IMPACT OF SUCCESSFUL PERCUTANEOUS CORONARY INTERVENTION TO PATIENTS WITH CHRONIC TOTAL OCCLUSION ON IN-HOSPITAL MORTALITY AND MORBIDITY

Thesis

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تأثير التدخل العلاجي التاجي عبر الجلد الناجح على مرضى القفل الكلي المزمن أثناء تواجدهم بالمستشفى على الوفيات و المرض

رسالة

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INTRODUCTION

Remarkable progress in percutaneous management of coronary artery disease has been achieved over the last 3 decades. The scaffolding properties of coronary stents have resulted in percutaneous coronary intervention (PCI) becoming a predictable procedure, with reduced rates of acute closure and late restenosis compared with balloon angioplasty alone.[1,2] More recently, the site-specific delivery of antiproliferative agents from drug-eluting stents has been demonstrated to markedly attenuate vascular responses leading to neointimal hyperplasia, further reducing the occurrence of clinical and angiographic restenosis to <10% in most patients.[3,4] PCI in patients with acute coronary syndromes and acute myocardial infarction (AMI) has also been proven to save lives, reduce rates of myocardial infarction (MI), and enhance quality of life compared with alternative treatment modalities.[5-7]

Any definition of coronary CTO must consider the degree of lumen narrowing, antegrade blood flow grade, and the degree of occlusion. CTOs are characterized by significant atherosclerotic vessel narrowing with lumen compromise that results in either complete interruption of antegrade blood flow as assessed by coronary angiography (Thrombolysis in MI [TIMI] grade 0 flow), also known as true total occlusion, or with minimal

contrast peneteration through the lesion without distal opacification (TIMI grade 1 flow), frequently referred to as functional total occlusion. In the absence of serial angiograms, the duration of coronary occlusion is difficult to specify with certainty and instead must be estimated from available clinical information related to the timing of the onset that caused the occlusion, eg, acute MI or sudden change in angina pattern with ECG changes consistent with the location of occlusion. However, many patients the age of CTO cannot be determined with confidence. Furthermore, the temporal criterion used to define a CTO has varied widely in prior reports, typically angina from > 2 weeks[8,9] to > 3 months[10], which in part explains interstudy differences in lesion characteristics and procedural data. In general, a total occlusion of duration > 3 months must be considered chronic.

Chronic total occlusions (CTO) are found in approximately one-third of patients with significant coronary disease who undergo angiography. Despite the introduction of novel technologies, newer guidewires, and a tremendous advancement of technical skills, percutaneous coronary intervention (PCI) for CTO remains a challenge. Even with stenting, there remains a significant rate of both restenosis (32% to 55%) and reocclusion (8% to 12%).[11-15]

AIM OF THE WORK

In a retrospective study design we sought to explore the impacts of successful percutaneous coronary intervention to patients with chronic total occlusion on in-hospital mortality and morbidity.

Chapter 1

INTRODUCTION TO CHRONIC TOTAL OCCLUSION

Definition of chronic total occlusion:

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reports, typically angina from > 2 weeks[8,9] to > 3 months[10], which in part explains interstudy differences in lesion characteristics and procedural data. In general, a total occlusion of duration > 3 months must be considered chronic.

Histology and pathophysiology of chronic total occlusion:

Histology:

A chronic total occlusion has several anatomic components[16,17]. An atherosclerotic plaque is invariably present as a major or minor part of the luminal obstruction. Thrombus is the complementary element. There may be a single clot of uniform structure and age associated with fibrointimal proliferation. The latter situation signifies the occurrence of prior thrombi from previous plaque fissures that might or might not have been totally occlusive. In cases in which they had been totally occlusive, these fissures were partially recanalized before subsequently re-occluding (Fig. 1).

