



Dear Colleagues & members of the ICTS

At last and after hard efforts we arranged to edit this issue of the Bulletin of the ICTS, hoping it will satisfy your needs.

I would like to urge our colleagues to participate actively through their articles, case reports, and personal experiences.

We are planning to have ICTS international congress by the end of this years, it called for all to send this articles and opinions so as to have successful cardiology congress.

Faisal Haba

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What is The Best Approach to Diagnose Renal Artery Stenosis ?

by: Qasim Mudelal

Intervensionist / Iraqi Center for Heart Diseases

Renal artery stenosis (RAS): narrowing of one of the renal arteries or their branches, is most commonly caused by atherosclerosis. lesss frequently, is caused by fibromuscular dysplasia, and is rarelt has other causes.

Diffrences between atherosclerotic renal artery stenosis and fibromuscular dysplasia.

Variables	Atherosclerosis	Fibromuscular
age at presentation	> 50	< 50
sex	either usually	female
lesion location	ostial, proximal	middle or distal
blood pressure response to revascularization	unclear	Normotension in most patients

The classic clinical clues that suggest the diagnosis of renal artery stenosis include:

1. The onset of stage 2 hypertension (BP> 160/100 mmHg) before 30 or after 50 years of age or in the absence of a family history of hypertension
2. Hypertension associated with renal insufficiency(especially if renal function worsen after the administration of any agent that blocks the renin angiotensin aldosterone system).

3. Hypertension with repeated hospital admissions for heart failure.
4. Drug resistant hypertension (BP above the goal despite treatment with at least three drugs of different classes at optimal doses including diuretics.

Once renal artery stenosis is suspected, confirmation of the diagnosis is typically made by means of imaging, since biochemical tests such as the measurement of plasma renin concentrations lack specificity.

Duplex ultrasonography: is an excellent tool because it is non invasive and has no apparent side effects. Doppler measurement of the renal artery velocity provides a functional assessment of the severity of the stenosis i.e. higher velocity correlates with a greater pressure differential a cross the stenosis. However, Duplex imaging is limited by abdominal obesity or bowel gas, is technically demanding, and is not available at all centres.

MRI and computed tomographic angiography (CTA): with the use of high resolution multislice detector devices. These techniques can provide elegant image of the renal arteries and the abdominal aorta and can show images in multiple planes to enhance clarity. However, equipment, technique, and reconstruction of the images may affect image quality, as can patient related factors, including the presence of calcium, the presence of stents, and the ability to hold one's breath during imaging. In patient with chronic kidney diseases, the use of MRA and CTA is limited by toxicity of the contrast medium: nephrogenic systemic fibrosis is associated with gadolinium and nephropathy is associated with

iodinated contrast dye.

Angiography: High quality digital subtraction angiography with or without selective renal angiography may be performed with the use of small diameter catheters and minimal amounts of contrast material in order to reduce the risk of vascular complications and contrast nephropathy.

Although the degree of atherosclerosis of the aorta, the size of the kidney, the extent of post stenotic dilatation and the rapidity of the appearance of the wash out of contrast material is useful in confirming or ruling out the diagnosis of RAS, no tests or findings conclusively establish the functional significance of the lesion or predict the response to revascularization.

Physiological measures: such as nuclear scintigraphy, renin sampling from the renal veins, determination of pressure gradient across stenoses, and ultrasonographic measurements. These may be useful in selected situations to determine whether a kidney supplied by an occluded renal artery is viable and is likely to be contributing to hypertension or whether stenosis within a renal artery is affecting intrarenal pressures.

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Brittle Asthma

Mohammad Y. Abdulrazaq CABM , FIBMS (Respiratory)
Pulmonologist, Specialized Center for Respiratory and Chest Disease, Directorate of Public Health.

Bronchial asthma is an inflammatory disease of the airways with a very wide clinical spectrum. With the advent of daily peak expiratory flow (PEF) monitoring several clinical phenotypes of asthma were described, based on the patterns of PEF variability. Asthmatics were labeled as '**morning dippers**' if they had an early morning fall in PEF, '**double dippers**' if the PEF dipped twice in a day or '**chronic persistent**' if PEF showed low values with less variability'.

