Is abdominal aortic aneurysm repair appropriate in oxygen-dependent chronic obstructive pulmonary disease patients?

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Background: **The life expectancy of patients with oxygen-dependent chronic obstructive pulmonary disease (COPD) is significantly reduced, but the risk of any intervention is considered prohibitive. However, severe COPD may increase the risk of abdominal aortic aneurysm (AAA) rupture. We reviewed our experience with AAA repair in oxygen-dependent patients to determine whether operative risk and expected long-term survival justify surgical intervention.**

Methods: **A retrospective review of 44 consecutive patients with oxygen-dependent COPD undergoing AAA repair over an 8-year period was performed. Information was recorded for survival, length of follow-up, patient age, medical comorbidities, pulmonary function tests, and operative approach. Survival data were analyzed by Kaplan-Meier curves and compared with published cohorts of oxygen-dependent patients and the natural history of untreated aneurysms.**

Results: **Twenty-four patients underwent endovascular aneurysm repair (EVAR), and 20 underwent open procedures (14 retroperitoneal and 6 transabdominal). The mean AAA diameter was 6.1 cm (range, 5-9.5 cm). The mean age was 71.4 years, and 82% of patients were male. Operative mortality was 0%. The mean length of stay was 11.2 days for open procedures and 4.3 days for EVAR (significantly longer than that for standard-risk patients). The mean survival time was 37.9 months (range, 2-91 months). Preoperative medical comorbidities, type of repair, and pulmonary function tests were not predictive of survival. Postoperative morbidity was significantly higher with open repair. Long term survival was comparable to historical series of the natural history of O2 dependent patients without AAA but better than untreated 6 cm AAA cohorts. At 42 months, almost 50% of patients in our study group were still alive, compared to 20% survival at 34 months for those with untreated 6 cm AAAs.**

Conclusions: **It is reasonable to continue to offer AAA repair to home oxygen–dependent COPD patients who are ambulatory and medically optimized and who are without untreated coronary artery disease. Although EVAR may be the most suitable treatment for oxygen-dependent COPD patients, our results show that even open repair may be safely performed in this population, with acceptable results. (J Vasc Surg 2005;42:650-3.)**

A significant comorbidity associated with poor outcome for any major surgical procedure has been dependency on home oxygen and severe chronic obstructive pulmonary disease (COPD).[1,2] In addition, this condition is known to be associated with an increased prevalence of abdominal aortic aneurysm (AAA)[3,4] and is an independent predictor of AAA rupture.[5] Thus, patients with severe COPD and AAA are at an increased risk of rupture and have an apparently higher risk for any type of intervention. The purpose of this study was to determine whether the long-term survival of patients with home oxygen–dependent COPD undergoing AAA repair (compared with the natural history of AAA and home oxygen dependence) justifies intervention.

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METHODS

Study group. All patients with home oxygen–dependent COPD who underwent elective infrarenal AAA repair in the last 11 years at the University of Pittsburgh Medical Center were retrospectively reviewed. From 1992 to 2003, 44 consecutive patients were identified at our institution. These patients were on home oxygen before surgery and satisfied one or more of the following criteria: a room air P_{O_2} of 60 mm Hg or less, P_{aCO_2} greater than 45 mm Hg, forced expiratory volume at 1 second (FEV_1) less than 50% of predicted, and/or forced vital capacity (FVC) less than 75% of predicted.

Follow-up intervals, survival, preoperative comorbidities, method of repair, length of stay, intensive care unit (ICU) use, and postoperative complications were recorded for all patients. This group was stratified with separate Kaplan-Meier curves. Data were compared via the log-rank test with respect to operative approach to determine whether this was predictive of outcome. The research protocol was reviewed and approved by the Institutional Review Board of the University of Pittsburgh.

Control groups. The long-term survival rate of our cohort was compared with that of two historical control groups from previous reports by using Kaplan-Meier curve estimates. The first control group represented the natural

history and long-term survival of home oxygen–dependent COPD patients.[6] The second comparison group included untreated AAAs in patients who were deemed unfit for intervention or who refused treatment.[7]

Clinical pathway. The previously reported clinical pathway[8] from our institution has been used at our institution since 1993 and was incorporated in the perioperative care of all patient groups. To summarize briefly, the pathway consisted of limited perioperative intravenous fluid administration, epidural analgesia, early extubation, aggressive pulmonary toilet, and early ambulation. The pathway was modified in our current study group slightly by admitting essentially all patients with oxygen-dependent COPD to the ICU after open repair for overnight observation. After endovascular aneurysm repair (EVAR), ICU admission was used only for respiratory compromise. All patients underwent preoperative pulmonary function testing and outpatient medical optimization by a pulmonary specialist. Patients were required to be ambulatory and have no or minimal evidence of reversible ischemia on noninvasive cardiac testing before they were offered therapy.

