesia.

After induction of anesthesia, scrubbing of the patient's abdominal wall with 5% chlorhexidine solution and a sterile draper were used to isolate the operating field.

A right subcostal incision (Kocher's incision) was done to all patients and after identification of the

Fine and delicate anatomical dissection is needed in this region to detect any of the congenital anomalies and to deal with it accordingly.

The gallbladder is removed from the bed until reaching its fundus, and then the gallbladder bed was inspected after packing to watch any bleeder or bile leak. Identification of a major bile leak was dealt with by a transfixation ligature with 0-0 chromic cat gut and after that haemostasis of the gallbladder bed was achieved by a coagulation diathermy or local application of a haemostatic agent like gel foam.A decision to suture the gallbladder bed by leave peritoneal leaf cover on the gallbladder to suture it on the gallbladder bed without injury to the liver or leave the gallbladder bed uncover made in random fashion. A tube drain was put in Morison's pouch in all our patients and the amount of drainage material was measured and recorded over a period of 48 hours at the end of which the drain was removed (Fig.1).

All our patients had received intravenous fluid postoperatively and 500 mg keflin six hourly for three days and then changed to oral antibiotic for five days.

A sample of drainage material was sent to the laboratory to measure the level of bilirubin in the first postoperative day as well as a total serum bilirubin and Serum GPT levels were estimated. An ultrasonography was done on the 4th and 10th postoperative day to detect any collection in the gallbladder bed.

We followed up our patients for a period of six months by doing liver function test and abdominal ultrasound on monthly intervals operation to detect any possible remote complications related to our surgery.gallbladder, the cystic duct, and the cystic artery; ligation of both cystic duct and artery with 0-0 chromic cat gut and 2-0 silk was done respectively.

Pati	ent's Data	Sutured Gallbladder bed	Non-sutured Gallbladder bed
Number		32	32
Age (year)	Range Mean	23-68 46	26-56 48
Sex	Male Female	9 23	7 25
Average amount of Drainage (ml)		38 ± 4.9	46 ± 4.7

Table 1: Clinical Data

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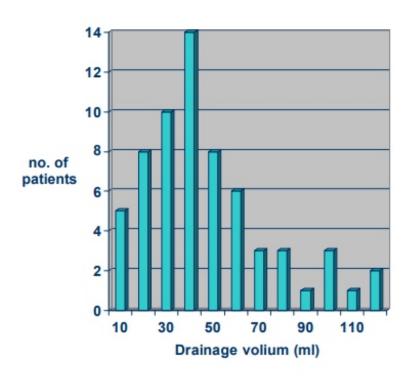


Fig. I: Distribution of drainage volume after simple cholecystectomy

Results:

In our studied groups, 8 patients were hypertensive and 3 were diabetics, and both diabetes and hypertension were controlled.

The fluid in the drainage bottles was blood stained serum in all patients of both groups.

The amount of drainage material in the first group (sutured gallbladder bed group) was found to be in the range of 8ml to 111 ml, and the mean of drainage was 38 ± 4.9 ml; (mean \pm SE).

In the second group (unsutured gallbladder bed group), the amount of drainage found to be in the range of 9ml to 120ml, and the mean was 46 ± 4.7 ml. Analysis of the amount of drainage showed a similar distribution in both groups (Fig. II).

There was no significant difference (P value > 0.05) between the two groups regarding the amount of drainage material.

The level of bilirubin in the drainage material was estimated in all patients of both groups and it was found to be similar to bilirubin level in the serum or slightly more, and the highest value of bilirubin concentration in the drainage material was 1.4mg/dl in a patient in the sutured gallbladder bed group and 1.6 mg/dl in patient in the nonsutured gallbladder bed group.All patients in both groups had the same results of total serum bilirubin and serum GPT postoperatively as those done preoperatively. Regarding the congenital anomalies, a major bile duct (cholecystohepatic duct) on the liver surface at the gallbladder bed was demonstrated in three patients, two in the non-sutured bed group and one in the sutured bed group. Various types of the cystic duct and artery were found in 8 patients of bothgroups and all congenital anomalies were dealt with accordingly.

Ultrasonography didn't show any collection at the gallbladder bed or in the subhepatic region or any intraabdominal abscesses, neither during the early

postoperative period nor in the follow up period when it was done monthly for six months. The average period of hospitalization was 3 days in both groups.

