Ahmadi Hospital Medical Group Journal

AMJ

Ahmadi Hospital

Volume II - 2020

Honorary President: Dr. Emad Al Awad

(Manager Medical Group)

Editorial Board :-

Dr. Hussain Younis Haji

Dr. Majdi Amasheh

Dr. Shaymaa Mohammad Abdulsamad Ahmad

Dr. Ahmed Abdul Malek

Mrs. Abeer Mohammed Al-Kandery

Mr. Mohammad Al-Qenaei

Contents:

Chief Executive Officer's

Health, Safety, Security & Environment Award 2019

Ahmadi Hospital Transition Plan/Execution from the Existing to the Newly Built Facility

The Health Information Management System (HIMS) Team has won Health, Safety, Security & Environment Award 2019 for Ahmadi Hospital Transition Plan/Execution from the Existing to the Newly Built Facility project.

Moreover, Ahmadi Hospital is considered as a main pillar and pioneer in advancing the HSSE for the KOC Community, as well in empowering KOC employees' to take control over their health, and advancing in the adoption of the innovative medical and technology solutions.

The hospital transition project from the existing facility to the newly built one was an exceptional and outside the scope of normal duties project that was exclusively handled by the hospital teams, especially The Health Information Management System (HIMS) Team which is considered as the technology backbone of the hospital.

The Ahmadi Hospital transition project was a tremendous success. The Health Information Management System (HIMS) Team, with the endeavor for operational excellence and patient-focus approach, handled the project very effectively and concisely, and executed it exclusively utilizing the internal resources and expertise.

Tips for planning a Clinical Audit

By :Dr. Mahmood Saeed, (Consultant Paediatrician)

Clinical Audit is a tool, which if applied diligently can result in continual improvement in patient care. It is a process by which Clinicians can compare their performance / management of a condition / situation with recommended and internationally agreed standards. If gaps are found, reasons can be looked at and remedies applied to improve the care before a re-audit. Individuals for the shortcoming are not identifiable because confidentiality is the first requirement, so the sole purpose of the process is improving the consistency and quality of care of the patients. It is mandatory in most western hospitals.

1. What to Audit? Almost any aspect of care of any condition (Medical / Nursing / Support services) can be audited. However, it is better to audit a common condition / issue because improvement will benefit more patients. In addition, collecting information and drawing conclusions from the audit will be easier and more reliable. For example, if you look at your management of Acute Asthma or Diabetic Ketoacidosis on the ward, you will need to go back a few months only to collect reasonable data and any improvement in care will benefit many patients. On the other hand, if you want to audit your care of patients with Addison's disease, you may have to go back years for collecting data and any suggested improvement may not be practiced for a while.

2. Retrospective or Prospective? You can do either. There are pros and cons but both are acceptable. Retrospective are more commonly done because you can get the data quickly. The only drawback is that you may not have recorded the parameters you wish to analyze or you may not have agreed guidelines at the time. In prospective audits, you can plan it accurately but you run the risk of bias i.e. your practice may not reflect the reality because you are aware that an audit is being conducted.

3. How many patients? Any reasonable number that would reflect the practice. Unlike a clinical trial, there is no minimum number requirement. Obviously, bigger data will be more representative but more difficult to collect and analyze. So, choose a reasonable number depending upon the frequency of the condition. Generally, the number seen in the past 6 – 12 months should be sufficient.

4. The Audit Question / Questions? Think carefully about what you want to audit. Keep it simple. Once you start collecting the data, the temptation is to record everything. Avoid it. Unrelated parameters recording only confuses the issue. Be clear and collect only the data that is relevant to your audit question / questions

Similarly, keep the audit questions simple. If you are auditing a large issue, it is better to break it down in smaller multiple audits. For example, if you want to audit your care of Asthma patients, do not measure your in-patient and outpatient care in a single audit. In fact, even the in-patient care is preferably subdivided in to smaller, simpler audits i.e. emergency care, care on the ward and discharge policy etc. etc. Complex data is difficult to interpret and difficult to focus. It may also confuse the remedial recommendations. So, try not to address too many questions in a single audit.

5. Where to Start? Where to finish?

Step 1 - Set and agree standards.

If you have a departmental guideline on the subject that is your standard, against which you need to measure your performance. It is critical that all the relevant clinicians agree with the guideline (hopefully the agreement was achieved before the guideline was instituted). If no guideline exists then the standards of good quality care can be adapted from national or international standards. Again, it is critical that there is a general agreement with those standards before you start, because it will be a waste of time to show a deviation from a standard which your colleagues do not see as a deviation. For example, it will be impossible to measure a compliance with antibiotic policy if there is no agreement within the group with the policy to begin with. Then set your audit questions against those agreed standards.

Step 2 - Plan / design a data collection sheet / form.

