Clinical Study

Hemosuccus Pancreaticus: 15-Year Experience from a Tertiary Care GI Bleed Centre

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Received 23 December 2012; Accepted 8 January 2013

Academic Editors: K. Camphausen and H.-X. Xu

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Background. Hemosuccus pancreaticus (HP) is a very rare and obscure cause of upper gastrointestinal bleeding. Due to its rarity, the diagnostic and therapeutic strategy for the management of this potentially life threatening problem remains undefined. The objective of our study is to highlight the challenges involved in the diagnosis and management of HP and to formulate a protocol to effectively and safely manage this condition.

Methods. We retrospectively reviewed the records of all patients who presented with HP over the last 15 years at our institution between January 1997 and December 2011. Results. There were a total of 51 patients with a mean age of 32 years. Nineteen patients had chronic alcoholic pancreatitis; twenty-six, five, and one patient had tropical pancreatitis, acute pancreatitis, and idiopathic pancreatitis, respectively. Six patients were managed conservatively. Selective arterial embolization was attempted in 40 of 45 (89%) patients and was successful in 29 of the 40 (72.5%). 16 of 51 (31.4%) patients required surgery. Overall mortality was 7.8%. Length of followup ranged from 6 months to 15 years. Conclusions. Upper gastrointestinal bleeding in a patient with a history of chronic pancreatitis could be caused by HP. All hemodynamically stable patients with HP should undergo prompt initial angiographic evaluation, and if possible, embolization. Hemodynamically unstable patients and those following unsuccessful embolization should undergo emergency haemostatic surgery. Centralization of GI bleed services along with a multidisciplinary team approach and a well-defined management protocol is essential to reduce the mortality and morbidity of this condition.

1. Introduction

Hemosuccus pancreaticus (HP) is a rare and potentially life threatening clinical entity and is described as bleeding from the ampulla of Vater via the pancreatic duct. It is the least frequent cause of upper gastrointestinal bleeding (1/1500) and is most often caused by chronic pancreatitis, pancreatic pseudocysts, or pancreatic tumors [1–3]. HP is often difficult to diagnose, partly because of its rarity and due to its anatomical location and also because the bleeding is often intermittent and cannot be easily diagnosed by esophagogastroduodenoscopy (OGDscopy) in the intermittent phase [1, 4]. The diagnosis is not always easy to establish and often a long period elapses between the onset of the first symptoms and the precise location of the source of bleeding [2, 4]. The objective of our study was to highlight the challenges involved in the diagnosis and management of HP and to audit our unit’s HP protocol to effectively and safely manage this condition.

2. Materials and Methods

We retrospectively reviewed the records of all 51 patients who presented with HP over the last 15 years at our institution between January 1997 and December 2011. We noted demographic data, history, diagnostic features including symptoms, physical examination and time to diagnosis, investigations and therapeutic modalities, as well as follow-up data.
All patients were managed as per our department's algorithm. Following aggressive resuscitation, they were subjected to esophagogastroduodenoscopy and a Duplex scan. After hemodynamic stabilization, a CT angiogram followed by angiography ± angioembolization was done. In the event of continued destabilization or failed angioembolization, the patient was taken up for emergency surgery. Celiac trunk and superior mesenteric arteriography were performed transfemorally using a 4-F catheter. Images were obtained using the digital subtraction technique. Demonstration of contrast agent extravasation and/or vascular anomalies like pseudoaneurysm was an attempt to attempt the use of a microcatheter and microcoils. The treatment technique included a superselective catheterization of the feeding artery, followed by endosaccular treatment of the pseudoaneurysm. Proximal and distal feeding artery occlusions were done. All patients had a clinical examination after 1 month and were kept on routine followup. Angioembolized patients had clinical examinations during the 6-month period and if a pseudocyst existed on US or CT scan, endoscopic or surgical therapy was planned. Persistently symptomatic patients with chronic pancreatitis were offered surgery when indicated.