In our study the complications were divided into general and specific complications.

Regarding the general complications, six of our patients developed postoperative pyrexia; two of those patients were in the non-sutured group and four in the sutured gallbladder bed group. The cause of fever was found to be chest infection in three patients, phlebitis at the site of intravenous line in two patients and urinary tract infection in the sixth one.

On the other hand, specific complications were:

1. Wound infection was found in two patients, one in each group. The infection was superficial and swab was sent for culture and sensitivity which revealed staphylococcus aureus in one patient and no growth of bacteria could be detected in the other.

2. No postoperative bleeding or hematoma collection at the site of operation in both groups.

3. No bile leakage was recorded in any patient.

4. No subhepatic collection or intra-abdominal abscesses were recorded in any patient.

5. Two patients remained with symptoms same as preoperative complaints for which cholecystectomy had been performed. These two patients were fully investigated to find the cause, which was duodenal ulceration in one of them and no cause could be found in the other. Both of these patients were in the nonsutured gallbladder bed group.

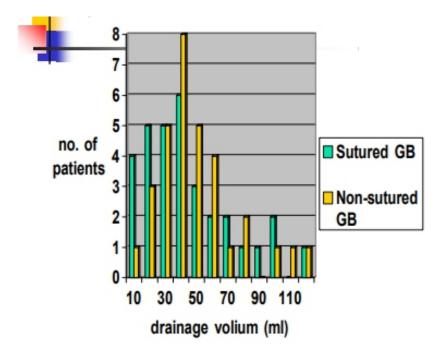


Fig-II Comparison of drainage volume in patients with and without gallbladder bed reperitonealization

Discussion:

Review of the literatures showed that the majority of surgeons who have favored non-drainage procedures following cholecystectomy routinely close the gallbladder bed, believing such a procedure would decrease the amount of drainage postoperatively. (1, 2, 13, 15, 16, 18).

Mok and Li studied the effects of reperitonealization of the gallbladder bed after elective cholecystectomy for uncomplicated gallstones in 88 patients who were divided randomly into two groups. In the first group the gallbladder bed was sutured whereas in the second group, the raw surface was left uncovered. They noticed no significant difference between the two groups in terms of drainage (18).

In our study we found no significant difference between the two groups regarding the amount of drainage material. Mok and Li noticed that the drainage fluid from all patients was serosanguinous in character, and the bilirubin level did not exceed that normally found in plasma, therefore: bile leakage was not recorded in any patient of their studied groups (18).

In our study we found that the drainage material in all our patients was blood stained serum and its bilirubin level did not exceed the bilirubin level in the serum except in five of our patients who were allocated, two in the sutured gallbladder bed group (6.2%) and three in the non-sutured gallbladder bed group (9.3%). In these five patients the level of bilirubin in the drainage fluid is slightly higher than its level in serum but it was not more than double its serum level. The cause of this high bilirubin level in the drainage fluid may be the seepage of bile from the divided Luschka's ducts and in two of these patients the gallbladder had been opened and bile escaped into the peritoneal cavity during it's removal from it's bed, or it may be due to a laboratory error, and this rise in the bilirubin level is clinically insignificant since all these five patients passed a smooth postoperative period with no clinical manifestation of bile leak and it's toxicity.

Vander Linden demonstrated active bile leakage after cholecystectomy. He found that after cholecystectomy when the gallbladder bed was denuded, injection of technetium99m resulted in the rapid appearance of radioactivity in the collected drainage fluid inferring that newly formed bile escaped freely into the abdominal cavity. However, suturing of the raw gallbladder bed did not seem to alter the level of radioactivity in the discharged fluid but at a slower rate than in the raw gallbladder bed ,though by U/S unsuspected sub hepatic fluid c ollections were often present in postcholecystectomy patients(20).

In our study regarding the sub hepatic collections following cholecystectomy,little is known about the incidence of postcholecystectomy fluid collection despite much debate in the literatures about the efficiency of routine postoperative drainage.

Ultrasound is a useful modality for detection of intraabdominal fluid collections as with increased use of ultrasound, clinically unsuspected subhepatic fluid collections were often present in postcholecystectomy patients, there is no facility to do technetium 99m.

Elboim et al had studied the occurrence of subhepatic fluid collection following cholecystectomy. He found that the subhepatic collections present in 25% of patients and 42% of these collections were of no clinical significance detected by ultrasonographic examination. These asymptomatic collections were more common in emergency operations (40.7% vs. 17.9%) and when the gallbladder bed was not closed (35.9% vs. 4.1%) (4).

