

Review Article

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**Short Term and Long Term Complications after Percutaneous Transluminal Coronary Angioplasty (PTCA)**

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**Abstract**

**Background:** The nonsurgical treatment of arteries narrowed by atherosclerosis was introduced in 1964, when Dotter and Judkin performed transluminal angioplasty of femoral artery stenoses[1]. In 1970, Gruntzig modified the dilation catheter to allow its use in coronary arteries dilation [2]. Subsequently, In 1977 he performed the first Percutaneous Transluminal Coronary Angioplasty (PTCA) in a patient. Since onward, PTCA has been used in many patients with stable angina, unstable angina or acute myocardial infarction. Its use was initially limited to the treatment of discrete stenoses in proximal segment of a coronary artery, but improvements in equipments and techniques have lead its use in patients with stenoses, that are more complex or located in distal arterial segments and in patients relatively at high risk for complications[3]. Despite the wider application of PTCA, the success rate remained high. Over the past decade, the number of procedure performed has steadily increased, with over 300,000 performed in united state in 1990[4].

**Introduction**

**Short- Term Complications**

Although PTCA is generally safer, complication occasionally occurs including myocardial infarction in 3 to 5 percent [5,6], Yet it is need for emergency bypass surgery in 3 to 7 percents [7,8] and death in 0 to 2 percent [9,10]. These event usually caused by extensive coronary arterial dissection, intracoronary thrombosis, or both, with resultant vessel occlusion. Coronary arterial perforation, rupture, or remobilization is rare. The last is more likely in a sphenous- vein graft than in a native vessel.

Acute or abrupt closure occurs in 2 to 8 percent of patients undergoing PTCA [11,12], and accounts for most of the short – term morbidity and mortality associated with the procedure. In about 75% of patients with abrupt closure, it occurs within minutes after PTCA, when they are still in the catheterization laboratory; in the other 25 percent it usually occurs within 24 hours after the procedure [13].

**Mechanism of Abrupt Closure**

Three pathophysiologic processes may contribute to the occurrence of abrupt closure after PTCA: extensive dissection, thrombosis, and coronary vasospasm [14]. Some degree of intimal dissection- characterized angiographically as a linear interluminal filling defect or flap associated with a hazy, ground glass appearance- is frequently observed after successful PTCA. When the dissection is limited, its angiographic characteristics may be subtle

and evident only on selected views. However, when it is extensive, the radiographic features are readily apparent, and the vessel lumen may be compromised. 10 % of patients with coronary arterial dissection after PTCA require emergency bypass surgery, sustain a myocardial infarction, or die. In contrast, less than 2 % of patients without extensive dissection have a serious complication [15].

Acute thrombus formation after PTCA is characterized angiographically as an intraluminal filling defect or an area stained with radiographic contrast material. Thrombus formation is most likely to occur in patients with extensive dissection, those with a severe residual stenosis after PTCA, those with pre-existing intracoronary thrombus, and those not receiving an antiplatelet agent. In One study, 22% of the patients undergoing PTCA without antiplatelet therapy had thrombi, demonstrable by angiography after dilation. In half of these patients, the thrombi caused occlusion, requiring repeated PTCA, bypass surgery, or thrombolysis [16]. Conversely, those who received aspirin before PTCA were much less likely to have demonstrable thrombi afterward. Since; most patients receive nitrates, calcium-channel blockers, or both before, and after PTCA, coronary vasospasm is an uncommon cause of abrupt closure.

**Risk Factors for Abrupt Closure**

Several clinical, anatomical, and procedural variables are associated with an increased incidence of abrupt closure after PTCA. The

influence of these variables is cumulative: the more variables that are present in a particular case, the greater the likelihood of abrupt closure.

### Consequences of Abrupt Closure

The consequences of abrupt closure vary widely. Patients with adequate collateral perfusion of the occluded vessel may have abrupt chest pain, electrocardiographic abnormalities, or hemodynamic compromise. More commonly, abrupt closure is accompanied by chest discomfort and electrocardiographic evidence of ischemia and requires immediate revascularization of the occluded vessel to prevent or limit myocardial injury.

The common Variables associated with an increased risk of abrupt closure of the artery in patients undergoing PTCA may be clinical, such as; female sex [17], unstable angina[18], MVD[19] or Anatomical such as; angiographically demonstrable intracoronary thrombus, eccentric stenosis [20], stenosis located at or near a bend or branch, severe pre PTCA stenosis [21], stenosis or 2 luminal diameter in length, sequential stenosis, diffusely diseased artery, procedural variables, extensive coronary arterial dissection and use of oversized balloon[22].

### Management of Abrupt Closure

Since the most effective treatment of abrupt closure is prevention, strategies aimed at precluding its possible causes (dissection, thrombosis, and vasospasm) are routinely instituted. Calcium-channel blockers, nitrates, or both are usually administered before, during, and after PTCA, even though no controlled study has shown that they reduce the incidence of abrupt closure. All patients receive aspirin before and after PTCA, and all are given heparin during the procedure, with the adequacy of anticoagulation monitored. In many centres, patients at high risk of abrupt closure are given infusion of heparin for 12 to 24 hours after PTCA, although the efficacy of this approach has not been proved. Only aspirin has been shown to reduce the incidence of abrupt closure [23].