In 1977, Turner-Warwick used the term '**brittle asthma**' for the first time to describe asthmatics, with chaotic PEF. This type of asthma was difficult to control and could lead to death in a sudden attack. Two types of brittle asthma have been described, type I and II.

Definitions

Type I brittle asthma: characterized by more than 40% diurnal variation in PEF for more than 50% of time maintained over a period of at least 150 days despite maximal medical treatment including inhaled gluco-corticosteroids of at least 1500 ug of beclomethasone .

Type II brittle asthma: characterized by sudden acute attacks occurring in less than three hours without an obvious trigger on a background of apparent normal airway function.

Diagnosis: Careful exclusion of all the factors that may be responsible for poorly controlled asthma, with PEF monitor-

ing.

Treatment of type I: Allergen avoidance, Immune-modulators (cyclosporin and methotrexate), Long term subcutaneous B2 agonists, that is delivered by a syringe pump in doses usually of 3-12 mg/day, and Long acting inhaled B2 agonists.

Treatment of type II: The mainstay of treatment is immediate administration of adrenaline. Adrenaline may have theoretical advantages over selective β_2 agonists, because of its action as an α -adrenoceptor against reducing airway oedema .Preloaded syringes (Epi-Pen; Ana Pen) may be useful as an emergency treatment.

Summary: There is now good evidence that brittle asthma should be regarded as a separate clinical phenotype of asthma at the severe end of the spectrum.

Two types of brittle asthma can be identified. Type I is characterized by wide swings in peak expiratory flow and type II by very sudden attacks out of the blue.

Type I brittle asthma is more common in females several factors including allergen sensitization and psychosocial factors may be important. Peak expiratory flow monitoring is essential for recognizing these patients. Treatment of type I brittle asthma is difficult and needs to be holistic. Continuous subcutaneous infusion of terbutaline (or salbutamol) and dietary exclusion of relevant foods to which the patient may be allergic may be helpful in selected patients.

Type II brittle asthma is less difficult to manage and includes the use of subcutaneous adrenaline .

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Glycemic Control in Patient with Acute Coronary Syndrome

Abbas Ali Mansour (MD, FRCP Edin).

Assistant Professor of Medicine, Department of Medicine/ Basrah College of Medicine

Abnormal glucose metabolism, including prediabetes and type 2 diabetes, was more common than normoglycemia in three prospective studies on patients with acute coronary syndrome (ACS) -(Table-1).1-3

The relationship between admission hyperglycemia and mortality: Studies have documented the association between higher admission glucose levels and increased risk of mortality and complications in patients with ACS regardless the diabetes state. 4

Hyperglycemia definition: No uniform definition of hyperglycemia in the setting of ACS exists. However it appears reasonable to consider random glucose levels > 140 mg/dL (8.0 mmol/L) as the definition of hyperglycemia in the ACS setting.4

Is there evidence that glucose lowering during ACS hospitalisation impacts patient outcomes? Prior studies have definitively demonstrated the prognostic association between elevated glucose and adverse patient outcomes in ACS, but these findings cannot answer the critical question: is hyperglycaemia a direct mediator of increased mortality and complications, or is it just a marker of greater disease severity and co-morbidity burden. Most of studies on reduction of hyperglycemia after ACS showed only survival benefit of glucose reduction if the patient was non-diabetics than those with established diabetes mellitus.⁴ There are few epidemiological data regarding the impact of in-hospital insulin therapy on outcomes in hyperglycemic ACS patients. However, whether insulin therapy is associated with any clinical benefit in ACS above and beyond its associated glucose-lowering effect remains a subject of debate. No study was able to determine whether patients who received insulin actually or oral antihyperglycemia had better outcome during hospitalisation.

Patterns of hyperglycemia management in hospitalized ACS: Current consensus recommended that treatment with insulin should be considered in ACS patients with severe hyperglycaemia (>180 mg/dL, 9.9 mmol/L), regardless the diabetes state. Unfortunately insulin underused in ACS unless the patients are diabetic before.

Glucose-insulin-potassium (GIK) therapy in ACS: Trials of fixed-dose GIK therapy in ACS have largely shown no clinical benefit, and the focus has shifted to conducting trials in which insulin is dosed to achieve and maintain glucose levels in the near-normal range.