RESULTS

Of the 44 patients in the study group, 24 underwent EVAR and 20 underwent direct, open procedures. Fourteen of the 20 patients who underwent an open operation had the repair performed by using a limited left flank retroperitoneal approach. Twenty-three patients underwent general anesthesia: 20 open and 3 EVAR. Twenty-one patients underwent epidural anesthesia alone. The mean AAA diameter was 6.1 cm (range, 5-9.1 cm). The median AAA diameter was 6.0 cm (25th percentile, 5.5 cm; 75th percentile, 6.7 cm). The mean age of the patients was 71.4 years; 82% of the patients were male. All patients had Pa_{O_2} less than 60 mm Hg and Pa_{CO_2} greater than 45 mm Hg on room air before surgery, and 79% had FEV_1 less than 50% and FVC less than 75% of predicted on pulmonary function testing.

The average length of stay was 11.2 days for open AAA repair vs 4.3 days for EVAR ($P = .004$). Eighteen (90%) of the 20 open repair patients were admitted to the ICU after surgery as part of the routine pathway, compared with 4 (17%) of 24 for the EVAR group ($P = .001$). Overall, 30% of patients required longer than 24 hours in the ICU. Operative mortality was 0%. The mean actuarial survival was 37.9 months (range, 2-91 months), and the mean follow-up was 23.7 months. There was no difference in survival for open vs endovascular approaches (Figure 1). Five-year survival was 30%; this is in contrast to a 5-year survival of 66% for the general population undergoing elective AAA repair.[9] Age older than 80 years seemed to decrease survival. Postoperative morbidity was predictably higher in the open group than in the EVAR group (Table).

However, the long-term survival for oxygen-dependent patients undergoing AAA repair compared favorably to the natural history of home oxygen dependence[10] (Figure 2. The long-term survival of home oxygen–dependent COPD patients undergoing AAA repair also compared favorably to the natural history of untreated 6-cm aneurysms.[11] Most (80%) of the patients with untreated 6-cm AAAs either experienced ruptured aneurysms or died within 34 months. At 42 months, almost 50% of patients in our study group were still alive (Figure 3).

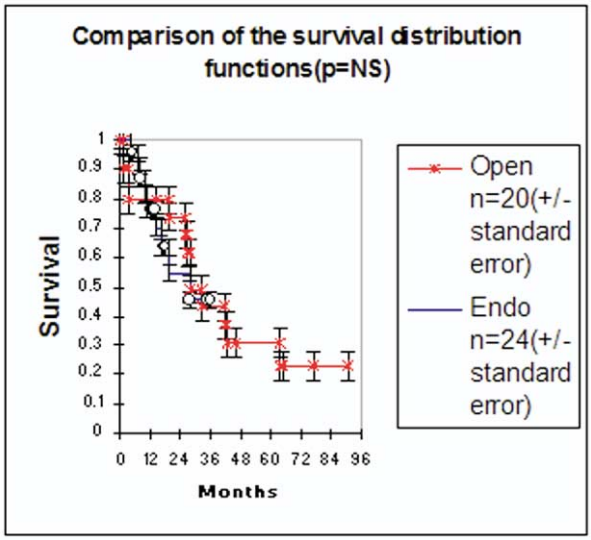

Fig 1. Kaplan-Meier survival analysis for open vs endovascular (Endo) repair of abdominal aortic aneurysm in oxygen-dependent chronic obstructive pulmonary disease patients. *NS,* Not significant.

Table 1. Post-operative morbidity

Morbidity	*Overall*	*Open*	*Endo*
Respiratory Failure	5/44 (11%)	3/20	2/24
Atrial fibrillation	3/44 (7%)	3/20	0/24
Pneumonia	4/44 (9%)	3/20	1/24
Reintubation	4/44 (9%)	3/20	1/24
GI Bleed	1/44 (2%)	1/20	0/24
Urinary Retention	3/44 (7%)	1/20	2/24
Myocardial Infarction	0/44 (0%)	0/20	0/24
Renal Failure	1/44 (2%)	1/20	0/24
ARDS	1/44 (2%)	1/20	0/24
Total	22	16	6

DISCUSSION

The decision to offer elective repair to patients with AAAs hinges on the estimate of the risks and benefits involved. One must weigh the risk of rupture and almost certain death[12] after such an event with the risks associated with elective repair. Most estimates of AAA rupture risk have been based on aneurysm size.[13] The natural history of AAA is still unclear, but recent reports have elucidated some of the trends.[14,15] It is clear that large AAA (>6 cm) poses a significant threat of rupture, and repair should not be denied in good-risk patients.[16] The difficult decision stems from determining whether a patient with a significant morbidity such as COPD would benefit from intervention.

