Arterial Blood Gases (ABG)

BY

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Simple &practical

Introduction to ABG

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Basics:

➢ $CO2 + H20 \longrightarrow H2CO3 \longrightarrow H (+) + HCO3 (-)$ ….. (And vise versa).

By the effect of Carbonic anhydrase enzyme which acts in both directions

This equation represents the buffer system of acid base status in the body.

- ➢ H = acid (+)
- ➢ HCO3 = base (-) or alkali
- ➢ PH : is the value that reflecting acid base status

Normally the blood and body fluids are neutral (i.e the media is not acidic or alkaline)

Because there is balance between Acid and base values

- *Acid:* represented by H^+ or $CO2^-$
- Base or alkali: represented by $HCO3^-$

➢ So Normal PH value ranges from: 7.45 - 7.35

- PH < 7.35 ----> Acidosis
- PH > 7.45 ----> Alkalosis

**** Control of Acid base status:**

➢ There are 2 major systems which are responsible for Control of Acid base status:

"Respiratory System Vs Metabolic System."

Respiratory system is "Rapid"

I- Respiratory System which is composed of:

A) Lungs (ventilation): bronchial tree + Gas exchange unit (Alveolo- capillary unit)

B) Respiratory muscles which are responsible for Inspiration & Expiration process (help lungs mechanically).

Muscles + NMJ + Nerves.

C) Respiratory centers:

i. Central control (brain stem)

Central chemoreceptors which are responsible for monitoring of CO2 level in blood.

It controls CO2 level by stimulation or inhibition of respiratory muscles, according to the state of Acidity or Alkalinity of the blood.

- If media is acidic (↑ H^+) ---> elimination of H+ by elimination of CO2 ----> CO2 wash --> by stimulation of resp muscles --- >Hyperventilation or Tachypnea.

Result: ↓ PaCO2 = induction of alkalosis by respiratory system

⬇⬇

Respiratory alkalosis

- If media is Alkaline (↓H) ---> addition of H^+ by CO2 retention ---> by inhibition of respiratory muscles ---> hypoventilation or Bradypnea.

Result: PaCO2 ↑ = induction of acidosis by respiratory system

⬇⬇

Respiratory Acidosis

ii. Peripheral respiratory center: (chemoreceptors in carotid bodies)

Which are responsibe for O2 monitoring in blood

- If there is ↓ in blood O2 = hypoxia or hypoxemia ---> stimulation of respiratory muscles
  ---> Increase ventilation for O2 compensation (get more O2) to correct O2 in blood
  ---> If still not corrected ---> more stimulation of respiratory muscles
  --->Hyperventilation or tachypnea ---> unwanted CO2 wash ---> ↓PaCO2

⬇⬇

Respiratory alkalosis

- Mild hypoxia --> normal PaCO2 level (Normocapnea)
- Marked or prolonged hypoxia -- > low PaCO2 (hypocapnea)
- Hypoxia + respiratory alkalosis = Hypoxia + Hypocapnia

⬇⬇

[Type 1 Respiratory Impairment]

II- Metabolic System which is composed of : Kidneys (mainly)+ buffer system

A) Kidneys (slow)

Kidneys are able to control acid base status by the following mechanisms

i) Bicarbonate reabsorption via tubules

ii) Secretion of H^+ in urine

So,

- If there is an acidosis, the kidneys will correct this condition by induction of alkalosis

----->↑reabsorption of HCO3 --> ↑ HCO3

⬇⬇

Metabolic alkalosis as compensation

----->Secretion of H in urine to ↑ acidity

- If there is an alkalosis , the kidneys will correct the condition by induction of acidosis

--->↓reabsorption of HCO3 --> ↓HCO3

⬇⬇

Metabolic acidosis as compensation

B) buffer system (weak)

It acts only in acute respiratory abnormalities (acidosis& alkalosis) to compensate for acidity or alkalinity because the kidneys are slow organs

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Oxygen Status:

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- O2 is present in blood in 2 forms

a) O2 bound to HGB - expressed in O2 saturation
- Value (≥ 95 %)

b) Free O2 in blood - it has PaO2 (Partial pressure of arterial O2)
- value (80 - 100) mmHg

- Hypoxia means O2 saturation < 95% and/or PaO2 < 80 mmHg.