When abrupt closure occurs, redilation with a standard balloon catheter is usually attempted, which is successful in about 50 percent of patients [ 24]. When this strategy fails, a special balloon catheter may be used, if the coronary anatomy is suitable. Holes in the catheter proximal and distal to the balloon allow blood to flow through the catheter during inflation of the balloon, thus maintaining perfusion of the distal artery during prolonged inflation. In the majority of patients in whom a standard balloon catheter fails to restore sustained antegrade perfusion, a perfusion catheter is effective.

Peripheral vascular complications, such as arterial pseudoaneurysm, laceration, arteriovenous fistula, embolism, arterial occlusion and hematoma formation, occur in about 3 %t of patients undergoing PTCA. The use of large arterial sheaths, the concomitant use of anticoagulant or thrombolytic therapy, an advanced age, and the presence of peripheral vascular disease increase the risk of these vascular complications. Other less common acute complications of PTCA are similar to those of diagnostic coronary angiography, including arterial or ventricular arrhythmias, conduction abnormalities, coronary arterial embolization, cardiac tamponade, allergic reactions to contrast material or one of the medications given during PTCA, vasovagal episodes, and cerebrovascular em-

bolization resulting in aneurologic deficit [25, 26].

### Long Term- Complications

In patients who have undergone successful PTCA, the chief limitation on long term, event- free survival is recurrence of the stenosis, or restenosis. Although improved medical therapy and technical advances over the past decade have reduced the incidence of abrupt closure, the incidence of restenosis has not changed. Several definitions of restenosis have been suggested, but it is most commonly defined as more than 50 % narrowing of the diameter lumen at the site previously successful PTCA. Restenosis occurs in about 60% of those whom a chronically occluded artery has been dialated [27, 28, 29, 30] . Restnosis occurs in one to three month's afterPTCA, and in 95 percent of patients, it occurs within six months after the procedure. Restenosis is uncommon less than one month or more than six months after PTCA [31].

### Mechanism of Restenosis

The process of restenosis is initiated by injury of the vessel, with the subsequent release of thrombogenic, vasoactive, and mitogenic factors [32]. Endothelial and deep vessel injury leads platelet aggregation, thrombus formation, inflammation, and activation of macrophages. These events induce production and release of growth factors and cytokines, which in turn may promote their own synthesis and release from target cells. Thus, a perpetuating process is initiated [33], which results the migration of smooth muscle cells from their usual location in the arterial media to the intima, where they change to a synthetic phenotype, produce extracellular matrix, and proliferate, thereby resulting in a stenosis within the vessel lumen. In addition, scar contraction may occur, further reducing the size of the lumen. These processes make up the wound healing response that occurs in all patients undergoing PTCA. In patients with the most pronounced reparative response to the intimal and medial damage induced by balloon inflation, luminal encroachment is particularly marked, and such patients are said to have restenosis. Contraction of the dialated and stretched medial and adventitial layers- so called elastic recoil- may contribute to restenosis, but such contraction is usually apperent within hours to days after PTCA [34].

### Risk Factors for Restenosis

Numerous clinical, anatomical, and procedural variables have been associated with an increased incidence of restenosis after successful PTCA. The common clinical variables are male sex [35], cigarette smoking [36], diabetes mellitus [37], hypertension [38], hypercholestromia [39], renal disease, vasospatic angina and unstable angina [40, 41]. Among the clinical variables, diabetes mellitus and unstable angina are reported most frequently. The anatomical variables such as; proximal stenosis, saphenous- vein graft involvement of the left anterior descending artery, chronicaly occluded artery, stenosis more 5 to 10 mm in length, severe pre PTCA stenosis [42,43,44], and procedural variable such as residual stenosis, small residual lumen, and used undersized balloon [45] may increase incidences of restenosis after PTCA.

### Consequences of Restenosis

Most patients with restenosis after successful PTCA have recurrent angina, but some of those with angiographic evidence of restenosis

are asymptomatic. Since such patients have a good prognosis, a second angioplasty should be reserved for those recurrent symptoms [46]. Myocardial infarction is rarely the initial manifestation of restenosis. The severity of narrowing at the site of restenosis is usually similar to that PTCA. However, when PTCA is performed in a minimally narrowed coronary artery, the restenosis may be more severe than initial stenosis.

### Management of Restenosis

Restenosis is often treated successfully with a second PTCA. The second procedure is more likely to be successful than the first and less likely to be associated with an acute complication [47]. Probably a second procedure is performed only in patients in whom first was successful, and because the restenosis consist primarily of fibro proliferative tissue rather than atherosclerotic plaque. Numerous pharmacological approaches and devices have been evaluated in an attempt to prevent restenosis. Fish oil, and trapidil (growth inhibitor) have shown promise in some trials [48, 49]. Some trials that failed to demonstrate the benefit of a particular pharmacologic agent may have too few patients and uses adequate doses. The incidence of restenosis after elective placement of an intracoronary stent appears to be low [50, 51], but the result of randomized trials comparing the use of a stent with PTCA alone are not yet available.

### Future Directions

Since the introduction of PTCA is more than 15 years ago, there have been notable advances in technique and equipment. Many stenosis; regardless of their location, severity, or morphologic characteristics, can now be dilated successfully. The development of devices should make it possible to expand the use of PTCA even further. Moreover, new antiplatelet and antithrombin agents may reduce the incidence of acute thrombotic complications. Restenosis remains challenge unfortunately; there has been little progress in reducing its incidence. The physiology of restenosis is multifactorial and poorly understood. In all probability, therapeutic approach that several pharmacologic and procedural innovations will be required to decrease the incidence of restenosis.

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