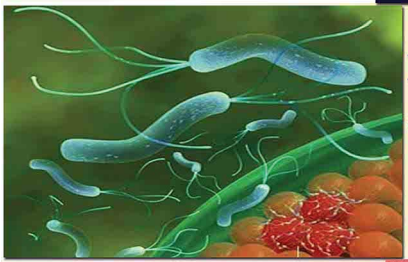
MEDICAL UPDATES

EGYPHAR

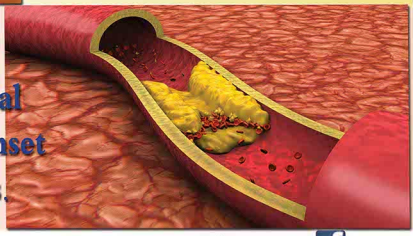
Issue No.:19 October 2014

The state of health in the Arab world, 1990–2010



Helicobacter pylori and Non-malignant Diseases.

Impacts of atorvastatin on blood lipids and arterial media thickness in new-onset type 2 diabetes patients.



Lancet. 2014 Jan 25

The state of health in the Arab world, 1990–2010

Mokdad AH et .al

Abstract

BACKGROUND:

The Arab world has a set of historical, geopolitical, social, cultural, and economic characteristics and has been involved in several wars that have affected theburden of disease.
Moreover, financial and human resources vary widelyacross the region. We aimed to examine the burden of diseases and injuries in the Arab world for 1990,2005, and 2010 using data from the Global Burden of Diseases, Injuries, and Risk Factors Study 2010 (GBD 2010).

METHODS:

We divided the 22 countries of the Arab League into three categories according to their gross national income: low-income countries (LICs; Comoros, Djibouti, Mauritania, Yemen, and Somalia), middle-income countries (MICs; Algeria, Egypt, Iraq, Jordan, Lebanon, Libya, Morocco, occupied Palestinian territory, Sudan, Syria, and Tunisia), and high-income countries (HICs; Bahrain, Kuwait, Oman, Qatar, Saudi Arabia, and the United Arab Emirates). For the whole Arab world, each income group, and each individual country, we estimated causes of death, disability-adjusted life years (DALYs), DALY-attributable risk factors, years of life lived with disability (YLDs), years of life lost due to premature mortality (YLLs), and life expectancy by age and sex for 1990, 2005, and 2010.

FINDINGS:

Ischaemic heart disease was the top cause of death in the Arab world in 2010 (contributing to 14·3% of deaths), replacing lower respiratory infections, which were the leading cause of death in 1990 (11·0%). Lower respiratory infections contributed to the highest proportion of DALYs overall (6·0%), and in female indivduals (6·1%), but ischaemic heart disease was the leading cause of DALYs in male individuals (6·0%). DALYs from non-communicable diseases--especially ischaemic heart disease, mental disorders such as depression and anxiety, musculoskeletal disorders including low back pain and neck pain, diabetes, and cirrhosis--increased since 1990.

Major depressive disorder was ranked first as a cause of YLDs in 1990, 2005, and 2010, and lower respiratory infections remained the leading cause of YLLs in 2010 (9·2%). The burden from HIV/AIDS also increased substantially, specifically in LICs and MICs, and road injuries continued to rank highly as a cause of death and DALYs, especially in HICs.

Deaths due to suboptimal breastfeeding declined from sixth place in 1990 to tenth place in 2010, and childhood underweight declined from fifth to 11th place.

INTERPRETATION:

Since 1990, premature death and disability caused by communicable, newborn, nutritional, and maternal disorders (with the exception of HIV/AIDS) has decreased in the Arab world--although these disorders do still persist in LICs--whereas the burden of non-communicable diseases and injuries has increased.

The changes in the burden of disease will challenge already stretched human and financial resources because many Arab countries are now dealing with both non-communicable and infectious diseases. A road map for health in the Arab world is urgently needed.

Helicobacter pylori and Non-malignant Diseases.

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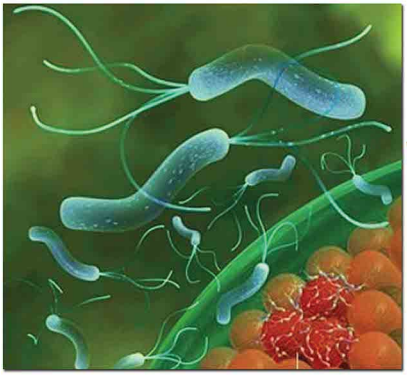
Abstract



Peptic ulcer bleeding and recurrence rate are strongly linked to Helicobacter pylori infection even if nonsteroidal anti-inflammatory drugs (NSAIDs) play a relevant role in this setting. Further studies confirm that H. pylori eradication lowers the risk of recurrent peptic ulcer bleeding.