CO2 Status:

==========

It is the product of catabolism.

- PaCO2 (partial pressure of arterial O2)
- Value : 35 - 45 mmHg ~ median 40
- PaC02 reflects the RESPIRATORY SYSTEM (ALL PARTS)

- PaCO2 = CAPNIA state

↳ if PaCO2 is higher than normal or expected ↑ = high H+

-----> This means Hypercapnia or Respiratory acidosis.

↳ if PaCO2 is lower than normal or expected ↓ = low H+

-----> This means Hypocapnia or respiratory alkalosis.

HCO3- Bicarbonate Or simply (Bicarb)

==========

- It represents the base status
- It presents in blood and in intestine

- It reflects the Metabolic System (kidney and Buffer system(
  - Normal value (22 - 26) mmHg With median ~ 24

↳ If HCO3 is Higher than normal or expected ↑ --> excess base = alkali → metabolic alkalosis.

↳ If HCO3 is lower than normal or expected ↓ --> Low base= acid --> metabolic acidosis.

****

Compensation

لابد من فهمه

If there is Pathology in "Metabolic system" It would be compensated by "Respiratory system" and VICE VERSA

Examples:

A} if there is a pathological metabolic process causing low HCO3

So, it leads to acidosis (Low PH)

↓HCO3 : ↓ PH (زى بعض فى الاتجاه)

The Type of Acidosis here is Metabolic and hence it is the original problem so, it is "primary metabolic acidosis"

- It will be compensated by respiratory system ↓↓↓

Lowers Acidity (↓ H+) or induction of alkalinity by ↓↓↓

CO2 wash or hypocapnia or *Respiratory alkalosis*

PH ↓: HCO3 ↓: PaCO2↓ (الاسهم كلها لتحت)

كله بيقل فى ال (primary metabolic acidosis) لما تكون simple يعنى هيا ال Only abnormality.

- **Conclusion:**

Primary simple *Metabolic acidosis* because it lowers PH

And compensated with *Respiratory alkalosis*

****

B} if metabolic process causes high bicarb (↑ HCO3) --> high PH (alkalinity)

--- > primary metabolic alkalosis

- It will be compensated by ↓↓↓

Respiratory acidosis (adding H+ or acid) … ↑PaCO2

PH↑: HCO3 ↑: PaCO2↑ (الاسهم كلها لفوق)

- كله بيزيد فى ال primary metabolic alkalosis لما تكون simple

- **Conclusion :**

Primary simple *Metabolic alkalosis* as it increases PH value

And compensated by *Respiratory acidosis*

C) If there is a primary pathological respiratory process that causing Excess CO2

↓↓↓

Respiratory acidosis --> High PaCO2 ---> high H ---> low PH

↑PaCO2 --> ↓ PH (عكس بعض فى الاتجاه)

It will compensated by↓↓↓

Metabolic alkalosis by adding base (high HCO3)

PH ↓: PaCO3 ↑: HCO3↑

(اتجاه السهم فى ال PH عكس اتجاه PaCO3 وال HCO3 كمان)

Conclusion :

Primary simpe *Respiratory acidosis* as it decreases PH value and it

Compensated by *Metabolic alkalosis*

****

D} if there is a primary respiratory process that causing low CO2

↓↓↓

Low CO2 = respiratory alkalosis

-->Low H --> alkalinity = high PH

PaCO2 ↓ : PH↑

(الاسهم عكس بعض)

It will be compensated by

Lowering base level= low HCO3

PH ↑: PaCO2 ↓: HCO3↓

Conclusion :

Primary simple *Respiratory alkalosis* as it increases PH value and it is

Compensated by *Metabolic acidosis*

SUMMARY(الخلاصه)

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✌ To read the acid base abnormality

Look at PH

A) If it is **low** **(< 7.35)**

Acidosis

Which is the primary process?

Metabolic (↓ HCO3) Respiratory (↑ PaCO2)

==========

↳ If HCO3 ↓ and ↓ PaCO2

Diagnosis

Primary metabolic acidosis with respiratory compensation

↳ if PaCO2 ↑ and HCO3↑

Diagnosis

Primary respiratory acidosis with metabolic compensation

↳ Finally if PaCO2 ↑ and HCO3↓

Both causes acidity

So *Diagnosis*

Is mixed Respiratory acidosis + metabolic acidosis

****

B) If PH is **high (> 7.45)**

Alkalosis

Which is the primary process?