To collect relevant information, you would need a sheet / form. One form to be filed for each patient / episode. While designing this sheet, do not forget that you would need to analyze the data you are collecting. So collect the data in a way that you can interpret it in a meaningful way at the end. Avoid free hand comments, as these are almost impossible to analyze statistically. Breakdown / arrange the information into small tick boxes of relevance. For example if you were auditing the timing of first dose of Prednisolone in emergency management of Acute Asthma, it will be inappropriate to collect this data in free hand because you will end up 50 different times (if you were auditing 50 patients) which you will not be able to interpret meaningfully. Break it into clinically significant groups i.e. less than 60 minutes, 60 – 90 mins, 90 – 120 mins and >120 mins. You can then easily calculate patients in each group, each of which describes the level of quality of care. This is just an example; you can set the groups according to the clinical relevance.

Step 3 – Analyze the data. Sum up the conclusions.

Be honest. It is confidential. It is for information of the group. It is to improve patient care. You cannot address a problem if you refuse to identify it and accept it. The fact that you are prepared to look at yourselves critically implies good practice and is much better than those

who assume that they are giving high quality care without wanting to measure it. You do not need to share the information with anyone outside the group if you prefer not to. You will often be surprised to find out what actually happens compared to what you think is happening but then that is the whole purpose.

Step 4 – Identify deficiencies, its reasons and formulate remedies.

Only the group can decide as to what are" acceptable "results but if they are not which will often be the case, then you need to recommend the steps / interventions to improve after analyzing the reasons / causes for unsatisfactory results. Be practical and realistic, remembering the SMART goals formula. Small, sustained improvements are much better than transient large improvements.

Step 5 – Apply Changes / recommendations.

Make sure you get agreement of all involved. You are mostly dealing with highly skilled professionals and they are not going to alter their practice unless you make them see the deficiencies caused by the current practice and do not convince them that "change" is needed. All involved need to have a sense of ownership of the proposed "changes" and be convinced of its benefits.

Step 6 – Re-Audit in 6 months.

Carry out the same clinical audit again to see if you have achieved an improvement, if so document it, share it, celebrate it.

Step 7 – Keep Going

If you have not achieved the desired results, do not be disappointed. You will not be the first or last to face this. Keep going with the same audit repeatedly until you achieve the desired standards. Even when you do, do not forget to check in a year or two to make sure you are maintaining the standards.

Happy Auditing.

P.S I will be more than happy to assist / help in any way (if I can) to organize a clinical audit project, if anyone wishes me to do so. You can contact me by email msaeed@kockw.com or via my mobile 99993752.

Endothelin A receptor antagonist reduces renal events in selected high-risk T2DM patients

Atrasentan and renal events in patients with type 2 diabetes and chronic kidney disease (SONAR): a double-blind, randomized, placebo-controlled trial

By: Dr. Mohamed Foda

Introduction and methods : People with type 2 diabetes (T2DM) are at high risk of developing end-stage kidney disease (ESKD) and CV complications, despite treatment, especially when albuminuria is high .Endothelin receptor antagonists reduce albuminuria and blood pressure, but they can cause sodium retention. Avosentan is a non-selective endothelin receptor antagonist, and was studied at high dose in a trial that was stopped prematurely, because of an increased incidence of heart failure. Atrasentan is a more selective endothelin A receptor antagonist that was found to reduce albuminuria at low doses during short-term treatment, without causing significant fluid retention .

The Study of Diabetic Nephropathy with Atrasentan (SONAR) aimed to evaluate the efficacy and safety of atrasentan in patients with T2DM and chronic kidney disease (CKD). SONAR was a double-blind, randomized, placebo-controlled event-driven trial in adults with T2DM and eGFR 25-75 mL/min/1.73m², a urine albumin-to-creatinine ratio (UACR) of 300-5000 mg/g, serum albumin ≥25 g/L, brain natriuretic peptide (BNP) concentration ≤200 pg/mL, serum potassium ≥3.5 mmol/L and systolic BP 110-180 mmHg. Those who showed evidence of fluid retention were excluded from SONAR, to minimize the risk of heart failure. Responders to treatment were selected, based on the extent of reduction in albuminuria during an 6-week open-label period of treatment with atrasentan (0.75 mg orally once daily). After this enrichment period, responders to atrasentan were randomized to atrasentan or placebo. A subset of non-responders was also randomized to atrasentan or placebo, to evaluate whether the drug yielded renal benefit in this population.

The primary outcome was the efficacy to delay progression of CKD, defined as time from randomization to the first occurrence of any of the following components of a composite endpoint: doubling of serum creatinine, onset of ESKD (chronic dialysis for >90 days, kidney transplantation, eGFR <15 mL/min/1.73m², or death from kidney failure). The trial was stopped prematurely after it had become apparent that the rate of the primary outcome was much lower than anticipated. At completion of the trial, 184 primary renal events had occurred (as opposed to the 425 aimed for). 2648 Patients out of 4711 who completed the enrichment period, were responders, and subsequently randomized to atrasentan (n=1325) or placebo (n=1323). 1020 Non-responders were randomized (n=509 to atrasentan, n=511 to placebo). Responders were followed for a median of 2.2 years (IQR: 1.4-2.9).

