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Atrio-oesophageal fistula: dismal outcome of a rare complication with no common solution

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Abstract

Atrio-oesophageal fistula caused by diffusion of ablative energy through the left atrial wall to the oesophagus is a lethal complication. A high level of suspicion, rapid diagnosis and early correction are of crucial importance. In this review, we highlight the key points that will aid in the thought processes and provide guidance in the prevention, early diagnosis and management to reduce complications and improve outcome when atrio-oesophageal fistula is suspected.

Keywords: Atrio-oesophageal fistula • Left atrium • Radiofrequency ablation

INTRODUCTION

Atrio-oesophageal fistula (AOF), first reported in 1970 [1], is an uncommonly encountered condition that has been shown to be quickly lethal, with a mortality rate of ∼80%. Iatrogenic-induced AOF develops after radiofrequency ablation (RFA) beyond the atrial wall and into the nearby oesophageal tissue along the posterior left atrium (LA) in both intraoperative open-heart procedures and percutaneous catheter ablation procedures.

The incidence of AOF following percutaneous ablation ranged from 0.01 to 0.2% [2–8], whereas it has been as high as 1–1.5% for patients undergoing surgical ablations [4, 9] including the MAZE procedure [10]. In addition, the concomitant combined surgical endocardial ablation approach was reported recently to increase the incidence of complications in patients with AOF by as much as 4% [11].

In a nationwide survey on the prevalence of AOF after LA radiofrequency catheter ablation, AOF was reported in 6 of the 20 425 patients who underwent an LA ablation procedure (0.03%). All 6 patients suffered from major cerebrovascular events. Five of the 6 patients died (83%). Based on the responses to the survey, the risk of AOF appears to be <1%. However, AOF is associated with major cerebrovascular events and leads to death in >80% of the patients [5]. In Canada, the reported prevalence of atrial fibrillation (AF) ablation-related AOF is ∼0.07%. Operator use of general anaesthesia (GA) and of the non-brushing technique was reported to have been associated with occurrence of AOF [12].

Postablative AOF first appeared in the surgical literature in 2001 [13] and in the medical literature in 2004 [6]. The Cleveland Clinic was first to report 9 cases in 10 years, with 100% mortality for this complication [14]. However, there have been isolated reports of survivors after the operation, but the overall mortality rate has been reported to be ∼80%, and most survivors had significant permanent neurological deficits [5].

A recent multicentre, retrospective study reported 32 deaths after catheter ablation of 45 115 AF procedures (0.098%). AOF was the second most frequent cause of death after tamponade (15.6%), and the overall mortality rate of AOF was 71.4% [2]. Tamponade, presumably due to perforation into the pericardial space, has been the most commonly reported complication of percutaneous radiofrequency ablation, with an incidence ranging from 0.6 to 1.3% [2, 3, 15].

In this review, we examine the current evidence accumulated over the last decade and highlight important facts in regard to clinical presentations, diagnostic investigations and understanding of the underlying pathophysiology. We specifically focused our attention on the management of this rare condition with special reference to an appropriate and timely surgical intervention.

CLINICAL FEATURES AND DIAGNOSTIC INVESTIGATIONS

AOF has been diagnosed as soon as 2 days post-procedure to as late as 6 weeks postablation [16, 17]. In patients presenting with meningitis, stroke, seizures, or impaired consciousness and fever, it should be determined whether they had RFA and AOF previously, especially if there are also symptoms such as dysphagia or chest pain. After RFA, the patient, his or her family, and his or her treating physicians should be informed about the signs of AOF, which may occur even weeks after RFA [18].

Although reports of AOF have been documented in clinical journals and in the cardiothoracic radiology literature, there has been

little focus on the neurological manifestations and imaging findings of this entity [19]. Cerebral air embolism is often an iatrogenic complication of an invasive procedure. However, most air emboli are caused by the introduction of gas into the venous system as a result of trauma, central line placement, or cardiothoracic or neurological surgerical procedures. The mechanism by which air enters the systemic circulation may relate to an anatomic cardiac defect, or in certain conditions, it may involve oxygen toxicity and excessive volumes of gas, either of which can lead to the passage of air bubbles through the pulmonary vasculature. The classic course of arterial air emboli is to enter the first major branch off the aorta, the innominate artery, and to proceed to the right carotid circulation.

