CHEST PAIN IN PEDIATRIC PATIENTS

THESIS
Submitted for the Partial Fulfillment of Master Degree in Pediatrics

by

Mazar Mohammed Ali Hussein
M.B.B.CH Ommderman Islamic University

Supervised by

Prof. Dr .Yasser Hussien Kamel
Professor of Pediatrics
Faculty of Medicine
Cairo University

.

Prof. Dr. Wael Nabil Lotfy
Professor of Pediatrics
Faculty of Medicine
Cairo University

Assist. Prof. Dr. Ranya Aly Hegazy
Assistant professor of Pediatrics
Faculty of Medicine
Cairo University

Cairo
2010

Abstract

Cardiac chest pain among Egyptian children referred to a tertiary arrhythmology service. Retrospective study reviewing files of patients presenting with chest pain over the past 5 years to the arrhythmology clinic. The study included 475 files of children who referred with primary complaint of chest pain from 2004 to 2008. 199 male and 276 female.

Key word:

Chest pain pediatric

Acknowledgments

First and foremost thanks to Allah who gave me the strength to finish my work.

I had an amazing committee of supervisors who kindly and patiently added their prints to this work and this is an opportunity to acknowledge their influence and publicly express my gratitude to them.

I cannot find enough words to express my thanks to Prof. Dr .Yasser Hussien Kamel, Professor of Pediatrics, Cairo University, for his help, support and encouragement to accomplish this work.

Deeply from my heart I want to thanks Prof. Dr. Wael Nabil Lotfy, Professor of Pediatrics, Cairo University who always been offered me continuous support and advice step by step to accomplish this work. It truly would be difficult to continue without his support.

From my depth of my heart I want to thank Prof. Dr. Ranya Aly Hegazy, Assistant Professor of Pediatrics, Cairo University for her assistance and help, I am really lucky to have a supervisor like her. Her continuous support motivated me to accomplish this work. She will be always a figure in my life.

Words cannot express my gratitude and appreciation to the medical staff who work in arrhythmology service, Pediatric Hospital, Cairo University.

My endless thank to the patients by whom and for whom my present work is done.

List of figures

List of Tables

List of abbreviations

ACC/ AHA	American college of cardiology/American heart association
ACS	Acute chest syndrome
ALCAPA	Left coronary artery from pulmory artery
AMI	Acute myocardial infarction
APCA	African Palliative Care Association
AS	Aortic stenosis
ASD	Atrial septal defect
BHR	Bronchial hyper responsivness
CBC	Complete blood count
CHF	Congestive heart failure
CK	Creatine kinase
CNS	Central nervous system
COX	Cyclo oxygenase
CPK	Creatine phosphokinas
cTnI	Cardiac troponin I
cTnT	Cardic troponine T
CXR	Chest X-ray
D- TGA	dextro-transposition of the great arteries
DCM	Dilated cardiomyopathy
ECG	Electrocardiogram
ECHO	Echocardiogram
ED	Emergency department
ESR	Erythrocyte sedimentation rate
FLACC	Face, Legs, Activity, Cry, Consolability
FMF	Familial Mediterranean fever
GER	Gastro esophageal reflux
HCM	Hypertrophic cardiomyopathy
HF	Heart failure
HM	Holter monitor
IASP	International association for study of pain

JRA	Juvenile rheumatoid arthritis
KD	Kawasaki disease
LD-1	Lactose dehydrogenase isoenzyme I
LVH	Left ventricle hypertrophy
MVP	Mitral valve prolapsed
NIPS	Neonatal Infant Pain Scale
NSAIDS	Non steroidal anti inflammatory drugs
OPS	Objective pain scale
PIPP	Premature Infant Pain Profile
PMI	Point of maximal impulse
PVCS	Premature ventricular contractures
RBBB	Right bundle branch block
RHD	Rheumatoid heart disease
SLE	Systemic lupus erythematosus
SVT	Supra ventricular tachycardia
TENS	Trans electrical nerve stimulation
VSD	Ventricular septal defect
VZV	Varicella zoster virus
WHO	World health organization
WMA	Wall motion abnormality
WPW	Wolf Parkinson white

Introduction

Children's chest pain often causes anxiety to patients and parents. It has numerous causes, most of which are benign. Serious illnesses occasionally present with this symptom. Chest pain accounts for 0.2% to 0.6% of patient visits to pediatric outpatient clinics or emergency departments. Chest pain affects children of all ages, with a peak incidence between 12 and 14 years, there is no sex predilection, although traumatic causes are more frequent in males and costochondritis and psychogenic causes are more frequent in females. ***Rowe et al,1990***