3. Results

The sample included forty-three men and eight women. Mean age was 32 years (11–55 years). Most common presenting symptoms were worsening anemia and malena in forty-seven and forty-eight patients. Transfusion requirements ranged from 3 units to 12 units (mean-7 units). (Tables 1 and 2). Twenty-six had tropical pancreatitis, nineteen patients had a diagnosis of chronic alcoholic pancreatitis, five patients had acute pancreatitis, and one patient was diagnosed as having idiopathic pancreatitis (Table 3).

3.1. Diagnosis. Upper GI endoscopy showed the presence of blood in the duodenum in 26 of 51 (51%) patients. The remaining patients had normal UGI endoscopic findings. Abdominal ultrasonography with Doppler examination was performed in all patients and diagnosed aneurysmal bleeding in nineteen patients. Contrast-enhanced computed tomography (CECT) angiogram was performed in all 51 patients and showed a pseudoaneurysm in 46 (90%) patients. Selective angiography was done in 45 patients with a therapeutic intent (Table 4). The combined modality of CECT angiogram and conventional angiogram showed that the pseudoaneurysm was located in the head of the pancreas in 31 patients and in the body or tail in the remaining 20 patients. The mean (range) diameter of the pseudocyst was 67 (10–150) mm and the mean diameter of the pseudoaneurysm was 24 (7–80) mm. The pseudoaneurysm arose from the splenic artery in twenty-seven patients, from the gastroduodenal artery in nine, from a branch of the superior pancreaticoduodenal artery in two, from the inferior pancreaticoduodenal artery in two, and from the superior mesenteric artery in one patient. An unnamed vessel in the pseudocyst wall was the cause of bleed in nine patients. A ductal communication with the splenic vein was found in one patient. An arterial abnormality was found to be the principle cause in 50 of 51 (98%) patients (Table 5).

3.2. Management. Selective arterial embolization was attempted in forty of forty-five (89%) patients and was successful in 29 (72.5%) patients (Figure 1). Sixteen of forty-five (36%) patients required surgery to control bleeding after the failure of arterial embolization in eleven and surgery in an emergent setting in five patients. Procedures included distal pancreatectomy and splenectomy in nine cases, central pancreatectomy in one case, intracystic ligation of the blood vessel in five cases, and aneurysmal ligation and bypass graft in one case (Table 6 and Figure 2). Six patients were managed without any therapeutic intervention apart from hemodynamic resuscitation, out of which four refused any form of therapy, and two patients exsanguinated before any therapeutic modality could be offered. There were four mortalities, one patient following surgery alone and one following surgery for a failed embolization. Two patients expired before any procedure could be performed. Morbidity included external pancreatic fistula in four patients (managed conservatively), ischemic cholecystitis (managed with percutaneous cholecystostomy) in one, wound infection in eleven patients, incisional hernia in three, and pneumonia in six patients (Table 7).

3.3. Followup. Length of followup ranged from 6 months to 15 years. Twelve patients were lost to followup. None of

### Table 1: Demographics.

| Demographics | 
|--------------|
| Mean age, years (range) | 32 (11–55) |
| Male : female | 43 : 8 |
| Transfusion requirements, units (range) | 7 (3–12) |

### Table 2: Presenting symptoms.

| Presenting symptoms | N = 51 |
|---------------------|--------|
| Haematemesis        | 16     |
| Malena              | 48     |
| Pain abdomen        | 31     |
| Worsening anemia    | 47     |

### Table 3: Etiology.

| Etiology                        | N = 51 |
|---------------------------------|--------|
| Tropical chronic pancreatitis   | 26     |
| Alcoholic chronic pancreatitis  | 19     |
| Alcoholic acute pancreatitis    | 05     |
| Idiopathic pancreatitis         | 01     |

### Table 4: Investigations.

| Investigations, positive yield | n = 51 |
|-------------------------------|--------|
| Upper gastrointestinal endoscopy | 26/51 (51%) |
| Ultrasound and Doppler study   | 19/51 (38%) |
| CECT                           | 46/51 (90%) |
| Selective angiography          | 40/45 (89%) |
the thirty-five patients, who were followed up, experienced a recurrent bleed. Seven of the patients with chronic pancreatitis, who had undergone previous embolization, developed intractable pain and needed drainage procedures.

