Acute Asthma in the Casualty Department, Emergency room assessment and management at H.F. Verwoerd Hospital Pretoria

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OPSOMMING:
Die belangrike kliniese tekens van akute asma word bespreek. Insiggewende inligting in verband met die waarde van eenvoudige kliniese waarnemings soos byvoorbeeld die graad van pulsus paradoxus word verduidelik en die betekenisvolle gebruik van spesiale ondersoeke word uiteengesit. Noodbehandeling geniet dan aandag op 'n redelik didaktiese maar gebalanceerde manier.

Dr. C.J. van der Merwe graduated with MBChb at the University of Pretoria in 1955. After completion of the Internship at the Krugersdorp General Hospital he returned to the then Pretoria General Hospital as Senior House Officer to the Department of Surgery.

During 1957 he travelled to the United Kingdom and after six months as Senior House Officer he was appointed Surgical Registrar to the Birmingham Regional Hospital Board. A post he held for 2½ years.

On return to South Africa he worked as Surgical Registrar at King Edward VIII Hospital Durban and after 2 years was appointed Senior Surgical Registrar to the Department of Plastic and Reconstructive Surgery, Wentworth Hospital, Durban.

After a second visit to the United Kingdom where he was appointed Senior Surgical Registrar he returned to the R.S.A. and practised as a family practitioner in Pretoria.

During 1970 he was appointed to the staff of the H.F. Verwoerd Hospital. In 1973 he was appointed Senior Medical Officer and in 1974 Principle Medical Officer in charge of the Casualty Department of the H.F. Verwoerd Hospital, a position he is still holding at present.

In 1977 he gained the qualification M. Prax Med and was appointed Senior Lecturer to the Department of Family Medicine, University of Pretoria.

Recognising acute asthma presents few problems. It has nevertheless become evident that "sudden unexpected" deaths frequently occur. The reason for these catastrophies is usually misassessment and undertreatment.

It is important to emphasise that the clinical features of asthma do not necessarily reflect the physiological alterations present. It is for the same reason that the degree of response to treatment may be poorly correlated with existing physiological changes associated with inadequate treatment.

It can thus be fallacious to accept that an improvement in the clinical picture is necessarily an indication for cessation of therapy. It is therefore crucial to utilize available laboratory assessment to prevent undertreatment and too early discharge.

Pathophysiology of acute asthma: The pathophysiologic hallmark of asthma is a reduction in airway diameter. This is brought about by: (1) Contraction of smooth muscle; (2) Oedema of bronchial wall; (3) Thick secretions.

The relative contribution of each of these to the patients' overall ventilatory impairment vary. The net result however is: (1) An increase in airway resistance; (2) decreased forced expiratory volumes and flow rates; (3) hyperinflation of the lungs; (4) increased work load of breathing; (5) abnormal ventilation and perfusion; (6) abnormal blood gasses.

Assessment of the acute asthmatic episode: Assessment is based on questioning and careful clinical examination assisted by laboratory and X-ray examination. It is important to realize that a given episode of asthma may be of greater severity than is superficially indicated by the clinical picture.

Symptoms: Dyspnoea is the major complaint. This is a reflection of the increased workload of breathing. An acute asthma attack can be a serious problem in patients with a...
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history of previous hospital admissions for a similar attack, in
those who had been on steroid therapy and in patients on whom
the usual therapy has now become ineffective.

SIGNs:
PULMONARY SYSTEM
Dyspnoea:
Dyspnoea is obvious in these
patients and expiration is
prolonged.
The patients insist on sitting in an
upright position (this causes a
reduced venous return and
lessens the effect of intra-abdo-
minal pressure on the thoracic
cage). The shoulder girdle is
thrust forward so as to compli-
ment the action of the accessory
muscles which may become
necessary for breathing.
Retraction of the sternomastoid
muscle during inspiration is
obvious in the patient with a
serious asthma attack.

Cyanosis:
The development of central
cyanosis is a serious sign
because the asthmatic patient
normally hyperventilates even in
the presence of moderately
severe obstructive airways dis-
ease. Because hyperventilation
continues during sleep it cannot be
explained on emotional
grounds. The increased ventila-
try drive is due to afferent vagal
stimulation. Asthma is a condi-
tion which causes stimulation of
the respiratory centre via the
vagus and results in hyperventila-
tion. The net result is an elevation
of PO2 and a reduced PCO2
during the early phase of this
disease.

It must be emphasised that the
presence of central cyanosis indicates serious respiratory
disease.

Auscultation:
The characteristic auscultatory
findings are of prolongation of
expiration and the presence of
respiratory ronchi.

Equal inspiratory and expiratory
phases indicates severe obstruc-
tion. It is important to realise that a
"silent" chest can indicate a
serious degree of obstructive
airways disease.

Hyperinflation:
Thoracic overinflation is present
during episodes of acute asth-
ma and alters with the degree of
airway obstruction. Even when
bronchospasm has been reliev-
ed, some degree of temporary
hyperinflation may still persist.

CARDIOVASCULAR SYSTEM:
Tachycardia:
A sinus tachycardia greater than
110/min. can be an indication of
a severe attack.(2) The degree of
tachycardia may be related to the
level of arterial hypeoxaemia.(3)
A drug induced tachycardia must
be born in mind in patients
receiving treatment.