An oesophagogram with thin barium or water-soluble contrast medium may show extravasation of material in communication with the atrium. The oesophagogram should be followed immediately by a computed tomographic (CT) scan of the chest, which may be diagnostic if air is visualized in the mediastinum or heart, or if intravenous contrast medium enters the oesophagus from the LA.

Differential diagnosis between oesophagopericardial and oesophagoatrial fistulas is based on the clinical criteria. Oesophagopericardial fistulas are characterized by non-specific thoracic complaints resulting from pericardial effusion and pericarditis accompanied by signs of infection. Non-specific symptoms along with severe chest or epigastric pain and fever combined with leucocytosis have been described [4, 14]. Furthermore, direct connection of the oesophageal lumen with the LA might result in additional severe complications like embolization of air or food components resulting in neurological complications. Furthermore, pericardial effusion as detected by transthoracic echo (TTE) or CT should raise suspicion of oesophagopericardial communication. Limited communication between the LA and the pericardium might result in severe pericardial but not endovascular complications such as stroke or infection. As such, oesophago-pericardial fistula might be successfully managed with oesophageal stenting (Fig. 1).

It is important also to consider AOF in the differential diagnosis of patients presenting with upper gastrointestinal (GI) bleeding with or without neurological symptoms who have undergone recent RFA therapy for AF. The diagnosis may be made with endoscopic examination of the upper gastrointestinal tract. However, minimal air insufflation during endoscopy or deferral of endoscopy until AOF is ruled out is advisable to prevent possible air emboli through an AOF [20].

Noninvasive imaging such as magnetic resonance imaging (MRI), TTE or CT is preferable to endoscopy. Thoracic CT scan with water-soluble contrast material appears to be particularly helpful for identifying the fistula and pneumomediastinum.

Thoracic or cardiac CT scanning demonstrating the presence of pneumomediastinum or intra-atrial air has been the most reliable tool for the diagnosis of AOF [4]. Rarely, the patient might have an unremarkable initial CT scan [14], but it is unusual to require repeat scans to establish the diagnosis [21]. A CT with intravenous and water-soluble oral contrast material should further be performed to exclude oesophagoatrial communication, intravascular air and potential embolic lesions, because in these cases transoesophageal echo (TOE) or endoscopy should be avoided to prevent air insufflation with fatal consequences [4, 22, 23]. Once a diagnosis is made, immediate surgical intervention is required for this rare complication that has extremely high rates of morbidity and mortality [24].

Thus, any patients who have undergone catheter ablation along the posterior aspect of the LA who present with a clinical picture of endocarditis should have AOF excluded. Echocardiography has not been a useful diagnostic tool for patients with AOF, and it did not identify AOF in any patients for whom the procedure had been performed [25]; however, it is important to exclude endocarditis. AOF patients occasionally have bacteraemia and have been misdiagnosed with endocarditis [25], or they could present with images of coronary air embolism in gas-containing endocarditis caused by AOF after catheter ablation for AF [26].

In an exceptionally rare case of an AOF, which was diagnosed during a forensic post-mortem examination, the authors did not manage to identify the aetiology of the fistula. The possible iatrogenic aetiology of the AOF was only implied [27].

So what diagnostic tests should be considered safe and what tests should be off-limits and not performed in patients with prior ablation procedures presenting with systemic symptoms? Barium swallow is to be avoided because of the detrimental effects of barium in the circulation. Likewise, endoscopy and TOE should be avoided, as they may increase fistula size and the risk of food or air embolism secondary to instrumentation and insufflation. If a fistula is present, instrumentation of the oesophagus may cause rapid deterioration and even death, as highlighted in previous surgical cases. Similarly, oesophagoscopy is contraindicated, because gas insufflation may result in a massive air embolism and/or barotrauma to the damaged tissue, resulting in