In our study we could not detect any subhepatic collections in all patients of the two groups. The difference between our results and Elboim's results regarding the subhepatic collections is that he carried cholecystectomy both in elective and emergency conditions, but in our study we did only elective cholecystectomy for uncomplicated gallstones i.e. (simple chronic cholecystitis).

The detection of subhepatic collection by ultrasound depends on the patient's position during the examination and necessitates special interpretation by the ultrasonographer and use of a highly sophisticated ultrasound machine.Regarding the postoperative adhesions and their occurrence following cholecystectomy, it has been found from both clinical and experimental evidence of many studies that defects in the peritoneum, if left alone, heal speedily and usually without adhesions.

The teaching that raw surfaces within the abdominal cavity must be avoided wherever possible results from a faulty concept based on a false comparison with healing of similar lesions of cutaneous surfaces, which inevitably result in formation of scar tissue.

Peritoneum (a mesodermal derivative) is quite different in its behavior and a gap in it heals, not by a process of encroachment from the edges of the defect to form scartissue, but by differentiation of a new mesothelium from the underlying connective tissue cells which was completed within 7 days (18).

So a lack of serosal integrity is not an-important factor in the genesis of adhesions, it is the local ischemia that created by suturing of peritoneum over the gallbladder bed and attempts that made to suture a wide gallbladder bed often lead to further damage to liver and hemorrhage which end in ischemia at the sutured edges.

So the concept of suturing the gallbladder bed which is believed to reduce the likelihood of adhesions in the subhepatic region is incorrect because the postoperative fibrous adhesions are now regarded as a vascular graft to ischemic areas (2, 5, 18).

So the process of reperitonealization can safely be left to nature itself.

Mok and Li found that obliteration of the gallbladder bed by suture may in fact create local ischemia and promote more adhesions, making subsequent reexploration difficult and tedious (18).

In our study we can not prove which procedure is superior to the other in preventing the occurrence of adhesions and we depend on our clinical examination and assessment of all patient during the period of follow up and since none of our patients in both groups had presented with colicky pain in the right hypochondrium or recurrent attacks of vomiting or any signs and symptoms of intestinal obstruction and since we did not re-explore any patient for any cause, so we do not suspect or confirm the occurrence of post operative adhesion in any patient of both groups, in all patients the ultrasonography showed a clear bed.

So there is no difference between the two groups in enhancing or developing postoperative adhesions.

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Table2:Comparism between our study& previous study

Discussion	previous study	our study
1.effects of reperitonealization of the gallbladder bed	(by Mok and Li studied): no significant difference between the two groups	no significant difference
2. character of drainage fluid	serosanguinous	blood stained serum
3. bilirubin level	did not exceed that normally	In 5 patient slightly higher than its level in serum but it was not more than double
4. adhesion	obliteration of the gallbladder bed by suture may in fact create local ischemia and promote more adhesions	none of our patients in both groups had been developing postoperative adhesion

Discussion	previous study	our study
4. bile leakage after cholecystectomy	(by Vander Linden): Injection of TC99m result in rapid appearance in drainage fluid ,suturing of bed did not alter the level of TC99m.but slower rate by U/S unsuspected subhepatic fluid collection	No facility to do TC99& Clinically unsuspected subhepatic fluid collection is detect by U/S.
5. subhepatic fluid collection	(by Elboim et al): present in 25%, common in emergency &carried cholecystectomy both in elective and emergency	We could not detect any subhepatic collections in all patients of the two groups. in our study we did only elective cholecystectomy

Conclusions & recommendation:-

1. Routine closure of the gallbladder bed does not reduce the amount of postoperative drainage and will not prevent bile leakage if it will occur.

2. There is no significant difference between the two groups in terms of drainage, and drainage from the gallbladder bed is independent on suturing the bed or not.

3. Non-suturing technique is easier, safer and less time consuming with minimal anatomical disturbance.

4. Suturing technique may cause trauma to the liver tissue, bleeding at the site of needle puncture and takes more time with an evident changes in the anatomy of the region.

5. There is no prove which procedure is superior to the other in preventing the occurrence of adhesions and both give similar results.

6. There is no significant morbidity related to each group and both with minimal postoperative complications.

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