Conclusion: Hyperglycemia regardless the diabetes state was seen in two third of patients admitted with ACS. Maintaining near normal glycemia is a target, but the way to achieve that still not resolved.

Table (1): Abnormal glucose metabolism in patients with cardiovascular disease as reported in three prospective studies.

	Type 2 diabetes %	Prediabetes %	Normoglycemia %
*GAMI	31	35	34
**EHS	18	37	45
***CHS	27	37	36

*GAMI= Glucose tolerance in patients with Acute Myocardial Infarction study.

**EHS= Euro Heart Survey

***CHS= China Heart Survey.

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Acute Myocardial Infarction in Young Patients

Rafid Fayadh Al-Aqeedi MD, EAPCI, FESC

Department of Cardiology and Cardiovascular Surgery, Hamad Medical Corporation, Doha, Qatar

Acute myocardial infarction (AMI) in young patients of less than 40 years has been estimated in 2-10% of all AMI^(1,2). Young patients had different characteristics from that of older ages. Furthermore, their presentation is frequently silent and more vulnerable to misdiagnosis since they do not frequently have the traditional coronary risk factors⁽³⁾.

Young patients with AMI usually have multiple risk factors for coronary artery disease (CAD). In some studies more than 90 % had one or more traditional risk factors for atherosclerosis Al-Koubaisy et al⁽⁴⁾. Risk factor analysis in young AMI patients has recognized smoking as stronger factor for the occurrence of AMI in men and women under the age of 40. Smoking affects all phases of atherosclerosis since it potentiates thrombosis, causes endothelial dysfunction, induces coronary vasoconstriction even in patients with normal coronary vessels and has pro-inflammatory effects. Hypercholesterolemia is common in young patients with CAD, but its high prevalence is similar to that in older patients. However, when compared to older patients, young patients have lower mean serum high density lipoprotein concentrations and higher serum triglycerides⁽⁵⁾. Coronary artery disease has both genetic and environmental determinants on cardiovascular risk. A higher incidence of a positive family history in young patients (64 %) was reported by Cole et al⁽⁶⁾. It has been suggested that genetic factors are more likely to affect the young rather than older individuals and they may contribute to the different mechanisms leading to atherosclerotic lesions. Since the discovery of the first genetic variant, 9p21 in 2007, genomic studies gained attraction in predisposition to CAD. The 9p21 variant risk for CAD and its independence of conventional risk factors have been confirmed worldwide by many investigators. Two other important coronary risk factors, diabetes mellitus and hypertension, appear to be less common in young patients. Increasing body mass index was associated with both fatty streaks and raised atherosclerotic lesions in the coronary arteries of young men, but not young women.

Patient with AMI may present either with or without normal coronary angiography. The prevalence of normal coronary arteries in AMI is higher in younger individuals than their older counterparts and it has been reported in 16-35% of patients below 30 years of age^(7,8). Their coronary angiograms characteristically showed a less extensive disease, mainly as no-vessel or one-vessel form, as compared to older adults. The pathophysiology of AMI in the presence of “normal” coronary arteries remains unclear but can be explained on the basis of coronary artery thrombosis, embolisation, spasm, or a combination of these processes. Myocardial infarctions caused by coagulation disorders constitute 5% of all cases. Coronary artery spasm causing myocardial infarction is reported as the likely mechanism with use of drug or substance like cocaine⁽¹⁰⁾, marijuana, alcohol binges, butane or amphetamine. A potential diagnostic problem that

is most common in younger subjects is that myocarditis can mimic an AMI. This disorder should be particularly considered in young patients with a clinical presentation of an AMI who have a normal coronary angiogram ⁽¹¹⁾.

Accelerated atherosclerosis in young adults is remaining an important cause that resulted in AMI, attributable to rupture of a vulnerable plaque or erosion of the endothelial layer. Spontaneous coronary artery dissection as a cause of myocardial infarction although is a rare condition, it shows greater prevalence in young women around partum period. Coronary artery aneurysms can be congenital or acquired secondary to Kawasaki's disease in childhood ⁽¹²⁾. They have been linked to AMI in young adults, though the mechanism is not understood. Anomalous origins of either left or right coronary arteries have been associated with AMI and are related to acute angulation and compression of the artery at its origin or along its course.