Main results

- *UACR increased more during follow-up in the placebo group than the atrasentan group (difference: 33.6%, 95%CI: 29.1-38.2, P<0.0001). Increase in BNP was higher with atrasentan than with placebo (difference: 10.5%, 95%CI: 5.1-15.4, P<0.0001).*
- *The composite primary outcome occurred significantly less often in the atrasentan group (6.05% vs. 7.95%, HR: 0.65, 95%CI: 0.49-0.88, P=0.0047).*
- *The risk of doubling of serum creatinine was lower with atrasentan (HR: 0.61, 95%CI: 0.43-0.87, P=0.0055).HR for ESKD was 0.73 (95%CI: 0.53-1.01, P=0.06).*

- *The first secondary endpoint of ≥50% eGFR decline showed a risk reduction with atrasentan (HR: 0.73, 95%CI: 0.55-0.98, P=0.038).*

- *HR for the cardiorenal composite outcome was 0.80 (95%CI: 0.64-0.999, P=0.049), and no effect of atrasentan on the cardiovascular composite outcome was seen (HR: 0.88, 95%CI: 0.64-1.22, P=0.448).*

- *In the atrasentan group, the mean rate of change in eGFR during the trial was -2.4 mL/min/1.73m² per year, as compared with -3.1 mL/min/1.73m² per year (P=0.00049).*

- *Among non-responders, no significant effect of atrasentan on the primary renal outcome was seen (14.3% vs. 17.0%, HR: 0.75, 95%CI: 0.55-1.03, P=0.079).*

- *No interaction of being a responder or non-responder on the effect of atrasentan on the primary renal outcome was seen (P-interaction: 0.41). When the two groups were combined, the primary renal outcome was seen in 8.3% of patients treated with atrasentan and in 10.5% of the placebo group (HR: 0.72, 95%CI: 0.58-0.89, P=0.0023).*

- *Fluid retention and anemia were the most frequently reported adverse events, and more often in the atrasentan group. Serious adverse events occurred more frequently in the atrasentan group (36.3% vs. 32.6%). Admission for HF occurred in 3.5% of patients on atrasentan and in 2.6% of those on placebo (HR: 1.33, 95%CI: 0.85-2.07, P=0.208). 4.5% And 3.9% patients died, respectively (HR: 1.09, 95%CI: 0.75-1.59, P=0.65).*

Conclusion

In a selected population of patients with T2DM and CKD, long-term low-dose treatment with atrasentan significantly reduced the risk of the primary composite renal outcome of doubling of serum creatinine or end-stage kidney disease, as compared with placebo. These findings were obtained after these patients had demonstrated a substantial UACR reduction and minimal clinical signs of sodium retention during short-term treatment with atrasentan. Thus, in clinical setting, strict interpretation of these results would imply that monitoring of UACR response after atrasentan initiation is required. This study was only adequately powered to assess the effect of atrasentan in responders, but the consistent effect in the combined analysis of responders and non-responders may suggest a broader indication. These findings inform the position of endothelin receptor antagonists as a future treatment option in diabetes patients at high renal and CV risk.

References

- *LAMBERS HEERSPINK HJ, PARVING H-H, ANDRESS DL, ET AL., - THE LANCET 2019. APRIL 14. HTTPS://DOI.ORG/10.1016/S0140-6736(19)30772-X,*

- *Presented during ISN-WCN 2019 in Melbourne, Australia on April 15, 2019.*

Nomenclature of Monoclonal Antibodies

By : Dr. Rami Alhanbali , MD Ophthalmology Specialist

Emil Von Behring and Kitasato Shibasaburo discovered in 1890 that diphtheria and tetanus toxins were neutralized in the bloodstream of animals by substances they called antitoxins, which were specific for the respective toxin. Behring received the first Nobel Prize in medicine for their finding in 1901. A year after the discovery, Paul Ehlrich used the term antibodies for these antitoxins.

Monoclonal antibodies, by definition, are antibodies produced by a single clone of lymphocytes and are directed to a specific single epitope of the antigen (this could be a receptor, a protein or another antibody).They differ from polyclonal antibodies normally present in serum to combat several antigens or several epitopes in the same antigen.

Monoclonal antibodies are rapidly expanding and are becoming the current state of art in medical therapy of many diseases. Because of the wide expansion, an international nomenclature is adopted to facilitate the understanding of the function of the monoclonal antibody. This naming scheme is used for both the World Health Organization's International Nonproprietary Names (INN) and the United States Adopted Names (USAN) for pharmaceuticals.