4. Discussion

Hemosuccus pancreaticus is a very rare cause of upper gastrointestinal bleeding. Approximately 150 cases have been reported in the literature since it was first reported by Lower and Farrell in 1931 [3]. In 80% of the cases, hemosuccus pancreaticus complicates an underlying pancreatic disease [5, 6]. Clinical symptoms and signs include UGI bleeding as evidenced by haematemesis and malena, of which malena is more common. Epigastric pain results from the elevation of pressure in the pancreatic ducts caused by blood clots [1, 5–7]. The haemorrhage is usually intermittent, repetitive and, most often, not severe enough to cause haemodynamic instability.
finding of clotted blood in the pancreatic duct, known as
cystic and pseudoaneurysms. On precontrast CT, the characteristic
demonstrate features of chronic pancreatitis, pseudocysts,
for demonstrating the pancreatic pathology and can also
diagnostic. Contrast-enhanced CT is an excellent modality
ultrasound or dynamic ultrasound has been reported to be
Ultrasonography can be used to visualize pancreatic pseu-
and oesophageal and gastric fundus varices, etc.) [8–10].
of upper digestive bleeding (erosive gastritis, peptic ulcers,
endoscopy may be normal, it helps to rule out other causes
despite the usual arterial origin of bleeding [7, 8]. Other
clinical signs are more exceptional and include jaundice,
vomiting, weight loss, and a palpable pulsating mass with a
systolic thrill in the event of aneurysm [1, 7–9]. Liver function
test is normal apart from an increased serum bilirubin in
the event of pancreaticobiliary reflux. Serum amylase is
normal outside episodes of acute pancreatitis. It is difficult
to diagnose HP because the bleeding is usually intermittent.
Endoscopy is essential in ruling out other causes of upper
gastrointestinal bleeding and in rare cases; active bleeding
can be seen from the duodenal ampulla [9–11]. Even though
endoscopy may be normal, it helps to rule out other causes
of upper digestive bleeding (erosive gastritis, peptic ulcers,
and oesophageal and gastric fundus varices, etc.) [8–10].
Ultrasonography can be used to visualize pancreatic pseudo-
cysts or aneurysm of the peripancreatic arteries. Doppler
ultrasound or dynamic ultrasound has been reported to be
diagnostic. Contrast-enhanced CT is an excellent modality
for demonstrating the pancreatic pathology and can also
demonstrate features of chronic pancreatitis, pseudocysts,
and pseudoaneurysms. On precontrast CT, the characteristic
finding of clotted blood in the pancreatic duct, known as
the sentinel clot, is seldom seen. Computed tomography
may show simultaneous opacification of an aneurysmal
artery and pseudocyst or persistence of contrast within a
pseudoaneurysm after the arterial phase. Again, these findings
are only suggestive of the diagnosis. Ultimately, angiography
is the diagnostic reference standard. Angiography identifies
the causative artery and allows for delineation of the arterial
anatomy and therapeutic intervention [9, 12–16]. HP is an
entity diagnosed on clinical, endoscopic, and radiological
findings, and a definitive diagnosis can be established only
with angiography. Overall, the diagnosis of HP requires a
high index of suspicion in patients with pancreatitis and GI
bleeding. The natural history of chronic pancreatic pseudo-
cysts and the risk of pseudoaneurysm formation are not well
known [14–16]. The rate of pseudoaneurysm formation varies
from 4% to 17% in operated pseudocyst patients and is about
7% in endoscopically treated series [17–19]. In a series of 14
patients with chronic pancreatitis and bleeding pseudocysts,
11 were treated successfully with embolization and 3 needed
an operation. The overall mortality rate of 14% was related
to the failure of the embolization or a complication. This
compares favorably with the mortality rate of our series
(7.8%). The authors concluded that arterial embolization is
recommended as the initial therapeutic method, and
further surgery should be reserved for patients in a good
general condition who have other complications of chronic
pancreatitis that need surgery [20]. Distal pancreatectomy for
bleeding pancreatic pseudoaneurysms in the body or tail of
the pancreas is a surgical alternative to angioembolization
[21, 22]. When the pseudoaneurysm is located in the head of
the pancreas, surgical resection is associated with increased
mortality and morbidity, and angioembolization alone has
been proposed as the recommended treatment modality
of choice [19, 20, 23, 24]. Bleeding from the pancreato-
duodenal artery has a higher mortality rate than bleeding
from the splenic or gastroduodenal artery (46% versus 21%
and 28%, resp.) [23, 24]. Early angiography has halved the
mortality rate. Once the haemodynamic situation is under
control, interventional radiographic methods are used for
initial treatment, with immediate good results in 60–100%
of cases (72.5% in our series) [9, 15, 21]. Angiographic
intervention of a haemorrhage from pseudoaneurysm in HP
can be carried out either to stabilize the patient in order to
perform elective surgery or as a definitive treatment [22, 23,
25]. Failure of catheter embolization may result from factors
such as inability to isolate the bleeding vessel, spasm of the
bleeding vessel, incomplete arterial occlusion, or misidenti-
fication of the bleeding vessel [21, 25, 26]. If a conservative
transarterial approach is selected in a patient with chronic
pancreatitis, the remaining diseased pancreas adjacent to the
previously injured artery may be the source of reoccurrence
of arterial injury and bleeding. With increasing expertise
and the use of superselective angiocatheters, therapeutic
embolization can serve as a definitive management strategy.
Surgical treatment is indicated in uncontrolled haemorrhage,
persistent shock, when embolization is not feasible or when
embolization fails (continued or recurrent bleeding). It is also
indicated in patients who have other indications for operative
intervention (pseudocyst, pancreatic abscess, gastric outlet