Because it is a subjective experience and difficult to quantify, epidemiological surveys of the prevalence of chest pain and physiological studies of its mechanisms are limited. ***Selbst &Brenner, 1997***

Chest pain is a problem, it represents the patient's/family's greatest fear because they know that in adults it often signifies myocardial infarction (heart attack). However, cardiac causes of chest pain are among the least likely causes in pediatric patients. However, chest pain must always be thoughtfully considered because it may be the first signal of serious, potentially lethal disease. ***Minocha & Teitelbaum et al, 2007***

Common causes of chest pain include idiopathic 12%-85%, pulmonary 12%-21% and musculoskeletal diseases 12%-31%.Less common causes are gastrointestinal 4%-7%, cardiac diseases 4%-6% and others as spinal cord or nerve root compression, and breast related pathologic condition and castleman disease (lymph node neoplasm). ***Kocis, 1999***

Chest pain in children and adolescents is common, but is generally benign. Cardiac (heart-related) causes of chest pain are uncommon. Chest pain with exercise or that associated with fast heart beat, dizziness, or

fainting can indicate a cardiac cause. Chest pain that occurs at rest without other associated symptoms is not typically due to a heart problem. ***Clements, 2008***

The most common diagnostic tests are chest X-RAY, ECG, determination of hemoglobin concentration and of the leukocyte count. Other tests rarely ordered are urinalysis, cardiac enzymes and erythrocyte sedimentation rate. Patients with chest pain of cardiac origin may need emergency hospitalization and are likely to require further diagnostic evaluation by a cardiologist for coronary artery disease or valvular dysfunction. This may entail echocardiography, cardiac catheterization, treadmill testing, or coronary angiography with possible angioplasty or stent placement. Consultation with a pulmonologist is needed for patients who might require fiberoptic bronchoscopy, pleural biopsy, or specialized pulmonary function testing. Similarly, if invasive procedures are contemplated to evaluate chest pain of possible esophageal origin or somewhere in the abdomen, referral to a gastroenterologist is warranted. In selected cases of intractable chest pain of presumed psychological origin, referral to a psychiatrist can be helpful. ***Murray et al, 1994*.**

Most patients/families with chest pain simply want reassurance that symptoms are not cardiac in origin. A careful history and physical exam are most important; however, a normal CXR and ECG provide therapeutic reassurance to the patient/family. Further cardiology consultation is rarely required but should be considered with patients experiencing chest pain with exercise, a history of Kawasaki disease, Marfan syndrome (this is an emergency), and for those patients with persistent chest pain. Appropriate therapy of identified pulmonary, gastrointestinal, or musculoskeletal problems is required. ***Teitelbaum et al, 2007.***

Objective:

The present work aims at identifying the prevalence of cardiac chest pain among Egyptian children referred to a tertiary arrhythmology service.

We aim to delineate its main causes, lines of investigations, their yield and ways of management.

Patients and Methods:

Study design:

Retrospective study reviewing files of patients presenting with chest pain over the past 5 years to the arrhythmology clinic. 475 files of children who referred with primary complaint of chest pain were reviewed from 2004 to 2008.

Study setting:

Arrhythomlogy clinic, Department of Pediatrics, Faculty of Medicine

Data collection:

1. Age, sex
2. Pain analysis: Onset, Duration, Course

   Precipitating events

   Offsetting events
3. Investigations: 12 lead ECG, transthoracic Echo, ambulatory 24 hour ECG.

Statistical analysis:

Statistical Package of Social Sciences (SPSS) version 11,0 was used for analysis of data. Analysis of differences in variables of groups was performed by Chi- square test. The significant level was set at p value smaller than 0.05.

PHYSIOLOGY OF PAIN

Pain is defined by the International Association for the Study of Pain (IASP) as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage. Thus pain has objective, physiologic sensory aspects as well as subjective emotional and psychological components. ***Venugopal &Swamy, 2006***

Pain is a complex phenomenon and includes both sensory discriminative and motivational –affective components. It is a sensory experience that is accompanied by emotional responses and by somatic and autonomic motor adjustments. ***Berne & Levy et al, 2004***

Pain, is important because it teaches us to avoid subsequent encounters with potentially damaging stimuli. Pain is also important clinically, because it is a sign that tissue damage may have occurred. ***Germann & Stanfield, 2002***

Pain is a protective mechanism, it occurs whenever any tissues are being damaged, and it causes the individual to react to remove the pain stimulus. Pain has been classified into two major types: fast pain and slow pain. Fast pain is felt within about 0.1 second after a pain stimulus is applied, whereas slow pain begins only after 1 second or more and then increases slowly over many seconds and sometimes even minutes. Fast pain is also described by many alternative names, such as sharp pain, pricking pain, acute pain, and electric pain. This type of pain is felt when a needle is stuck into the skin, when the skin is cut with a knife, or when the skin is acutely burned. It is also felt when the skin is subjected to

electric shock. Slow pain also goes by many names, such as slow burning pain, aching pain, throbbing pain, nauseous pain, and chronic pain. This type of pain is usually associated with tissue destruction. It can lead to prolonged, unbearable suffering. ***Guyton, 2006***