- *To understand the nomenclature the name should be read from right to left (reverse) direction:*

MAB: Monoclonal Antibody

• *The next word segment from the right is (Xi, zu or mu …) that indicates that the monoclonal antibody is:*

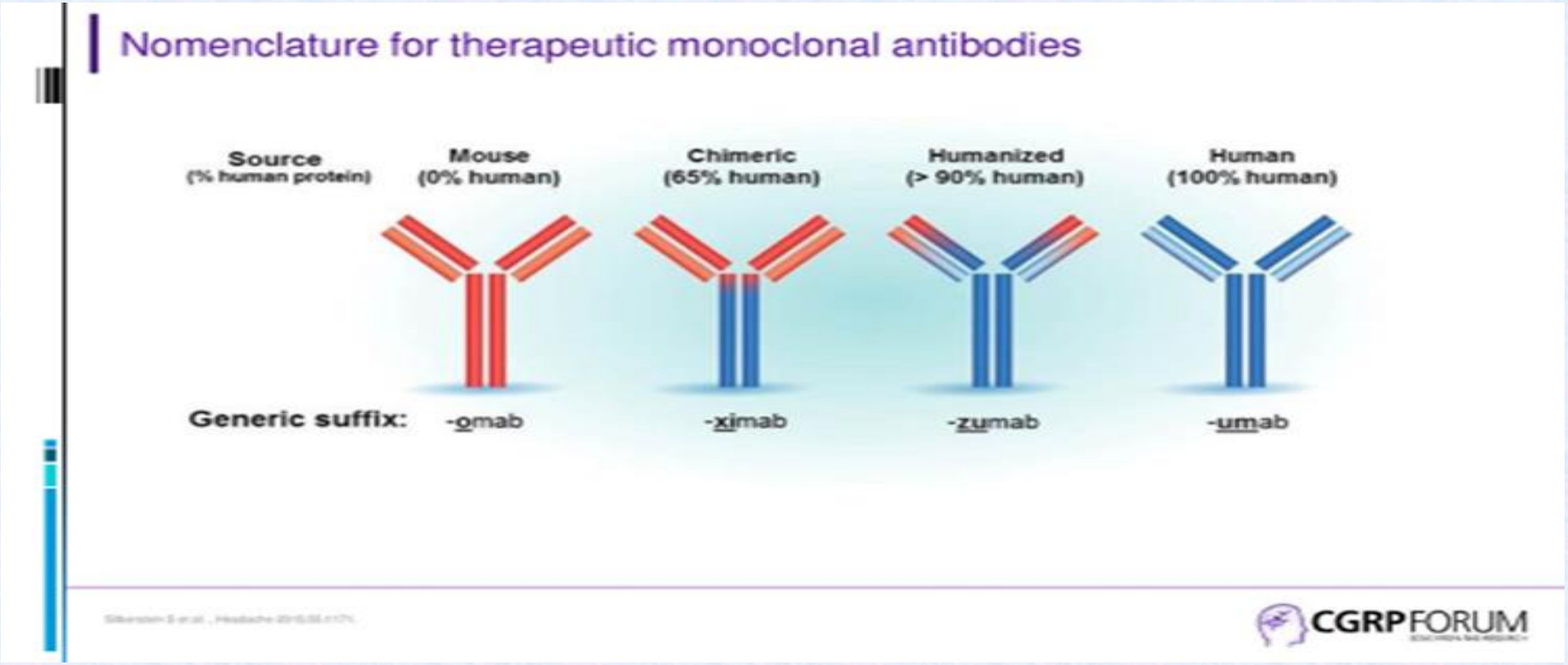
1. Mo mab: mouse in origin Moxetumomab

2. Xi mab: chimeric (65 % human) Infliximab

3. Zu mab: humanized (95 % human) Bevacizumab

4. Xizu mab: mixed chimeric and human

5. Mu mab: human (100 % human) Panitumumab



• *Then comes the segment that tells its primary site of action, common ones are:*

L and Li for lymphocytes and Tu for tumors and Ne for neural and Ci for circulation

Target			
-o(s)	Bone	-me(l)	Melanoma
-vi(r)	Viral	-ma(r)	Mammary tumor
-ba(c)	Bacterial	-go(t)	Testicular tumor
-li(m)	Immune system	-go(v)	Ovarian tumor
-le(s)	Infectious lesions	-pr(o)	Prostate tumor
-ci(r)	Cardiovascular	-tu(m)	Misc. tumor
-mu(l)	Musculoskeletal	-neu(r)	Nervous system
-ki(n)	Interleukin as target	-tox(a)	Toxin as target
-co(l)	Colonic tumor	-fu(ng)	Fungal

- *Then the prefix is company specific and carries no specific meaning. It should be unique for each medicine and contribute to a well –sounding name. This means that antibodies with the same source and target are only distinguished by their prefix . Even antibodies targeting exactly the same structure are differently prefixed such as adalimumab and golimumab,both of which are TNF inhibitors.*

Barone's Guide to Monoclonal Antibody Nomenclature

How to name a monoclonal antibody:

MABs are named by combining a **Prefix** + **Target** + **Source** + **Suffix** (usually "mab")

Prefix +

Antibody Target	
• Bone	-o(s)-
• Cardiovascular	-c(i)-
• Immune-modulating	-l(i)-
• Interleukin	-k(i)-
• Tumor	-t(u)-
• Virus	-v(i)-

+

Antibody Source	
• Human	-u-
• Humanized	-zu-
• Chimeric	-xi-
• Rat	-a-

+ **-mab**

A second word following the name of the antibody indicates that another substance is attached, which is done for several reasons.