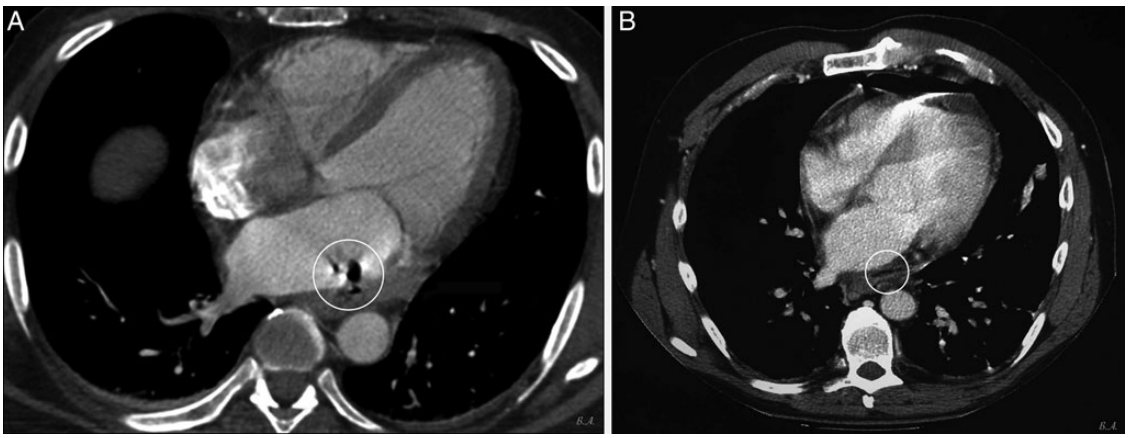

Figure 1: CT scan. (**A**) Contrast-enhanced chest CT scan shows a bubble of air in the LA and a smaller amount of extra cardiac air along the anterior wall of the oesophagus consistent with an AOF. (**B**) CT scan of the chest revealed air and fluid in the pericardium anterior to the oesophagus. LA: left atrium; CT: computed tomography; AOF: atrio-oesophageal fistula.

massive haemorrhage. CT scan and TTE are the two least invasive and safest tests [6, 22].

PATHOPHYSIOLOGY

An oesophageal injury following RFA for AF is classified into three different classes of injury [28] (Fig. 2):

Class I: Patients with this injury have erythema or discolouration of the anterior oesophageal wall with minimal mucosal disruption. These lesions should be shallow erosions without vessel involvement and should be less than 5 mm. These patients appear to have a low risk of frank perforation, and they can be managed with proton pump inhibitors and sucralfate, with clinical follow-up to assess for worsening symptoms.

Class II: Patients with this degree of injury have ulcers of the anterior oesophageal wall with or without exudate. These lesions tend to penetrate further into the oesophageal wall, without extension into the muscularis externa. In addition, these lesions should not have an overlying clot or vessel involvement. Patients with this injury appear to have an intermediate risk of developing a perforation. In addition to medical management with proton pump inhibitors and sucralfate, these patients require close follow-up to ensure lesion improvement.

Class III: Patients with this degree of injury have deep ulceration that extends into and beyond the muscular layer. These lesions can have evidence of eschar formation, overlying clot or necrosis. The lesions may also have vascular involvement. As a result, there is an increased probability of compromising tissue blood flow and worsening injury. Patients with this injury have a high risk of developing an oesophageal perforation and AOF. They should be considered nil per os and started on medical therapy. Cardiothoracic surgery consultation is warranted as well as a CT scan of the chest.

It is important to recognize the critical concept that oesophageal intraluminal pressures tend to be higher than left atrial pressures during retching, vomiting and coughing, resulting in food, bacteria and air entering the heart and causing embolization to the brain.

One hypothesis about the air emboli is the capability of the oesophagus to develop pressures 10 times greater than intra-atrial pressures [29]. Although the left atrial pressure is higher at rest, exsanguination from AOF is uncommon because the resistance to