Therefore, a test-and-treat strategy appears to be mandatory for patients with a history of ulcer bleeding and NSAIDs and/or aspirin use

Concerning gastroesophageal reflux disease (GERD), evidence clearly shows that H. pylori status has no effect on symptoms and treatment. Therefore, H. pylori treatment is not contraindicated in patients with GERD. The exact role of H. pylori in functional dyspepsia (FD) remains controversial.

Novel possible mechanisms by which H. pylori may elicit dyspeptic symptoms include alterations of gastric motility, as well as endocrine and acid-secretory abnormalities.
Hunger sensations, acid secretion, and gastrointestinal motility are regulated by ghrelin, particularly produced by the gastric enteroendocrine cell compartment.
The improvement of symptoms correlates with enhanced plasma ghrelin levels.
Apart from the need for more trials on this topic, these findings may give insight into the underlying pathophysiology of FD symptoms.
Recent reports suggest that the presence of bacterial DNA in the oral cavity may be relevant to its transmission.
A potential protective role of H. pylori on inflammatory bowel diseases needs to be better elucidated.

Prevention and Treatment of NSAID Gastropathy.

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Abstract

OPINION STATEMENT:

Nonsteroidal anti-inflammatory drug (NSAID) treatment will be necessary as part of our therapeutic armamentarium for many years to come.

Therefore, safe prescription is mandatory in order to prevent adverse events.

In the last two decades, new strategies and new drugs have been developed to reduce NSAID-associated upper gastrointestinal (GI) adverse events.

Although the implementation of guidelines into clinical practice takes time, several studies have shown a recent and profound decrease in hospitalizations due to upper GI complications, which has been linked to widespread use of proton pump inhibitors (PPIs), better NSAID prescription, and decreased prevalence of Helicobacter pylori infection.

This is encouraging.Safe NSAID prescription should be straightforward since the most relevant aspects are clinical in nature.

Before issuing any prescription, three key questions should be considered:1) Is NSAID treatment necessary for this patient?2) What cardiovascular (CV) and GI risk factors does this patient have?3) What is the most suitable NSAID for this patient?GI and CV risk are easy to estimate, and we know that these risks are not the same for all NSAIDs.

Selective cyclooxygenase (COX)-2 inhibitors, like celeSelective cox2 Inhib. at usual doses, carry the lowest GI risk and are the best option in patients with moderate/high GI risk without high CV risk Gastroprotective therapy (PPI as the drug of choice) should be considered if a non-selective NSAID is prescribed.

For those at the highest risk, a combination of PPI plus a Selective cox2 Inhib. is the best option. Also, eradication of H. pylori infection in patients with previous peptic ulcer or in NSAID-naïve users must be considered.

Naproxen is the best option in patients with high CV risk and low/moderate GI risk.Patients taking aspirin represent a real challenge for treatment, since interaction with frequently prescribed NSAIDs (eg, ibuprofen/naproxen) may alter its antiplatelet effect, representing a potential clinical problem. Switching treatment (e.g. taking aspirin before NSAID dosing) may not be an alternative since interaction may persist, especially when taking enteric-coated aspirin. Changing NSAID treatment to diclofenac/celeSelective cox2 Inhib./etoriSelective cox2 Inhib. may also not be an option in patients with high or previous CV event history. Under these circumstances, careful prescription should be considered at the individual patient level.When dyspepsia develops in an NSAID user, PPI co-therapy plus reduction of the NSAID dose or a change in the type of NSAID are valid alternatives, but clinical experience shows that, for some patients, stopping NSAID therapy may be the only option.

After a bleeding episode, most patients can be managed with alternative therapy to NSAIDs, but if needed, a Selective cox2 Inhib. plus a PPI and H. pylori eradication is a safe alternative.

Zhonghua Liu Xing Bing Xue Za Zhi. 2014 Jun

Impacts of atorvastatin on blood lipids and arterial media thickness in new-onset type 2 diabetes patients.

Yu D1, Wang Y2, et al

[Article in Chinese]

Abstract

OBJECTIVE:

To analyze the impact of atorvastatin on blood lipids and arterial media thickness (IMT) in new-onset type 2 diabetes patients.