Metabolic (↑ HCO3) Respiratory (↓PaCO2)

==========

↳ If HCO3 ↑ and PaCO2↑

Diagnosis

Primary metabolic alkalosis with respiratory compensation

↳ if PaCO2 ↓ and HCO3↓

Diagnosis

Primary respiratory alkalosis with metabolic compensation

↳ Finally If PaCO2↓ and HCO3↑ (Both cause alkalinity)

Diagnosis

Mixed respiratory alkalosis and metabolic alkalosis

****

C) If PH ranges from **7.35 - 7.45**

↳ If PaCO2 is in normal range (35-45) and HCO3 is in normal range(22-26)

Diagnosis

Normal ABG

↳ But if PaCO2 and/Or HCO3 are not in normal ranges

↳ if PH < 7.4 ↳ If PH > 7.4

Same pathway of Acidosis Same pathway of alkalosis

Example: (1)

☒ Patient 50 years old came to ER with confusion and rapid kaussmul breathing

His ABG shows↴↴↴

PH: 7.21	PaO2: 81
HCO3: 12	SPO2: 95%
PaCO2: 25	

ال ABG
ده فى ايه ؟
PH: 7.21
يعنى اقل من 7.35
يبقى acidosis

بعد كده عاوزين نعرف مصدر ال Acidosis
هل هو
Metabolic (low HCO3)
ولا
Respiratory (high PaCO2)

هنا
HCO3: 12 (< 22)
Low
يبقى ده
Metabolic acidosis

وال
PaCO2: 25 (< 35) low
Loss of acid = by CO2 wash ---> respiratory alkalosis

يبقى
نحط الاسهم
PH: HCO3: PaCO2
↓ ↓ ↓

الاسهم كلها لتحت (طريقه تانيه اسهل)
ببقى التشخيص

Diagnosis: Primary metabolic acidosis compensated by respiratory alkalosis

Example: (2)

☒ 60 years old man came to ER with confusion and cyanosis

ABG shows ↴↴↴

PH: 7.15	PaO2: 46
HCO3: 28	SPO2: 78%
PaCO2: 80	

Diagnosis: Primary Respiratory acidosis with metabolic compensation

Example: (3)

☒ 47 years old man presents with repeated vomiting

ABG shows↴↴↴

PH: 7.51	PaO2: 83
HCO3: 32	SPO2: 96%
PaCO2: 50	

PH: ↑ > 7.45

HCO3: ↑ > 28

PaCO3: ↑ > 45

الاسهم كلها لفوق تبقى

Diagnosis: Primary metabolic alkalosis with respiratory compensation

Example: (4)

☒ 59 years old woman presents with acute onset of dyspnea & cough , and diagnosed to have COVID-19 infection by CT chest

ABG shows ↴↴↴

PH: 7.49	PaO2: 62
HCO3: 17	SPO2: 84%
PaCO2: 26	

Diagnosis: Respiratory alkalosis with metabolic compensation

Example: (5)

☒ **60** years old diabetic man with history of COPD came with rapid kaussmul breathing and excessive urination & dehydration , confusion

ABG shows↴↴↴

PH: 7. 1	PaO2: 72
HCO3: 9	SPO2: 90%
PaCO2: 57	

PH: low --> acidosis

HCO3: low --> metabolic acidosis

PaCO2: high --> respiratory acidosis

Both cause acidity

This lowers PH

Diagnosis: Mixed respiratory acidosis with metabolic acidosis

All what you need to know bout 👇👇👇

Metabolic acidosis

↓PH (7.4): ↓HCO3 (<22): ↓PACO2 (<35)

الاسهم كلها لتحت (الارقام كلها قليله) فى حالة لما تكون simple.

** Causes:

==========

Acidosis is either High Acid or low base

Hence, it is metabolic, so causes are of metabolic sources:

A) High acids (H+)

I- Endogenous :👇

1) DKA : ketone bodies (acetoacetate)
2) Renal failure (excess phosphoric acids, uric acids)
3) Lactic acidosis : (Excess lactic acids)

[Causes of lactic acidosis: any type of shock, Sepsis, metformin toxicity].