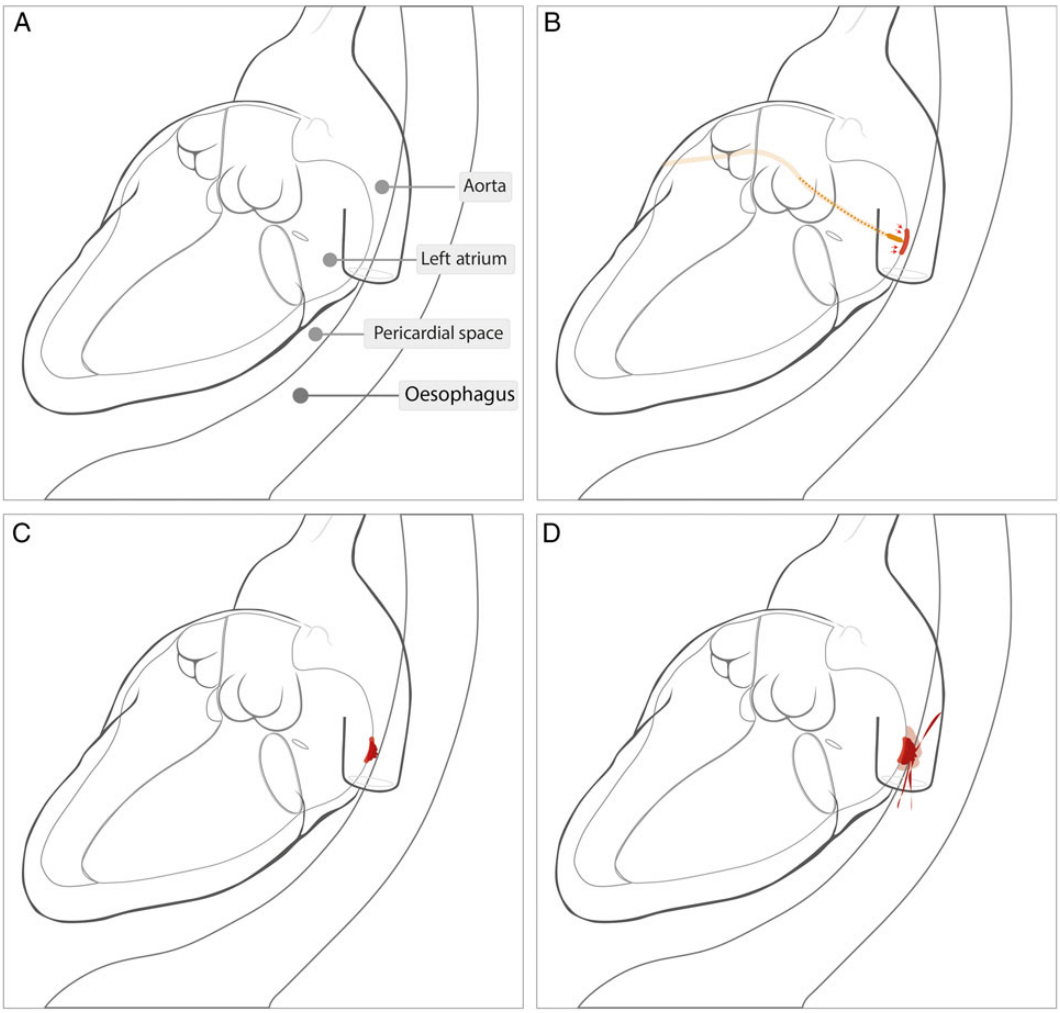

Figure 2: Possible natural history of oesophageal injury and AOF formation. (**A**) Right lateral view of atrial oesophageal junction. (**B**) Ablation is done on the posterior wall with possible thermal injury to the anterior oesophageal wall. (**C**) In some instances, this leads to oesophageal ulcer formation and perforation. (**D**) Digestion of the oblique sinus parietal layer results in communication between the oesophagus and the pericardial space with resulting pneumopericardium and effusion. AOF: atrio-oesophageal fistula.

flow across the fistula is high, and air or food emboli are more likely to occur [22]. Success in saving these critically ill patients rests with early closure of the LA communication before any surgical manipulation of the oesophagus.

RISK FACTORS

It has been shown via cadaveric studies that the oesophagus may come within 5 mm of the left atrial endocardium at some point along its course [30]. Therefore, RFA lesions pose an obvious risk of oesophageal injury. Furthermore, it has been shown that intestinal tissue (oesophagus being part of the aerodigestive system) is far more susceptible to RFA-induced thermal injury than skeletal muscle tissue [9]. It has been noted that irrigated catheters reduce the formation of coagulum and steam pops known to cause cardiac damage and perforation. However, the lesions formed by an irrigated catheter have a teardrop shape, with the largest lesion projecting more deeply into the tissue. The geometry and the greater fragility of oesophageal mucosa provide an explanation for ulcers forming in the oesophagus and extending towards the LA.