Table 5: Source of bleed.

| Source of bleed                  | n = 51 |
|----------------------------------|-------|
| Splenic artery                   | 27    |
| Gastroduodenal artery            | 09    |
| Unnamed Intracystic artery       | 09    |
| Sup. Pancreaticoduodenal art.    | 02    |
| Inf. Pancreaticoduodenal art.    | 02    |
| Superior mesenteric art.         | 01    |
| Superior mesenteric vein         | 01    |

Table 6: Management strategy.

| Management strategies            | (n = 51) |
|----------------------------------|----------|
| Angiographic embolization attempted | 40/45 (89%) |
| Angiographic embolization successful | 29/40 (72.5%) |
| Surgery                          | 16/45 (36%) |
| Distal pancreatectomy and splenectomy | 09 |
| Central pancreatectomy           | 01 |
| Intracystic ligation of blood vessel | 05 |
| Aneurysmal ligation and bypass graft | 01 |
| No therapeutic intervention (apart from hemodynamic resuscitation) | 06 |

Table 7: Complications.

| Complications               |       |
|----------------------------|-------|
| External pancreatic fistula | 4     |
| Ischemic cholecystitis      | 1     |
| Wound infection             | 11    |
| Pneumonia                   | 6     |
| Incisional hernia           | 3     |
| Mortality                   | 4     |
obstruction, obstructive jaundice, or incapacitating pain) and are otherwise appropriate surgical candidates [27]. In our series, 16 of 45 (36%) patients required surgery to control bleeding after the failure of arterial embolization in five cases and in an emergent setting in eleven. Arterial ligation is also effective, but it does not avoid the risk of recurrence. Drainage of the pancreatic pseudocysts associated with arterial ligation is particularly effective and is associated with fewer complications of infection and necrosis compared with aggressive surgery [21, 25–27]. More aggressive surgery with pancreatic resection enables the treatment of both the pancreatic and arterial diseases. Surgical procedures in our series included distal pancreatectomy and splenectomy, central pancreatectomy, intracystic ligation of the blood vessel, and aneurysmal ligation and bypass graft. In patients with chronic pancreatitis, pancreaticoduodenectomy or splenopancreatectomy are preferred by certain authors, but the problems of potential perioperative complications and postoperative pancreatic insufficiency should not be overlooked. Less radical approaches such as central pancreatectomy and intracystic ligation of pseudoaneurysm can be performed in place of pancreaticoduodenectomy. The documented success rates in most surgical series are in the range of 70–85%, with a mortality rates of 20–25% [9, 15–17].