I . An antibody can be PEGylated (attached to molecules of polyethylene glycol) to slow down its degradation by enzymes and to decrease its immunogenicity; this is shown by the word pegol as in alacizumab pegol.

ii. A cytotoxic agent can be linked to an anti-tumor antibody for drug targeting purposes. The word vedotin, for example, stands for monomethyl auristatin E which is toxic by itself but predominantly affects cancer cells if used in conjugates like glembatumumab vedotin.

iii. *A chelator for binding a radioisotope can be attached. Pendetide, a derivative of pentetic acid, is used for example in capromab pendetide to chelate indium-111.If the drug contains a radioisotope, the name of the isotope precedes the name of the antibody. Consequently, indium capromab pendetide is the name for the above example including indium*

One of the common examples in Ophthalmology is Ranibizumab (Lucentis):
R: company prefix
Anibi: vascular endothelial growth factor A
Zu: humanized
MAB: monoclonal Antibody

References:

1. Biologicals. 2019 Jul;60:15-23. doi: 10.1016/j.biologicals.2019.05.006. Epub 2019 May 23.
The INN global nomenclature of biological medicines: A continuous challenge.
Robertson JS1, Chui WK2, Genazzani AA3, Malan SF4, López de la Rica Manjavacas A5, Mignot G6, Thorpe R1, Balocco R7, Rizzi M8.

2. Hum Antibodies. 2019;27(1):37-51. doi: 10.3233/HAB-180347.
Nomenclature of humanized mAbs: Early concepts, current challenges and future perspectives.
Mayrhofer P, Kunert R.
3. https://www.wikiwand.com/en/World_Health_Organization

Athlete's foot

By : Dr. Mohamed Jamaledden Mohamed Nasr

It is a common fungal infection that results in flaky skin, cracking and itchiness on the soles of the foot and between the toes.

Most people get it by walking barefoot in a public place like a swimming pool deck or locker room. To reduce the chance of catching athlete's foot, you should do the following precautions.

1. Wear shower shoes, flip flops, or sandals when walking around pools, gyms, shower or locker areas, and hotel rooms . The fungus that causes athlete's foot may be on the floor.

2. Keep your feet dry even if you have not gone barefoot in public areas.

- *Fungus thrives in warm, moist areas such as the one created inside hot, sweaty shoes.*
- *Wearing sandals or flip flops helps when it's hot outside.*

3. Avoid shoes that are made from synthetic materials like plastic and rubber that are more likely to cause sweating.

4. Wash your feet every day with soap and completely dry them after washing.

5. Wear socks made of natural fabrics or fabrics that dry quickly or wick moisture away from the skin.

6. Change your socks every day and more often when your socks get wet.

7. Alternate what shoes you wear each day, if possible, to ensure shoes are dry when they're put on.

8. Avoid sharing towels, linens or shoes with someone who has athlete's foot.

9. Consult dermatologist to start treatment quickly if you think you have athlete's foot.

Reference: "How to Prevent Athlete's Foot." American Academy of Dermatology, www.aad.org/public/diseases/a-z/athletes-foot-prevent.

Management of Chronic rhinosinusitis

(Dupilumab for chronic rhinosinusitis with nasal polyposis (UpToDate November 2019)

By: Dr. Majdi Amasheh

Chronic rhinosinusitis (CRS) is an inflammatory condition of the paranasal sinuses and linings of the nasal passages that lasts 12 weeks or longer. In most cases, the disorder cannot be cured, and the goal of therapy is to reduce symptoms and improve quality of life. Multiple therapies are utilized in the management of CRS, including intranasal saline, intranasal and oral glucocorticoids, antibiotics, and antileukotriene agents. These are combined in various ways to manage specific subtypes of CRS.

Chronic rhinosinusitis with nasal polyposis (CRS with NP) significantly impairs quality of life, and patients often require repeated courses of oral steroids or multiple sinus surgeries over the course of a lifetime. Dupilumab is a monoclonal antibody that inhibits signaling of IL-4 and IL-13, cytokines that promote eosinophilic inflammation. In two placebo-controlled randomized trials including over 700 patients with severe CRS with NP, dupilumab for 24 or 52 weeks improved nasal congestion/obstruction, as well as scores assessing the severity of nasal polyps and the radiographic appearance of rhinosinusitis, and was well tolerated. Longer-term studies comparing dupilumab with surgery are needed. At present, dupilumab is appropriate for patients with CRS with NP with continuing nasal blockage or anosmia despite standard therapy with intranasal and oral glucocorticoids.