Propagation of the ulcer into the deeper tissue may be facilitated by the other effects of RFA energy such as damage to the arterial supply to the oesophagus (on the anterior wall of the oesophagus behind the LA), which could impair healing. Further, injury to adjacent vagal nerves or perioesophageal tissue may allow reflux, as vagal tone is needed for the function of the lower oesophageal sphincter [31].

Detailed studies of the anatomic relation between the oesophagus and the LA demonstrated that the distance between the two structures could often be less than 5 mm, well within the reach of lesions created by radiofrequency energy application. AOF development was exclusively associated with the use of an 8-mm tip ablation catheter, which at the time was the most commonly used catheter for LA ablation procedures to eliminate AF [32].

Animal studies have demonstrated that intestinal mucosal tissue is more susceptible to RFA-induced thermal injury than is muscle tissue [9, 33]. A recent report has shown that mucosal damage originates in the oesophagus and progresses to the atrium [33]. All the reported cases of bacteraemia have been caused by organisms that originate in the oropharynx, which supports the concept of dissemination from the proximal GI tract through the AOF.

The atrial dimensions might play a role especially if relatively small, and it is possible that their size predisposes them to fistula formation [6]. Other factors such as a thin atrial wall resulting from atrial enlargement (greater than 60 mm in diameter), female gender, higher energy settings, cachectic patients, who are likely to have a thin LA wall with very little tissue between the LA and the oesophagus, were the suspected culprits for the development of fistula [34].

The type of anaesthesia used during ablation procedures has been considered to predispose the patient to oesophageal injury during the ablation procedure. Interestingly, one randomized study involving percutaneous RFA showed a higher risk of oesophageal injury with GA in comparison to conscious sedation [35]. The group randomized to GA had significantly less time to peak temperature and higher maximum oesophageal temperature. This was attributed to reduced oesophageal motility from lack of swallowing ability, resulting in heat transfer to the same areas of the oesophageal wall. One study reported a sideways shift of at least 2 cm in the oesophagus secondary to peristalsis during LA catheter ablation performed with the patient under conscious sedation and recommended real-time imaging using barium and a fluoroscope to avoid ablation at sites in contact with the oesophagus [36].

PREVENTION OF INJURY

Several approaches are used during RFA to avoid oesophageal injury although no data are yet available on these approaches. In addition, several techniques have been used to control RFA and limit the lesion to tissues intended for ablation; these include modulation of power and time, orientation of the catheter and monitoring the oesophageal temperature. These approaches and techniques unfortunately remain theoretical due to the fact that complication rate is so rare [33].

Because no technique or ablation strategy has been shown to unequivocally eliminate the risk of AOF, a safe approach for the time being may be to avoid applications of energy over the oesophagus. This approach was put into practice by asking the patient to swallow a thick barium paste, which facilitated visualization of the oesophagus on the fluoroscope. This approach was permitted monitoring for oesophageal migration during the procedure, which has been recognized to occur in up to 50% of patients [36].

Monitoring oesophageal intraluminal temperature has been proposed as a method to protect against oesophageal injury. However, this approach may be limited by the difficulty of achieving adequate contact between the tissue and the temperature probe and the need to position the probe in the immediate proximity of the ablation catheter [37].

Certain measures have been suggested to diminish the risk of developing AOF [2, 3, 7, 15]. These measures might have resulted in reduced incidence of AOF at several institutions, but it is likely that this complication will continue to occur given that RFA is being performed more frequently for more indications at more institutions [25].

Radiofrequency generator settings and AF lesion tools for AF ablation have evolved through experience, reaching a point at which the procedure can now be safely performed in a relatively short period of time with success rates of between 80 and 90% [38, 39]. It is recommended that lower generator settings of 50 W and 55°C be used during ablation in the posterior wall to avoid excessively deep lesions and subsequent oesophageal injury. In addition, the transverse posterior line should be placed at the roof, where the atrium tends to be thicker and is not in direct contact with the oesophagus [6].

The bipolar technique in comparison with unipolar radiofrequency is limited in its ability to reach all portions of the LA. However, it focuses its energy between the two conduction probes, which theoretically prevents energy dispersion and thus the formation of AOF. It appears to be a safe alternative to unipolar surgical atrial ablation [6].