METHODS:

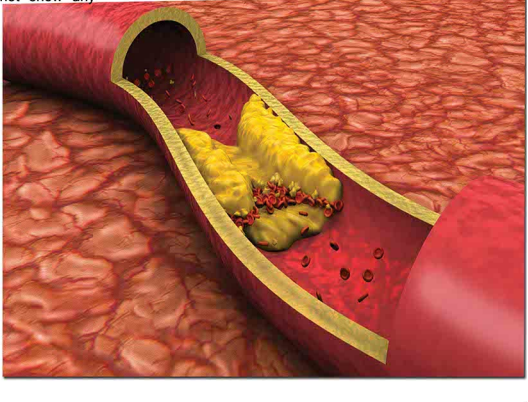
333 patients, 30-70 years old and diagnosed within one year as type 2 diabetes, were selected from the Chinese Diabetes Complication Prevention Study (CDCPS) to take part in this study.
Changes of blood lipids and IMT of carotid, femoral and iliac artery pre and post the administration of atorvastatin were tested and followed for 24 months.

RESULTS:

Total cholesterol, triglycerides and low-density lipoprotein decreased significantly (P = 0.000) and maintained at a low level.
The carotid artery IMT decreased significantly (P = 0.022) at the end of this study, but the femoral and iliac artery IMT did not show any obvious change.
There were no serious adverse events noticed, during the study period.

CONCLUSION:

Long-term use of atorvastatin seemed to be safe and effective in reducing blood lipids in patients with type 2 diabetes thus could delay the development of atherosclerosis.



Atorvastatin protects against cerebral ischemia/reperfusion injury through anti-inflammatory and antioxidant effects.

Tu Q1, et al.

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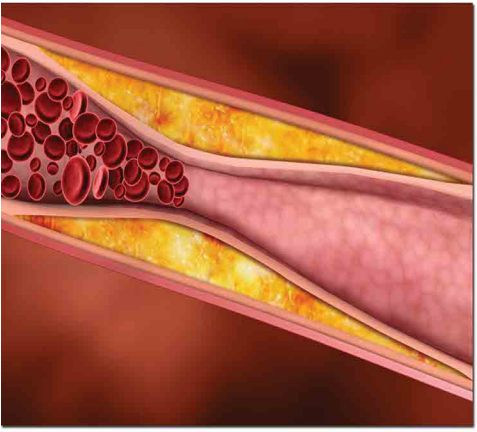
In addition to its lipid-lowering effect, atorvastatin exerts anti-inflammatory and antioxidant effects as well.



In this study, we hypothesized that atorvastatin could protect against cerebral ischemia/reperfusion injury. The middle cerebral artery ischemia/reperfusion model was established, and atorvastatin, 6.5 mg/kg, was administered by gavage.

We found that, after cerebral ischemia/reperfusion injury, levels of the inflammation-related factors E-selectin and myeloperoxidase were upregulated, the oxidative stress-related marker malondialdehyde was increased, and superoxide dismutase activity was decreased in the ischemic cerebral cortex.

Atorvastatin pretreatment significantly inhibited these changes.

Our findings indicate that atorvastatin protects against cerebral ischemia/reperfusion injury through anti-inflammatory and antioxidant effects.

Diabetes Metab Res Rev. 2007 Mar;

Gliclazide protects human islet beta-cells from apoptosis induced by intermittent high glucose.

Del Guerra S1, et al

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Abstract

BACKGROUND:

Decreased beta-cell mass, mainly due to apoptosis, is crucial for the development and progression of type 2 diabetes.
Chronic exposure to high glucose levels is a probable underlying mechanism, whereas the role of oral anti-diabetic agents (sulphonylureas in particular) is still unsettled.

METHODS:

To directly investigate more on such issues, we prepared isolated human islets, which were then cultured for 5 days in continuous normal glucose concentration (NG, 5.5 mmol/L) or normal and high (HG, 16.7 mmol/L) glucose levels (alternating every 24 h), with or without the addition of therapeutical concentration (10 micromol L) of gliclazide or glibenclamide.

RESULTS:

Intermittent high glucose caused a significant decrease of glucose-stimulated insulin secretion, which was not further affected by either sulphonylurea. Apoptosis, as assessed by electron microscopy, was also significantly increased by alternating high glucose exposure, which was accompanied by altered mitochondria morphology and density volume, and increased concentrations of nitrotyrosine, a marker of oxidative stress. Gliclazide, but not glibenclamide, was able to significantly reduce high glucose induced apoptosis, mitochondrial alterations, and nitrotyrosine concentration increase.

CONCLUSION:

Therefore, gliclazide protected human beta-cells from apoptosis induced by intermittent high glucose, and this effect was likely to be due, at least in part, to the anti-oxidant properties of the molecule.