II-Exogenous : Toxicities

1) Salicylate toxicity.
2) Ethanol toxicity.
3) Methanol toxicity.
4) Ethylene Glycol toxicity.

✋✋✋✋✋

Causes of High acids are the same causes of *"HIGH ANION GAP METABOLIC ACIDOSIS"*

B) Low Alkali (HCO3 loss)

Causes

* GIT loss (Diarrhoea)

* Renal Tubular acidosis (Renal loss)

* Adrenal insufficiency (acute or chronic)

Also due to renal loss of HCO3 due to low steroids & aldosterone which control HCO3 reabsorption in renal tubules.

✋✋✋✋✋✋✋

Causes of low HCO3are the same causes of "*NORMAL ANION GAP METABOLIC ACIDOSIS*"

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هتعمل الخطوات الاتيه 👇👇👇👇👇👇👇👇👇

الخطوه الاولى:

Compensation

Compensation: to detect if it is simple (pure metabolic) or mixed.

ملحوظه : لو حسيت موضوع ال compensation صعب الفهم عديه ،، وادخل على الحطوه الى بعدها✌✌✌✌.

**بعد ما تشخص ان فيه metabolic acidosis فى ال ABG تانى خطوه على طول تشوف ال

Compensation

عن طريق معادلة اسمها Winter's Formula

As we have ↓ HCO3 due to "Metabolic acidosis"

So, compensation will be Respiratory

By change in PaCO2

So, will will calculate

Expected PaCO2.

👇👇👇👇

Expected PaCO2 = 1.5× HCO3 + 8 **[±2]**

تحسب المعادله دى ولو طلع رقم ال PaCO2 فى ال ABG اللى قدامك يقع فى نفس ال range بتاع الرقم اللى حسبته من المعادله السابقه

كده نقدر نقول ان دى ↙

Primary simple metabolic acidosis
With appropriate respiratory compensation

✓ لو عايز تعرف دى full ولا partial compensation ↙

بص على الPH

لقيتها من 7.35 -7.40 تبقىfull

اقل من 7.35 تبقى partial

Example 👌
Patient with ABG containing

PH: 7.27	HCO3: 12	PaCO2: 25

الاجابه

اولا ↓ PH تبقىacidosis

ثانيا ال↓HCO3 تبقى

Primary metabolic acidosis

ثالثا ↓ PaCO3 تبقى respiratory alkalosis

✓ عايز اشوف هل ده فعلا compensation ولا لا ؟

هاحسب معادلة الwinter

Expected PaCO2 = 12 × 1.5 +8 [±2]
= 18 + 8 [±2].
= 26 ± 2
= 24 - 28

✓ هنا رقم ال PaCO2 فى المريض 25 يعنى بينحصر فى ال range ما بين 24 و 28
وده معناه ان العيان عنده ↙

Primary simple metabolic acidosis
With appropriate respiratory compensation...

✓ بعد كده ابص على ال PH ↙

هنا اقل من 7.35 تبقى نوع الcompensation
Partial respiratory compensation.

✓ طيب تخيل فى المثال ده رقم ال PaCO2 بتاع المريض ده كان ٣٠ مثلا ،

ده معناه انه اعلى من الExpected range

وده معناه Excess CO2

بالعقل كده مالوش علاقه بالcompensation

وده بيدل على ان المريض عنده درجه من درجات الrespiratory acidosis

نتيجه حاجه فى الrespiratory system

بعيده عن respiratory compensation

اللى لازم يطلع ال Expected PaCO2 فى ال range المتوقعه

زى ما شوفنا فى المثال اللى فات وده نسميه

Mixed metabolic acidosis + respiratory acidosis

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✓ المفروض ان الكل متخيل ان عشان يكون فيه Respiratory acidosis

Instead of respiratory alkalosis

مع metabolic acidosis فى المثال ده ..