LA wall thickness, as discussed before, varies considerably [40]. Overlapping lines in the posterior wall of the LA might have been responsible for oesophageal injury after intraoperative RFA of AF [34].

Even in the absence of oesophageal perforation, post-RFA patients commonly complain of GI symptoms commonly attributed to the procedure with resultant GI gastroparesis or mediastinal and oesophageal structural changes [41, 42].

MANAGEMENT OF ATRIO-OESOPHAGEAL FISTULA

There has not been a uniform approach to either the diagnosis or corrective therapy of AOF, and management options previously reported include oesophageal stenting and direct intracardiac or transthoracic extracardiac repair with or without cardiopulmonary bypass [21, 22, 43]. Antimicrobial therapy alone appears not to

improve the situation, and widespread gaseous or septic embolization will continue without prompt intervention. Definitive treatment should be expedited once the diagnosis has been confirmed, otherwise rapid deterioration and death are the more likely outcomes.

NON-SURGICAL MANAGEMENT

It was believed that the degree of fragility of a large portion of the LA wall and probable lack of healthy tissue for adequate closure precluded primary repair. Some had suggested conservative management, but the mortality rate is extraordinarily high. Of course, AOF and/or oesophageal damage may be present in some individuals for which this approach may be the most appropriate [44].

Stent deployment as treatment of AOF, although controversial due to the associated risk of stent placement, which in itself may lead to increased chance for air embolization, has been suggested for consideration in some scenarios [45]. Bunch *et al*. reported a successful case of temporary oesophageal stenting to allow healing of oesophageal perforation. A self-expanding plastic stent was used to urgently treat the oesophageal perforation. Such stents have been used for oesophageal stenosis fistulas and leaks from other causes [43]. It is important to note that most authorities in the field recommend avoidance of oesophagoscopy [6, 14, 34], the case in point being a fatal outcome after an oesophageal stent in AOF [23]. Interestingly, the authors reported the use of carbon dioxide instead of air during insufflation, yet the outcome was that of post-procedural neurological dysfunction followed by death.

Even after initial successful deployment, stents have been shown to migrate, thus eliminating the hypothetical value intended [46]. To the best of our knowledge, all published cases of stenting for proven AOF have been fatal [23], and it is unknown whether death with stenting is related to the procedure itself (due to the oesophageal instrumentation including air insufflations), the inherent lack of efficacy of stenting (due to ongoing communication between the atrium and adjacent structures) or inherent patient morbidity (secondary AOF-related stroke or mediastinitis) that might have precluded surgical repair. Furthermore, it remains unknown whether a role for stenting as a bridge to definitive surgical repair exists, but it is possible that stenting may be a temporary solution to facilitate transfer to a surgical centre experienced in managing AOF.

Another treatment option reported to eliminate intravascular air embolus is using hyperbaric oxygen, which diminishes gas volume and cerebral oedema and enhances the partial pressure of dissolved oxygen in the blood. This approach could be potentially beneficial and is indicated in the presence of neurological deficits caused by gaseous emboli [19].

Steroid use was previously proposed; however, there is a small but independent increase in peptic disease with the use of glucocorticoids with estimated relative risks anywhere from 1.1 to 1.5 [47]. The potent anti-inflammatory effects likely precluded optimal healing of the oesophageal ulcer. Perhaps less focus on the prevention of ulcer formation and more on allowing healing to occur is what really is needed.

Another proposed endoscopic approach requiring snaring of the oesophageal mucosa was recently suggested. It could be repeated several times, supported by nil per os and antibiotic therapy, and it could result in the improvement of the underlying condition with no recurrence of symptoms [48].

In summary, the potential value and safety of the non-surgical approaches are unclear. More data would be needed to determine which cases are indicated for conservative non-surgical intervention. At present, these approaches are of questionable and untested value.

SURGICAL MANAGEMENT

Scattered reports in the literature suggest that surgical intervention can be effective [49, 50], but the risks remain high, making surgery an inappropriate option according to many surgeons.

Cazavet and colleagues described a successful outcome after the operation was performed via a left thoracotomy. The choice of the left-sided approach was based on the finding of left pleural effusion, and the left femoral vessels were accessible in case cardiopulmonary bypass support was required [22]. However, access to the oesophagus from the right side may be more feasible for greater exposure of the oesophagus, without being encumbered by the arch of the aorta in the left chest. Tang *et al*. described the rational of right posterolateral access based on the location of the problem identified on MRI studies. Right thoracic access provided the greatest exposure for central cannulation for cardiopulmonary bypass and access to the LA and oesophagus in emergency settings [51] (Fig. 3).