PAIN RECEPTORS:

Pain receptors are free nerve endings, they are widespread in the superficial layers of the skin as well as in certain internal tissues, such as the preiosteum, the arterial wall, the joint surfaces, and the falx and tentorium in the cranial vault .Most other deep tissues are only sparsely supplied with pain endings; nevertheless, any widespread tissue damage can summate to cause the slow-chronic-aching type of pain in most these areas. ***Guyton, 2006***

A stimulus that causes tissue damage usually elicits a sensation of pain. Receptors for such stimuli are known as **nociceptors.** They respond to intense mechanical deformation, excessive heat, and many chemicals, including neuropeptide transmitters, bradykinin, histamine, cytokines, and prostaglandins, several of which are released by damaged cells. These substances act by combining with specific ligand-sensitive ion channels on the nociceptor Plasma membrane. ***Vander, 2003***

Nociceptors or nocireceptors (pain receptors) are responsive to stimuli that potentially cause injury. They are of two types. The fast adapting Aδ fiber mechanical nociceptors are high-threshold, finely myelinated afferents that originate superficially in the skin. The slowly adapting C-polymodal nociceptors are unmyelinated afferent fibers that originate in the deeper cutaneous tissue, and respond to various mechanical, thermal and chemical stimuli. ***Germann & Stanfield, 2002***

There are several different types of sensory receptors in the skin, each of which is specialized to be maximally sensitive to one modality of sensation. Nociceptors are also free sensory nerve endings of either myelinated or unmyelinated fibers. The initial sharp sensation of pain, as from a pin-prick, is transmitted by rapidly conducting myelinated axons, whereas a dull, persistent ache is transmitted by slower conducting unmyelinated axons. These afferent neurons synapse in the spinal cord, using substance P (an eleven-amino-acid polypeptide) and glutamate as neurotransmitters. ***Fox, 2003***

Myelinated Aδ fibers, 2-5 μm in diameter, conduct at rates of 12-30m⁄s. The other consists of unmyelinated C fibers 0.4-1.2 μm in diameter. These later fibers are found in the lateral division of the dorsal roots and are often called dorsal root C fibers. They conduct at the low rate of 0.5-2 m⁄s. Both fiber groups end in the dorsal horn. A δ fiber terminates primarily on neurons in laminas I and II. There is abundant evidence that the synaptic transmitter secreted by primary afferent fibers subserving pain is substance P. An important recent event was the isolation of vanilloid receptor-1 (VR1). Vanillins are a group of compounds, including capsaicin that causes pain. This necessitated revision of the concept that a single pathway carries pain and only pain to the cerebral cortex. The VR1 receptors respond not only to the pain-causing agents such as capsaicin but also to protons and to potentially harmful temperatures above 43 °C. **Ganong, 2003**

Pain stimulus

Pain can be elicited by multiple types of stimuli. They are classified as mechanical, thermal, and chemical pain stimuli. ***Guyton, 2006***

The adequate stimulus is the stimulus, for which the receptor has a lower energy threshold than for other stimuli (the stimulus to which the receptor is most sensitive). The adequate stimulus for pain receptors is mechanical deformation, extreme temperature or tissue damage. When nociceptors become sensitised (more responsive), their thresholds are reduced, thus causing hyperalgesia (hypersensitivity to pain). Many substances such as bradykinin, histamine, leukotrienes, prostaglandins, serotonin, and K+ that are often released near damaged or dying cells sensitise nociceptors. K+ activates the nociceptors. Substance P is also released from polymodal nociceptors through an axon reflex with antidromal signal transduction in afferent group IV fibres, causing hyperalgesia, vasodilatation and increased capillary permeability. ***Germann & Stanfield, 2002***

Nociceptors respond to noxious stimuli that can produce tissue damage. There are two major classes of nociceptors: thermal or mechanical nociceptors and polymodal nociceptors. Thermal or mechanical nociceptors are supplied by finely myelinated A-delta afferent nerve fibers and respond to mechanical stimuli such as sharp, pricking pain. Polymodal nociceptors are supplied by unmyelinated C fibers and respond to high-intensity mechanical or chemical stimuli and hot and cold stimuli. Damaged skin releases a variety of chemicals, including bradykinin, prostaglandins, substance P, K+, and H+, which initiate the inflammatory response. The blood vessels become permeable, and, as a