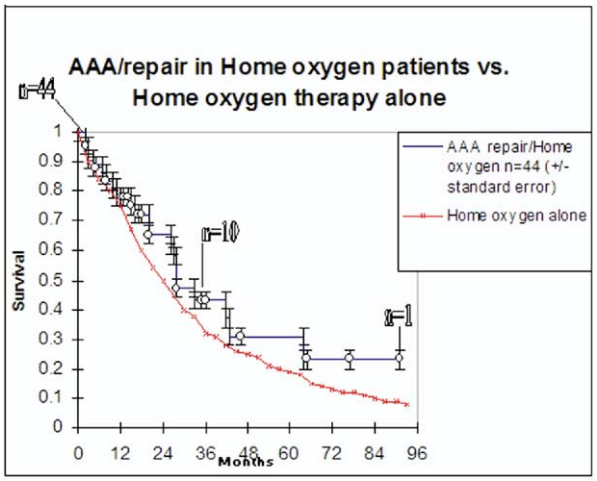

Fig 2. Kaplan-Meier survival analysis of oxygen-dependent chronic obstructive pulmonary disease (COPD) patients undergoing abdominal aortic aneurysm *(AAA)* repair vs the natural history of oxygen-dependent COPD.

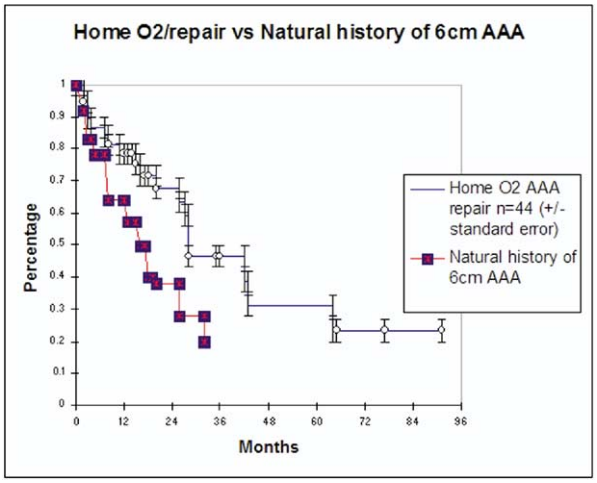

Fig 3. The Kaplan-Meier survival curve is shown for oxygen-dependent chronic obstructive pulmonary disease patients undergoing abdominal aortic aneurysm *(AAA)* repair vs the natural history of untreated 6-cm AAAs and the natural history of home oxygen dependence.

In the future, stress and strength analysis of the AAA wall may provide further information regarding rupture risk by using a more physiologic predictor.[17,18] However, these techniques have not yet been correlated clinically. Until that time, a risk analysis of the patient's comorbidities must be performed to justify the risks involved in the planned intervention.

Clinical factors, such as female sex[19] and pulmonary disease (COPD),[20,21] have been known to increase rupture risk at a given size, but the exact influence has been difficult to quantitate. A University of Michigan study found a 5-year predicted AAA rupture rate of 34% in patients with COPD, compared with only 2% in patients without this condition.

Unfortunately, however, this very group of patients who are at increased risk for rupture and death from AAA disease are often denied treatment because of the perceived increased risk with surgery. Several studies have demonstrated increased morbidity and mortality after AAA repair in patients with COPD.[22,23] It has been suggested that an increased risk of pulmonary complications is associated with a FEV_1 or FVC of less than 70% of the predicted value or a ratio of FEV_1 to FVC of less than 65%.[24] COPD is also associated with increased mortality after coronary artery bypass grafting[25] and laparotomy.[26]

Hence, patients with COPD not only are at increased risk for rupture, but also seem to have an increased risk for intervention. However, most of the literature regarding the risk of severe COPD is written in the setting of lung-reduction surgery or thoracotomy.[27,28]

Ultimately, the risk of intervention depends not only on the method of repair used, but also on the comorbidities present in an individual patient. Of course, the life expectancy of the patient is also important to judge the length of protection afforded by the aneurysm repair and whether it will justify the risks involved in the intervention.

The risk of AAA rupture in patients who are deemed unsuitable for therapy seems to be higher than that for standard-risk patients who are followed up prospectively. A Veterans Affairs Cooperative study that followed up AAA patients who were deemed unfit for or refused repair demonstrated a significant risk of rupture. The annual risk of rupture for aneurysms 5.5 to 5.9 cm in diameter was almost 10% and was 19% for those with diameters 6.5 to 6.9 cm. The all-cause mortality was 29.8% at 1 year, 55.5% at 2 years, and 75.4% at 3 years in this population of patients who were denied AAA repair.[29]

Our current study and our previous report[30] show a very low mortality and morbidity in such patients after AAA repair. It is interesting to note that a recent review also demonstrated that home oxygen use was not found to be predictive of poor outcome in COPD patients undergoing elective AAA repair.[31] With no operative mortality, a mean survival of longer than 3 years, and an average aneurysm diameter of more than 6 cm, our long-term results compared favorably to the natural history of oxygen dependence and untreated AAA disease.

In conclusion, it is reasonable to continue to offer AAA repair to home oxygen–dependent COPD patients who are ambulatory and medically optimized and who are without untreated coronary artery disease. Although EVAR may be the most suitable treatment for oxygen-dependent COPD patients, our results show that even open repair may be safely performed in this population, with acceptable results.[24]

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