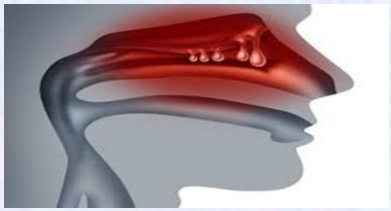
For patients with CRS without nasal polyposis (NP), we suggest initial treatment with one to three months of a combination of intranasal saline (sprays or irrigations) and intranasal glucocorticoids (Grade 2C). For patients who do not achieve adequate relief with intranasal saline and glucocorticoids, we suggest a course of oral glucocorticoids plus oral antibiotics (Grade 2C). A representative regimen for adults is prednisone, 40 mg daily for five days, followed by 20 mg daily for five days plus two to four weeks of an antibiotic. An alternative approach is to initiate treatment with long-term, low-dose macrolide antibiotics.

In patients in whom these medical treatments do not result in sufficient improvement in symptoms, we proceed to endoscopic sinus surgery. Any successful intervention for CRS without NP must be followed by maintenance

therapy, because without ongoing treatment symptoms will eventually return in most patients. For maintenance therapy, we suggest intranasal glucocorticoid nasal sprays and intranasal saline (Grade 2B). For patients with persistent or increasing symptoms despite consistent use of glucocorticoid sprays, we suggest changing to glucocorticoid instillations (Grade 2C).

Patients with underlying allergic rhinitis who have sneezing or nasal pruritus may benefit from additional therapies targeting that condition, including minimally sedating second-generation oral antihistamines, intranasal antihistamine sprays or antileukotriene agents (e.g., montelukast), and/or allergen immunotherapy.

A subset of patients with recurrent or refractory CRS without NP is found to have immunodeficiency, usually antibody defects. These individuals often have a history of pneumonia, recurrent otitis media, or recurrent acute sinus infections.



CRS with nasal polyposis:

- *For patients with CRS with NP, we suggest initial treatment with one to three months of a combination of intranasal saline (sprays or irrigations) and intranasal glucocorticoids (Grade 2C). Patients with severe polyposis may not be able to use intranasal medications because the nasal passages are blocked.*

- *For patients with CRS with NP who are seeking relief of nasal blockage or an impaired sense of smell, we recommend a course of oral glucocorticoids initially to shrink nasal polyps (Grade 1B). A typical adult regimen is prednisone 40 mg for five days, followed by 20 mg daily for five days. Antibiotics are not recommended unless a concomitant infection is suspected. The benefit of oral glucocorticoids is temporary and this intervention must be followed by maintenance therapy.*

- *For patients in whom intranasal and oral glucocorticoids fail to reduce polyp tissue sufficiently and the patient has persistent blockage or anosmia, we suggest either sinus surgery or therapy with a biologic agent (Grade 2B). The choice of approach depends upon availability and patient preference.*

• For patients with mild CRS with NP (i.e., nasal patency and an intact sense of smell) and for maintenance therapy after oral glucocorticoids or sinus surgery, we suggest intranasal glucocorticoids (Grade 2B).

We advise patients to use nasal sprays initially, but if symptoms worsen despite consistent use, we change this to nasal instillations.

• For patients with CRS with NP whose symptoms are not adequately controlled on maintenance treatment with intranasal glucocorticoids (sprays or instillations), nonsedating antihistamines and antileukotriene agents (e.g., montelukast) may provide some added benefit.

• For patients who have failed more traditional therapies, biologics, including dupilumab, mepolizumab, and omalizumab, may offer benefit. Only dupilumab is specifically approved for CRS with NP, although the others are available for the treatment of moderate or severe asthma in patients who have both disorders.

• Some patients with CRS with NP also have asthma and intolerance to aspirin (or other nonsteroidal anti-inflammatory drugs [NSAIDs]), a condition called aspirin-exacerbated respiratory disease (AERD). We suggest aspirin desensitization and daily aspirin therapy for these patients, provided they have no contraindications to aspirin therapy (Grade 2C). This intervention requires access to an allergy specialist with experience in drug desensitizations.

REFERENCES

1. Scadding GK, Durham SR, Mirakian R, et al. BSACI guidelines for the management of rhinosinusitis and nasal polyposis. Clin Exp Allergy 2008; 38:260.

2. Rosenfeld RM, Piccirillo JF, Chandrasekhar SS, et al. Clinical practice guideline (update): adult sinusitis. Otolaryngol Head Neck Surg 2015; 152:S1.

3. Fokkens W, Lund V, Mullol J, European Position Paper on Rhinosinusitis and Nasal Polyps Group. EP3OS 2007: European position paper on rhinosinusitis and nasal polyps 2007. A summary for otorhinolaryngologists. Rhinology 2007; 45:97.

Baseline HbA1c levels associated with CV outcomes in high risk T2DM patients

Impact of Baseline Glycemic Control on Residual Cardiovascular Risk in Patients with Diabetes Mellitus and High-Risk Vascular Disease Treated With Statin Therapy

By: Dr. Mohamed Foda

Introduction and methods

In the UKPDS study, an association was found between HbA1c levels and the risk of macrovascular and microvascular events in patients with T2DM. However, a reduction of HbA1c with glycemic control using predominantly sulfonylurea-based pharmacotherapy showed no association with macrovascular benefits . The number of pharmacotherapeutic options for treatment of T2DM that both improve glycemic control and reduce CV risk have increased in the last years , but it remains unclear whether HbA1c can be used as a marker of CV risk. This post-hoc analysis of the ACCELERATE (Assessment of Clinical Effects of Cholesteryl Ester Transfer Protein Inhibition With Evacetrapib in Patients at a High Risk for Vascular Outcomes) trial evaluated the usefulness of HbA1c as a biomarker of CV risk in optimally treated T2DM patients with high CV risk.