الكل متخيل ان لازم يبقى رقم PaCO2 اعلى من 45

احنا بنحكم برقم ال expected مش range ال high بتاع ال. PaCO3

يبقى المريض فى الحاله دى عنده ⬋

Mixed metabolic acidosis + respiratory acidosis

For Example:

Patient known COPD with baseline PaCO2 > normal, develop renal failure

هيبقى ABGفيه ⬋

Mixed metabolic acidosis +respiratory acidosis

✓ وده معناه ان فيه مشكله تانيه فى ال respiratory system

لازم نطلعها ونعالجها او على الاقل ان ماكانتش محتاجه علاج نحاول نقللها عشان ماتزيدش وتحتاج علاج معين

زى ال ventilator مثلا وتسوء الحاله.

✓ طيب لو كانت قيمه ال PaCO2 فى المريض ده ٢٠ مثلا ،،

ده معناه انها اقل من range ال Expected PaCO2

لانها اقل من ٢٤ زى ما حسبنا فى المعادله

وده معناه ان فيه excess CO2 wash

زياده عن اللازم يبقى ده مش compensation

✓ المفروض ان الكل متخيل ان طالما PaCO3 تحت ال ٣٥ يبقى ده alkalosis

يعنى compensation

لا طبعا ،، زى ما قولنا احنا بنحكم برقم ال expected PaCO2 اللى بنحسبه من المعادله

يبقى ده معاه مشكله تانيه فى ال respiratory system

عامله respiratory alkalosis

زياده عن اللى مفروض تعمله

وتبقى هنا

Mixed metabolic acidosis + respiratory alkalosis

✓ وده معناه ان احنا هندور على سبب تانى عامل respiratory alkalosis

فى الحاله غير ال metabolic acidosis ونعالجه كمان

Example:

Patient has DKA + pneumonia (cause of respiratory alkalosis)

❖ وبكده يتضح اهمية المعادله اللى العلماء وضعوها.

الخطوه التانية (مهمه جدا)

Estimation of Anion gap

AG = Na - [Cl+HCO3]

Normal: 10 - 14

If AG is > 14

يبقى الاحتمالات 👇👇

1) DKA: PH < 7.3

- Suspect if 👇
  - dehydration, dry tongue, skin,Polyuria , confusion ,
  - Rapid breathing (kussmaul), abd.pain, vomiting
  - Known Diabetic esp T1DM,young patient
  - Predisposing factor [infection, fever, MI, stroke, missed insulin].
- Next step 👇
  - Random blood glucose ≥ 250 + acetone in urine
  - Other labs & imaging according to case
- Ttt 👇
  1. ABC(airway, breathing, circulation)
  2. ER room : give 1 L saline bolus + 10 units' regular insulin IV
     Then ⬇
  3. ICU or special unit
     - Fluids: 1L Nacl /1 hr, then 1 L/2hr then 1 L/4 hrs.
     - K : check K
       - if < 3.5 give 40 m.mol (4 amp
       - If 3.5 - 5.5 give 20 mmol(2 amp)
       - If > 5.5 don't give K
     - Insulin: give regular insulin by infusion(syringe pump)
       - unit / Kg /hr
       - target BG reduction is 50 - 100 / hr
       - When BG reaches ≤ 250 Give Glucose 5% 1 L /8 hrs
     - Don't give Bicarb unless PH is ≤ 7
     - Ttt of predisposing (infection, MI, stroke
     - Improvement if PH > 7.3, HCO3 >18
     - Normalisation of anion gap, no dehydration--> Change to Subcut. Insulin protocols.

2) Renal failure

- Suspect if 👇
  - known CKD
  - chronic D like DM, HTN, Polycystic K D, CHF
  - Glomerulonephritis ---> Chronic
  - Oliguria, dehydration, shock, sepsis,
  - Obstructive uropathy, drugs -> acute
- Symptoms 👇
  - Pallor, earthy look, volume overload, HTN, Oliguria, Itching, vomiting, hiccup.
- Next step 👇
  - Urea, Creatinine
  - Other labs (K, Ca, P, CBC, Abdominal U/S
  - Estimation of GFR.
- Ttt 👇
  - If PH ≥ 7.25
    - don't give IV Bicarb
    - Consider Oral Bicarb.
  - If PH < 7.25
    - give IV bicarb
    - Use this equation
    - HCO3 deficit = [24- HCO3 value] × 1/2× Body weight.
    - HCO3 deficit / 25 = number of ampoules
    - Half of ampoules --> direct in central line or CVP slowly
    - Other half + 500 glucose 10% over 1 hr
  - Then recheck ABG again if no improvement-> dialysis

3) Lactic acidosis

- Suspect if 👇
  - signs of shock, sepsis, fever
  - Cardiac, hemorrhage, severe dehydration with normal BG
- Next step 👇
  - serum lactate (high)
- Ttt 👇
  - ABC, ICU
  - Ttt of the cause + fluids (cautious in cardiac patients)
  - Bicarb has no role.