The type and extent of oesophageal repair are chosen according to the local severity of necrosis and mediastinitis. Flap coverage is highly recommended to separate the atrial suture line from the oesophageal suture line. A viable muscle flap brings fresh blood along the skeletal muscle tissue to the injured cardiac and oesophageal tissue to facilitate wound healing. The choices of flaps vary with no particular difference and depend on surgeon preference and level of experience. In cases of pediculated autologous pericardial patch with or without cardiopulmonary bypass [22, 51], the repair could be buttressed with an intercostal muscle flap [49], or omental wrap supported with decompressive gastrostomy and feeding jejunostomy [52].

In our experience, we have favoured a pedicled intercostal muscle flap, mobilized off the intercostal space, while maintaining its blood supply via the intercostal arterial branches off the thoracic aorta. If the LA wall has a large necrotic area and cannot be closed primarily, bovine pericardial patch closure under cardiopulmonary bypass has been used [53].

The use of cardiopulmonary bypass support for the repair of AOF, however, has raised concerns for increased risk of embolization and bleeding [6, 34, 51, 53].

Upon completion of repair, intraoperative epicardial ultrasound has been suggested to be a beneficial approach to assess whether there is any air, blood clot or gastric content in the heart [54].

An alternative surgical approach, presumably less invasive yet with the reported successful management of AOF, is performing cervical oesophageal ligation and decompression, along with gastric drainage [50], thus avoiding open chest procedures, avoiding CPB and performing efficient neck-accessed oesophageal drainage procedure and gastric drainage and enteral feeding access port.

Despite numerous isolated successful surgical reports, managing AOF remains a formidable problem that requires early diagnosis, prompt surgical intervention and appropriate antibiotic if there is any hope for patient salvage [53].

It is concluded that when clinical factors permit, surgical exploration with definitive repair is strongly advocated as soon as the diagnosis of AOF is established with variations of a left or right thoracotomy, atrial and oesophageal repair with interposition flap and mediastinal drainage.

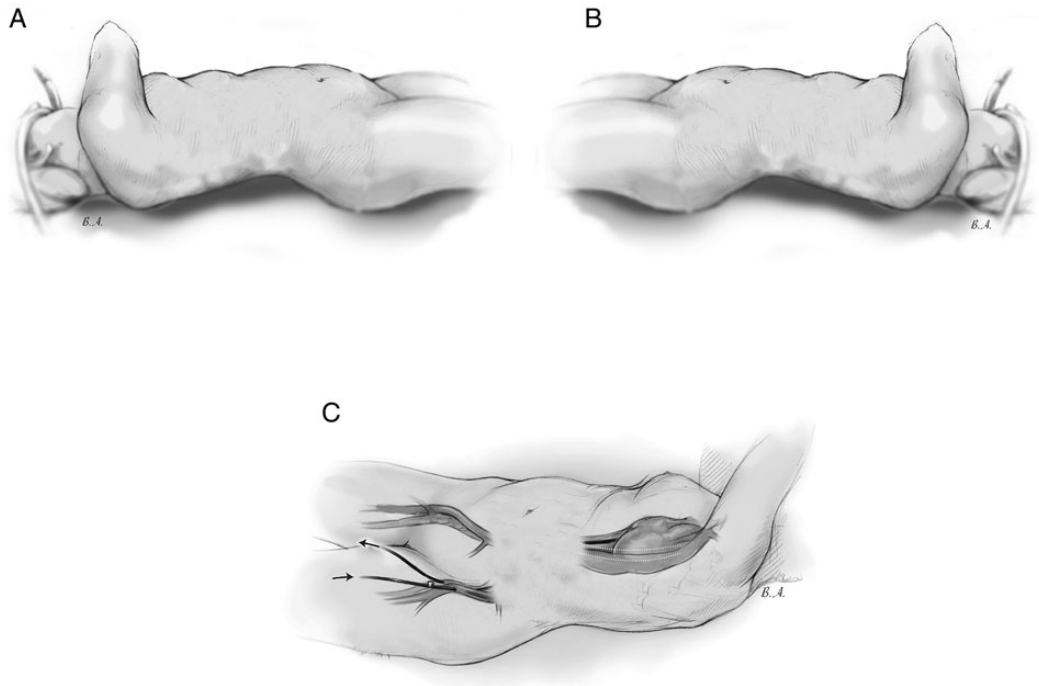

Figure 3: Surgical access. (**A**) Right thoracotomy position. (**B**) Left thoracotomy position. (**C**) Femoral access suggested for cardiopulmonary bypass.