Appropriate treatment of young patients with AMI was adapted from the routine adult management protocols. Numerous studies have shown significant benefits after smoking cessation, leading to a 36% decrease in mortality, Fournier et al ⁽⁷⁾. The physician should first exclude the existence of atherosclerosis and diagnostic coronary angiography should be performed in all cases to establish the wide range of etiologies of infarction and guide therapy. In those patients with atherosclerosis, early intervention with primary angioplasty has an improved outcome over thrombolysis. There are also reports of successful percutaneous coronary intervention (PCI) with or without stenting in the antiphospholipid syndrome ⁽¹³⁾. Long term anticoagulation is needed following an infarct and the international normalised ratio should be kept above 3. PCI and stenting should be considered in spontaneous coronary artery dissection ⁽¹⁴⁾. Anticoagulation should be considered in the nephrotic syndrome if serum albumin is less than 20 g/l. β -Blockers are best avoided in cocaine or amphetamine induced spasm as there is a potential risk of unopposed α 1-adrenergic action with worsening coronary spasm.

The in-hospital mortality in young patients has ranged from 0-4 %, a value lower than that in older patients ^(15,16), also have a good long-term outcome after AMI ⁽¹⁷⁾. However the long-term mortality in young patients with AMI can be higher in patients with high incidence of cardiovascular risk factors, delayed presentation and those with late initiation of revascularization due to delayed diagnosis.

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Primary Percutaneous Coronary Angiography

Aram Mierza

Sulaimaniyah Heart Center

Primary PCI is defined as percutaneous intervention in the setting of the STEMI without previous or concomitant fibrinolytic treatment. RCTs and meta-analysis comparing primary PCI with in-hospital fibrinolytic therapy in patients within 6-12 hrs after symptom onset treated in high volume, experienced centers have shown more effective restoration of vessel patency, less re-occlusion, improved residual LV function and better clinical outcome with primary PCI. Cities and countries switching from fibrinolysis to primary PCI have observed a sharp decrease in mortality after STEMI.

American College of Cardiology/ American Heart Association (ACC/AHA) guidelines specify that primary PCI should be performed by operators who perform 75 elective procedures per year & at least 11 procedures for STEMI in institutions with an annual volume of 400 elective and 36 primary PCI procedures, such a policy decision is justified by the strong inverse volume – outcome relationship observed in high risk and emergency PCI.

According to the ESC guidelines, primary PCI is therefore, generally the preferred reperfusion strategy if performed by experienced team.

Since November 2009 till November 2010, 93 cases of primary PCI is done in Sulaimany center for heart disease, all of them done within window period apart from one case which done after 24 hrs from the onset of chest pain due to severe ongoing chest pain & cardiogenic shock in a patient with inferior STEMI at 11:30 PM after inserting temporary pacemaker & intra aortic balloon pump with good final results.

The youngest patient who underwent primary PCI was 16 years old girl (case report) who presents with extensive anterior STEMI with recurrent attacks of VFs who resuscitated by DC shock (> 12 times) & then transferred to the cath lab where Primary PCI done for total occlusion of ostial LAD cut which was full of clot.

Success rate was 98.5% (only one case died 6 hrs after angioplasty possibly due to stent thrombosis). Follow up done till now for 87 patients (93.5%) all of them are asymptomatic with neither chest pain nor evidence of heart failure.

The key success points for Primary angioplasty are the presence of experienced team, "as soon as possible", optimized antiplatelets (loading dose of Clopidogrel & GP IIb/IIIa inhibitors) and anticoagulation, etc. Previously the patients were receiving Tirofiban as loading & maintenance dose in the cathlab but fortunately Abciximab is available now which we give for all patient undergoing primary PCI as loading & then maintenance dose.

Unfortunately, for the time being, in our center, only small proportion of patient with STEMI will undergo Primary PCI for many reasons, the most critical one is lack of transfer system & neglectance of stakeholders!

Finally, I hope that we can provide this service 24 hourly seven days per a week in early future in our centers & majority of other cardiac centers in Iraq.