5. Conclusion

Upper gastrointestinal bleeding in a patient with a history of chronic pancreatitis could be caused by HP. Diagnosis is based on investigations that should be performed in all patients, preferably during a period of active bleeding. Therapeutic options consist of selective embolization and surgery. All hemodynamically stable patients with HP should undergo prompt initial angiographic evaluation and if possible, embolization. Hemodynamically unstable patients and those following unsuccessful embolization should undergo emergency haemostatic surgery. A multidisciplinary team approach at a tertiary care centralized GI bleed centre with a well-defined protocol is indispensable in drastically reducing mortality and morbidity.

Conflict of Interests

The authors have no conflict of interests or financial ties to disclose.

Authors’ Contribution

Dr. A. Rammohan, Professor S. Jeswanth, Dr. R. Sukumar, Dr. L. Anand, and Dr. P. S. Kumar contributed to conception and design, acquisition, analysis, and interpretation of data. Dr. A. Rammohan, Professor S. Jeswanth, Dr. UP Srinivasan, Dr. R. Ravi drafted the paper and revised it critically for important intellectual content. Professor S. Jeswanth and Professor P. Ravichandran gave the final approval of the version to be published.

References

[1] A. M. Callinan, J. S. Samra, and R. C. Smith, "Haemosuccus pancreaticus," Australian and New Zealand Journal of Surgery, vol. 74, pp. 395–397, 2004.
[2] P. H. Sandblom, "Gastrointestinal haemorrhage through pancreatic duct," Annals of Surgery, vol. 171, pp. 61–66, 1970.
[3] W. E. Lower and J. T. Farrell, "Aneurysm of the splenic artery: report of a case and review of literature," Archives of Surgery, no. 23, pp. 182–190, 1931.
[4] S. Etienne, P. Pessaux, J. J. Tuech et al., "Hemosuccus pancreaticus: a rare cause of gastrointestinal bleeding. A series of 9 cases," Gastroenterologie Clinique et Biologique, vol. 29, no. 3, pp. 237–242, 2005.
[5] J. L. Peroux, J. P. Arput, M. C. Saint-Paul, R. Dumas, P. Hastier, and F. X. Caroli, "Wirsungorragie compliquant une pancréatite chronique associée à une tumeur neuroendocrine du pancréas," Gastroenterologie Clinique et Biologique, vol. 18, pp. 1142–1145, 1994.
[6] R. Frayssinet, J. Sahel, and H. Sarles, "Les wirsungorragies," Gastroenterologie Clinique et Biologique, vol. 2, pp. 993–1000, 1978.
[7] M. Suter, F. Doeniz, G. Chapuis, M. Gillet, and P. Sandblom, "Haemorrhage into the pancreatic duct (hemosuccus pancreaticus): recognition and management," European Journal of Surgery, vol. 161, no. 12, pp. 887–892, 1995.
[8] T. Sugiki, T. Hatori, T. Imaizumi et al., "Two cases of hemosuccus pancreaticus in which hemostasis was achieved by transcatheter arterial embolization," Journal of Hepato-Biliary-Pancreatic Surgery, vol. 10, no. 6, pp. 450–454, 2003.
[9] V. Vimalraj, D. G. Kannan, R. Sukumar et al., "Haemosuccus pancreaticus: diagnostic and therapeutic challenges," HPB, vol. 11, no. 4, pp. 345–350, 2009.
[10] J. P. Arnaud, R. Bergamaschi, V. Serra-Maudet, and C. Casa, "Pancreatoduodenectomy for hemosuccus pancreaticus in silent chronic pancreatitis," Archives of Surgery, vol. 129, no. 3, pp. 333–334, 1994.
[11] B. Risti, B. Marineck, R. Jost, M. Decurtins, and R. Ammann, "Hemosuccus pancreaticus as a source of obscure upper gastrointestinal bleeding: three cases and literature review," American Journal of Gastroenterology, vol. 90, no. 10, pp. 1878–1880, 1995.
[12] A. El Hamel, R. Parc, G. Adda, P. Y. Bouteloup, C. Huguet, and M. Malafosse, "Bleeding pseudocysts and pseudoaneurysms in chronic pancreatitis," British Journal of Surgery, vol. 78, no. 9, pp. 1059–1063, 1991.
[13] B. E. Stabile, S. E. Wilson, and H. T. Debas, "Reduced mortality from bleeding pseudocysts and pseudoaneurysms in chronic pancreatitis," British Journal of Surgery, vol. 86, no. 1, pp. 29–32, 1999.
[14] C. A. Benz, P. Jakob, R. Jakobs, and J. F. Riemann, "Hemosuccus pancreaticus—a rare cause of gastrointestinal bleeding: diagnosis and interventional radiological therapy," Endoscopy, vol. 32, no. 5, pp. 428–431, 2000.
[15] M. De Perrot, T. Berney, L. Bühler, X. Delgadillo, G. Mentha, and P. Morel, "Management of bleeding pseudoaneurysms in patients with pancreatitis," British Journal of Surgery, vol. 86, no. 1, pp. 45–51, 1993.
[16] J. Koizumi, S. Inoue, H. Yonekawa, and T. Kunieda, "Hemosuccus pancreaticus: diagnosis with CT and MRI and treatment with transcatheter embolization," Abdominal Imaging, vol. 27, no. 1, pp. 77–81, 2002.
[17] H. Bergert, I. Hintescher, S. Kersting, J. Leonhardt, A. Bloomthal, and H. D. Saeger, "Management and outcome of hemorrhage due to arterial pseudoaneurysms in pancreatitis," Surgery, vol. 137, no. 3, pp. 323–328, 2005.
[18] J. L. Bohl, L. A. Dossett, and A. M. Grau, “Gastrodouodenal artery pseudoaneurysm associated with hemosuccus pancreaticus and obstructive jaundice,” Journal of Gastrointestinal Surgery, vol. 11, no. 12, pp. 1752–1754, 2007.