COMMENT

AOF caused by diffusion of ablative energy through the LA wall to the oesophagus is lethal if not treated. A high index of suspicion is necessary after ablation. Rapid diagnosis and surgical correction are crucial for better outcomes. The key to managing these patients safely is preventing massive bleeding and multiple air embolization during the surgical repair, followed by the postoperative management of sepsis and multiple organ failure that commonly contribute to the increased risk of death from this complication.

Paramount to the management of those patients is a high index of suspicion in at-risk patients who received ablative procedures, followed by early radiological confirmatory diagnosis. The presence of air emboli in the heart or brain is sine qua non for AOF in postablation patients. Once radiological diagnosis is made, further pursuit of diagnostic testing will delay only the definitive surgical intervention. If endoscopy and TEE are done, they may even contribute to early death. A chest CT scan is perhaps the most useful diagnostic method and specifically shows air bubbles or a fistulous tract. TTE is useful to assess the absence of an endocarditis lesion. TEE and endoscopy are contraindicated considering the risk of air embolism [9]. Although the complication rate from RFA is low, when complications are suspected, CT imaging is very helpful as a problem-solving tool. Accordingly, radiologists should be aware of this potential complications and the role of CT scanning [16].

Oesophageal stenting in patients with suspected AOF is fraught with problems and is strongly discouraged. The preferred alternative that has been shown to be crucial for survival in patients with AOF [55] is prompt surgical repair. Patients occasionally have bacteraemia and have been misdiagnosed with endocarditis. Infectious disease specialists must have a high index of suspicion for this diagnosis for patients who develop postablative fever, chest discomfort, unexplained neurological deficits and occasionally bacteraemia for 5 weeks after the procedure. Safer ablation recommendations employ lower generator settings as well as target thicker LA areas not in direct contact with the oesophagus [6].

Oesophageal lesions associated with the AF ablation procedure are often transient and asymptomatic and heal without consequence. Non-invasive techniques such as delayed-enhancement MRI scans can be used to determine oesophageal injury after RFA. Acute oesophageal injury or inflammation may manifest as oesophageal ulceration in the immediate postablation state with resolution of the findings by later follow-up. The risk factors for the development of oesophageal ulcerations are LA size, distance between LA and oesophagus, and the set of ablation lines. Most patients with oesophageal ulcers are asymptomatic, and universal postablation acid suppression may be of benefit in healing those asymptomatic ulcers by minimizing gastric acid exacerbation of thermal injury [56].

The use of temperature probes in pulmonary vein isolation and cryoballoon ablation is not currently mandated or recommended by the manufacturer. Temperature monitoring of the oesophagus may be helpful to identify potentially dangerous lesions, but additional studies are warranted to better define safe levels of oesophageal temperature drops during lesion delivery and to examine the efficacy of abridged lesions when oesophageal proximity is an issue. Such studies would serve the dual purpose of defining methods for preventing this catastrophic complication and possibly leading to earlier detection if any symptoms occur after ablation.

Asymptomatic patients may benefit from routine prophylactic endoscopic examinations, but clinical and prognostic consequences have to be assessed critically. The benefits should be examined in further studies. Nevertheless, it is essential that interventional cardiologists and gastroenterologists work together closely to detect incidental and relevant GI findings to provide patients with the appropriate therapies [42].

Finally, we believe that raising awareness of AOF is important to prompt changes in the management of RFA patients. The work of the Canadian Heart Rhythm Society is a great example in this regard, highlighting the importance of national collaboration [57]. We hope the key points presented in this review will aid in the thought processes and provide guidance in the prevention, early diagnosis and management of AOF in an attempt to reduce complications and improve outcome.

Conflict of interest: none declared.

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