The ACCELERATE trials was a randomized, double-blinded placebo-controlled trial examining the treatment of evacetrapib, a CETP inhibitor, in patients with high CVD risk. Participants were randomized in a 1:1 manner to receive evacetrapib, 130 mg, or placebo. The trial was terminated prematurely after a mean follow-up of 30 months because no overall benefits with evacetrapib were found. This subanalysis investigated the relation between baseline HbA1c (measured at study initiation) and CV events during a follow-up of 30 months in 8145 T2DM patients with a history of coronary artery disease and on optimal medical therapy. Since no effect of evacetrapib was found in the ACCELERATE trial, the entire population with DM was evaluated, regardless of treatment assignment to evacetrapib or placebo.

The primary endpoint was MACE, which included CV death, nonfatal MI, stroke, coronary revascularization, or hospitalization for unstable angina. The secondary endpoint was a composite of CV death, nonfatal MI, and stroke.

Main results

Increasing HbA1c levels at baseline were associated with the occurrence of MACE (Kaplan-Meier (KM) estimates for risk of MACE were 12.6, 14.5, 14.0, 16.1, 16.3, 18.2 for baseline HbA1c levels (%) of <6.0, 6.0-<6.5, 6.5-<7.0, 7.0-<7.5, 7.5-<8.0, ≥8.0, respectively, P <0.001).

- *Increasing HbA1c levels at baseline were also associated with the secondary composite endpoint of CV death, nonfatal MI, and stroke (KM estimate, 7.8-11.3, P=0.003).*
- *Individual endpoints of nonfatal MI (KM estimate 3.1-7.0, P<0.001), hospitalization for unstable angina (KM estimate 1.8-5.0, P=0.003), and need for coronary revascularization (KM estimate 7.3-11.1, P=0.001) were also associated with increasing HbA1c levels at baseline.*
- *HbA1c at baseline was an independent predictor for the primary endpoint censored at 915 days, when adjusted for significant baseline characteristics in a multivariable model (HR 1.06, 95%CI 1.02-1.11, P=0.003).*
- *Rates of CV mortality and all-cause mortality were similar across HbA1c groups at baseline (KM estimate 2.6-4.3, P=0.21, for CV mortality and KM estimate 4.8-5.9, P=0.21, for all-cause mortality).*

Conclusion

This subanalysis of the ACCELERATE trial showed that HbA1c levels at study baseline are associated with CV outcomes in T2DM patients with a history of coronary artery disease and on optimal medical therapy.

References

LITERATURE - MENON V, KUMAR A, PATEL DR ET AL., - J AM HEART ASSOC. 2020, DOI: 10.1161/JAHA.119.014328.

Keto's Expiration Date

By: Dr. Ahmed Abdulmalek – Consultant family Medicine

Eating a ketogenic diet high in fat with minimal carbohydrates burns fat and restrains inflammation at first, but in the long term, it causes weight gain and impairs metabolic health, researchers studying mice report in the journal Nature Metabolism. The researchers found that when the mice first started on the ketogenic diet, their metabolically protective gamma delta T cells proliferated, but over time on the diet, they lost those cells. Without those protective immune cells, mice "have impaired glucose homeostasis," the authors write.

Given the limited long-term research on the ketogenic diet's effects in people, it's tempting to take the results of this study in mice and run with them. But because our clinician readers are treating human patients, not mice, we didn't cover this research, which doesn't have clear cross-species relevance.

Published: 20 January 2020

Ketogenesis activates metabolically protective γδ T cells in visceral adipose tissue

Emily L. Goldberg, rina Shchukina, Jennifer L. Asher, Sviatoslav Sidorov, Maxim N. Artyomov & Vishwa Deep Dixit.

https://www.nature.com/articles/s42255-019-0160-6

Quiz

A 53 years old type 2 diabetic patient attends the clinic at his GP practice. His Q Risk 2 assessment showed that he has a 12% risk of developing cardiovascular disease in the next 10 years . He had life style modification for the last six months with not much improvement in his lipid profile. Which statin should be offered ?