[19] L. P. Gambiez, O. J. Ernst, O. A. Merlier, H. L. Porte, J. P. M. Chambon, and P. A. Quandalle, “Arterial embolization for bleeding pseudocysts complicating chronic pancreatitis,” Archives of Surgery, vol. 132, no. 9, pp. 1016–1021, 1997.

[20] H. Akpinar, O. Dicle, E. Ellidokuz, A. Okan, Y. Gökçay, and E. Tankurt, “Hemosuccus pancreaticus treated by transvascular selective arterial embolization,” Endoscopy, vol. 31, no. 2, pp. 213–214, 1999.

[21] E. Lermite, N. Regenet, J. J. Tuech et al., “Diagnosis and treatment of hemosuccus pancreaticus: development of endovascular management,” Pancreas, vol. 34, no. 2, pp. 229–232, 2007.

[22] J. S. Bender, D. L. Bouwman, M. A. Levison, and D. W. Weaver, “Pseudocysts and pseudoaneurysms: surgical strategy,” Pancreas, vol. 10, no. 2, pp. 143–147, 1995.

[23] D. I. Heath, A. W. Reid, and W. R. Murray, “Bleeding pseudocysts and pseudoaneurysms in chronic pancreatitis,” British Journal of Surgery, vol. 79, no. 3, article 281, 1992.

[24] F. Boudghene, C. L’Hermine, and J. M. Bigot, “Arterial complications of pancreatitis: diagnosis and therapeutic aspects in 104 cases,” Journal of Vascular and Interventional Radiology, vol. 4, pp. 551–558, 1993.

[25] G. Singh, D. R. N. Lobo, A. Jindal, R. K. Marwaha, and S. K. Khanna, “Splenic arterial hemorrhage in pancreatitis: report of three cases,” Surgery Today, vol. 24, no. 8, pp. 752–755, 1994.

[26] A. C. Waltman, P. R. Luers, C. A. Athanasoulis, and A. L. Warshaw, “Massive arterial hemorrhage in patients with pancreatitis. Complementary roles of surgery and transcatheter occlusive techniques,” Archives of Surgery, vol. 121, no. 4, pp. 439–443, 1986.

[27] M. Udd, A. K. Leppäniemi, S. Bideli, P. Keto, W. D. Roth, and R. K. Haapaiainen, “Treatment of bleeding pseudoaneurysms in patients with chronic pancreatitis,” World Journal of Surgery, vol. 31, no. 3, pp. 504–510, 2007.