Pulsus paradoxus:
This is an exaggeration of the
normal reduction in arterial
pressure (usually 5mm of
mercury) during inspiration and
is associated with a combination of
high lung volume and increased
intrathoracic-pressure changes.(4)
Pulsus paradoxus is not present in patients with a
FEV1 higher than 60% of normal
but is present in all patients with a
FEV1 less than 20% and a
significant arterial paradoxus is
found in 66% patients with FEV1
of less than 40%. An inspiratory
fall in systolic pressure of more
than 10 mmg mercury is significant
proof of airway ob-
struction with severe hyperinfla-
tion of the lungs. The degree of
paradox is directly related to the
degree of airways obstruction.
Pulsus paradoxus usually dis-
appears rapidly, usually within a
few hours of the initiation of
therapy. (The degree of paradox
is measured with a blood
pressure manometer).

SPECIAL INVESTIGATIONS
PERFORMED IN THIS PRO-
GRAMME

Bloodgas analysis:
This investigation is performed
routinely on admission and
during treatment. Hypoxaemia
without hypecapnia(5) is the usual
finding. Carbon dioxide retention
appears to be less common in
patients suffering from severe
asthma than in those with chronic
obstructive disease with superim-
posed acute infections. A reduced
PCO2 to 60mm Hg (with a
reduced PCO2) is the common
bloodgas picture. An elevated
PCO2 (and possibly even a
normal PCO2) indicates serious
impairment of ventilation and
vigorous therapy is called for.

Electrocardiogram:(7)
Sinus tachycardia is always
present frequently accompanied
by other changes. The most
common changes are right axis
deivation, clockwise rotation of
the heart, P-pulmonale, S-T
segment or T wave abnormalities.
Sinus tachycardia diminishes
progressively as the asthma
diminishes and improvement of
the severe electrocardiographic
abnormalities usually follows
rapidly after the initiation of
therapy and all these changes
revert to normal after relief of the
asthmatic episode.

Chest Röntgenogram:
The most important use of the
X ray is to exclude complications.
The most common is pneumonia
or atelectasis due to mucus
obstruction(8) especially in the
right middle lobe. The changes of
a pneumothorax and a pneu-
moniemediastinum can be demon-
strated. The degree of hyperinfla-
tion is difficult to evaluate.

MANAGEMENT OF ACUTE
ASTHMA:
Oxygen:
Either a concentration mask
(Venturi type) is used or long
nasal catheters.

Fluid administration:
An infusion of 5% Dextrose and
water is preferred to enhance
rehydration of the interstitial
tissues and thus reduce the
viscosity of the bronchial secre-
tions. As much as 1 litre of the
solution is administered during
the first hour (provided no
contra-indication for fluid loading
exists) and an additional 1 litre
during the following 3 hours.

Bronchodilators:
A piggy back infusion (150ml
Dextrose and water) is chosen for
the administration of drugs with
bronchodilating properties. For
the patient with a heart rate of
115/min. or less aminophyllin
500mgm and 10mgm hexopre-
noline (β-adrenergic stimulant)
is added to the infusion. This
solution is titrated for the
administration of aminophyllin at
a rate of 6mgm/kg/hour during
the first 20 minutes followed by a
maintenance dosage of 0,9mgm/
kg/hour. In the event of the heart
rate being more than 115/min.,
the hexoprenaline is omitted and
the aminophyllin dosage is
adjusted accordingly.

Corticosteroid therapy:
The administration of steroids
during an acute attack of asthma
is mandatory. Hydrocortisone
200mgm I.V. is administered with
increased dosage to 400mgm in
patients already receiving ste-
roids. It must be emphasised that
the optimum dosage of steroids is
not equivocal.

The intravenous therapy is
followed by oral administration of
prednisolone (on a sliding scale).

Steroids in very large doses
(measured in grams of initial
doses of hydrocortisone) are
suggested by some to avoid
steroid resistances(9). The ad-
ministration of very large doses
of I V methylprednisolone have
been reported to be without side
effects(10).

Adrenaline:
This agent is not included in our
programme because of the
effectiveness of β-B-adrenergic
stimulants.

Sedation:
Sedation may be related to the
failure of effective treatment and
is not routinely administered.

Antibiotics:
Prophylactic antibiotic therapy is
defined. This regime is directed
theatre of the emergency care of the acute
asthmatic patient, and does not
cover other forms of therapy for
less urgent cases.

SURVEILLANCE IN THE
EMERGENCY ROOM:
Our patients remain in the emer-
gency room for approximately
four to six hours except in the
event of a life threatening attack in
which case admission to the
pulmonary intensive care is
arranged, after initial primary
critical care has been administer-
ed.

An assessment of the degree of
improvement is made on clinical
grounds, repeat bloodgas ana-
lysis and measurement of peak
airflow.
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On completion of treatment the peak airflow is measured. Patients with a 50% flow or less will be admitted and those with a reading of 50-60% will be observed in the emergency room for an extended period.

IDENTIFICATION OF THE HIGH RISK ASTHMATIC PATIENT:
A patients examined in our emergency room, presenting with any one or more of the undermentioned, will be admitted:

1. A history suggestive of a previous life threatening acute asthma attack.
2. Central cyanosis
3. Pulsus paradoxus in excess of 10mm Hg
4. Diminution of consciousness
5. Exhaustion
6. Pneumothorax or pneumomediastinum
7. Gross hyperinflation on chest röntgenogram
8. P.O. less than 60mm Hg
9. Significant elevation of PCO2
10. Electro cardio graphic changes suggestive of pulmonary hypertension in the presence of an acute attack
11. All patients presenting in a first attack and all young patients.

CONCLUSION:
Patients with an acute asthmatic attack should be carefully evaluated before and after emergency room therapy. This will ensure the admission of all high risk asthmatic patients after primary critical care measures had been taken. This will obviate the discharge of patients who have not yet adequately recovered.

The management for acute asthma is based on adequate fluid therapy, administration of bronchodilators and corticosteroids therapy.

References:

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