a) *Atorvastatin 20 mg*

b) *Atorvastatin 40 mg*

c) *Atorvastatin 80 mg*

d) *Simvastatin 20 mg*

e) *Simvastatin 40 mg*

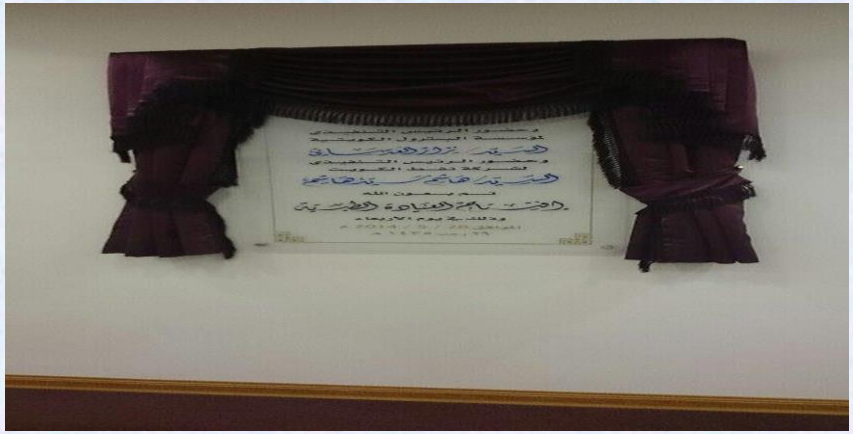
(The correct answer will be given in the last page)

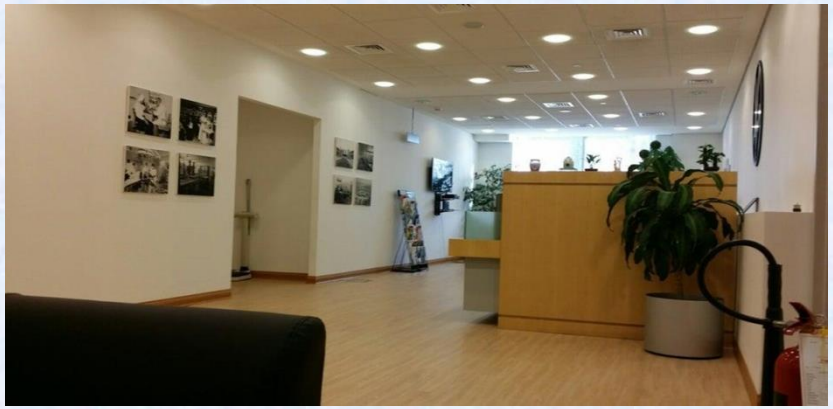
KPC clinic

By: Dr. Ahmed Abdulmalek /Consultant family Medicine

The new Kuwait petroleum clinic was reopened on the 28th of May 2005.It operates in the morning from 7 Am to 3 PM. It serves about 700 KPC employees .The clinic provides medical treatment to acute & emergency cases. It also runs a specialized clinic for chronic illnesses each Monday.

Beside that, it provides vaccination (e.g. flu vaccine) and runs the periodic medical examination (PME) of employees every two years.



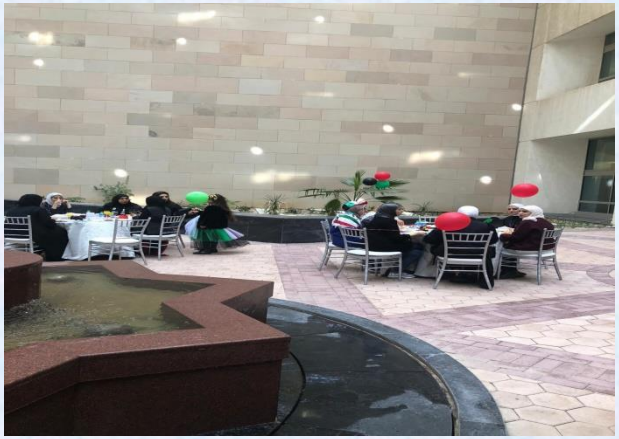


Activities

The department of Dermatology – Ahmadi hospital organized a marathon for Psoriasis patients named "Let Us Run Marathon". The activity took place in Ahmadi oasis on the 15th of February 2020.It lasted about three hours . Oxygen health club was the official sponsor of this activity and Dr. Zuhair Bitair from our Medical department shared in the activity. The activity was also sponsored by many Dermatology drug companies specializing in psoriasis treatment products. The winners of the competition were honored with health club subscriptions ,cups & shields.

Activities

The laboratories department - Ahmadi hospital organized the celebration of the national & liberation days of Kuwait. The event took place in the hospital on Wednesday the 19th of February 2020. The ceremony was attended by Dr. Saoud Al-Ajmi (Chief clinical officer), Mr. Shaker Al-Mutawa (head of laboratory department) and the laboratory staff.



Quiz Correct answer :

a) Atorvastatin 20 mg

Explanation : Nice guidelines recommends : if lifestyle modification is ineffective or inappropriate ,offer statin treatment after risk assessment. Offer atorvastatin 20 mg for the primary prevention of CVD to people who have a 10% or greater 10 years risk of developing CVD. Estimate the level of risk using the QRISK2 assessment tool. For people 85 years or older consider atorvastatin 20 mg as statins may be of benefit in reducing the risk of non fatal myocardial infarction.

Reference:

Nice Guidelines: Lipid modification therapy for the primary and secondary prevention of CVD.