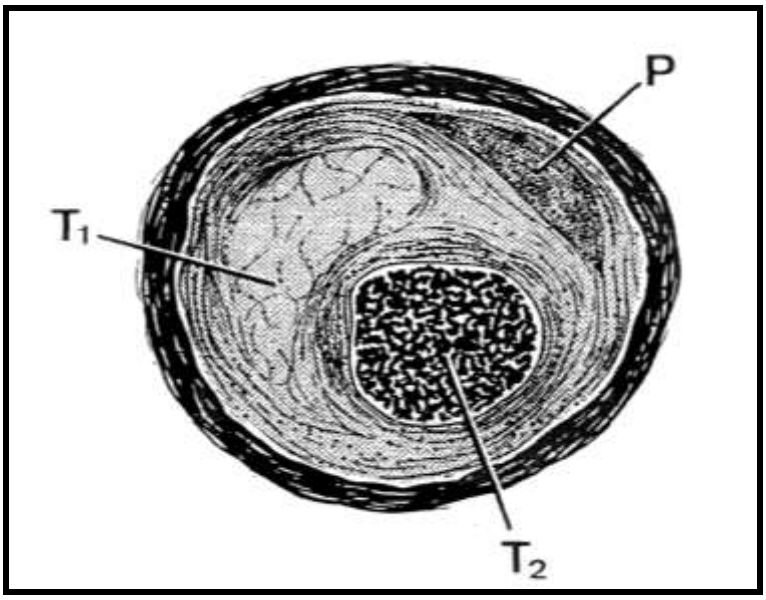

Figure (1): Schematic diagram of a cross section of a totally occluded coronary artery segment. There are thrombotic foci (T_1 and T_2) of different ages, indicating a first plaque (P) fissure with an organized and heavily fibrosed thrombus (T_1) and a more recent one causing complete occlusion (T_2). The extent of fibrosis of the most recent thrombus is the decisive factor in determining the chance of successful balloon recanalization.

The most recent thrombus is assumed to obstruct the last lumen that had been patent up to the final complete occlusion of the particular coronary segment. The recanalization equipment should be passed through this thrombus. The texture of the thrombus is crucial for success or failure of coronary angioplasty. The older and the more fibrosed a clot is, the smaller chance to cross it safely[18].

Spontaneous recanalization of a totally occluded segment may occur by lysis of a clot, development of several new channels through the thrombus (intra-

arterial arteries), dilatation of the vasa vasorum, or a combination of these mechanisms. Angiographically, such a recanalization can be readily distinguished from a total occlusion by the presence of antegrade flow, which may coexist with retrograde filling of the distal part of the vessel (demonstrable by angiography in case of ipsilateral collaterals). However, it cannot be discerned which of the aforementioned mechanisms is active for antegrade flow, and the situation is difficult to differentiate from a subtotal occlusion. Tackling a subtotal occlusion that had never been completely occluded before and that shows no collateralization creates the risk of an acute infarction resulting from abrupt vessel closure; tackling a recanalized segment does not. Conversely, it is usually easy to pass a subtotal occlusion with a coronary guidewire, but it may be tedious or impossible even with sophisticated equipment to pass a recanalized segment because the recanalization may consist of several tortuous micochannels in densely fibrosed tissue or be simulated by copious vasa vasorum[18].

Pathophsiology:

Collaterals and Preservation of Myocardial Function:

Well-developed collaterals at the time of the acute occlusion of the coronary artery avoid cell death of the subtended myocardium. Poor collaterals may still limit necrosis to the least perfused layers, usually the subendocardium. The performance of collaterals correlates well with duration of occlusion and initial lesion severity. In other words, collaterals are quite common in patients with long-standing coronary artery disease and subtotal occlusion of the vessel in question but are rare in young patients with mild coronary artery disease suffering an acute thrombotic coronary occlusion from a ruptured plaque rather than a significant stenosis[18]. There is a greater propensity for spontaneous recanalization in the young patient with an occlusion based on an insignificantstenosis. They often present with a recanalized vessel but completely lost myocardial function. The typical chronic occlusion to be tackled is therefore one in an elderly patient with established coronary artery disease, complex anatomy in and around the occlusion, and a fairly well-preserved distal myocardium[18].