4) Toxicities

(Salicylate, ethyl alcohol, methyl, ethylene glycol)

- Suspect if 👇
  - no apparent causes, normal BG, Creat, no shock or signs of sepsis ...
  - History ---> very important esp in young
- Next step 👇
  - Serum toxins , osmolal gap
- Ttt 👇
  - ICU , ABC
  - toxicology specialist

✋✋✋✋✋✋✋✋✋

If Anion gap is normal (10 - 14)

👇👇 يبقى الاحتمالات

1) Diarrhoea (severe acute, chronic)

- Suspect 👇
  - History, dehydration
- Next step 👇
  - Na, K, Urea, creat, CRP, CBC, stool culture, blood culture
- Ttt: if severe 👇
  - ABC + ICU
  - Fluids (ringer's lactate) + CVP value correction (8 - 12)
  - Blind Antibiotics

    (Cefotax 2g/8hrs or Cipro IV /12 hrs + flagyl IV /8 hrs)

    Till result of culture.
  - Antidiarrheal after exclusion of dysentry

2) Adrenal insufficiency

- Suspect if 👇
  - known Addison not compliant with medications or recent infection without change in dose
  - On steroid for any disease , then sudden stop,

- Symptoms 👇
  - Orthostatic hypotension, weight loss, fatigue, lethargy
  - vague abdominal pain ,
  - hyperpigmentatin(dark scars, pigment in mouth & lips)
- Next step 👇
  - Serum cortisol a.m
  - ACTH stimulation test
  - Na ↓ & K ↑ , blood glucose (may be low)
- Ttt 👇
  - If suspicious adrenal crisis
    - ABC, ICU, IV saline + solucortif 100 mg/6hrs
    - Ttt of precipitating factor.
  - If Addison :
    - low dose prednisolone 5 morning , 2.5 evening
    - Astonin : fludricortisone 1×1

3) Renal Tubular Acidosis {RTA}

- Suspect if 👇
  - No apparent cause
  - Mild chronic unexplained metabolic acidosis.
  - Unexplained glycosuria
  - Unexplained nephrocalcinosis
- Predisposing 👇
  - DM, multiple myeloma, Sjogren S,
  - Chemotherapy.
- Next step 👇
  - urine PH
  - urine anion gap (advanced & not available)
- Ttt 👇
  - Give oral bicarb
  - Ttt of the cause

✊✊✊✊✊✊✊✊✊

If ABG don't contain Cl

So, you can't calculate Anion Gap

وطبعا ده مشهور جداااا فى الواقع

غالبا حالات high Anion gap

هيا اكتر الحاجات اللى بنشوفها

وتكون severe

و هيا اللى هتيجى فى بالك الاول

✓ هتعمل الاتى👇👇👇

- Random blood glucose
- Renal functions
- Serum lactate (present in ABG)

✓ ولو طلع واحد من التلاته هتتعامل زى ما شرحنا قبل كده...

✓ لو التحاليل طلعت كويسه هتفكر فى التسمم بالحاجات اللى ذكرناها

وياريت تعمل تحاليلها لو شاكك فيها وعرض سموم

وطبعا هتحجز المريض فى العنايه لو حالته حرجه

✓ اما لو كله negative

ودرجة ال acidosis بسيطه

هتفكر فى الاحتمالات الاخيره

وتعمل التحاليل اللى قولنا عليها...

Delta - Delta gap

Delta -delta gap?
كلمة
Delta = change
او الفرق الرقم
بين ايه ؟
ده معادله من خلالها باحسب حاصل قسمه

$$\frac{\Delta \text{ Anion gap}}{\Delta \text{ Bicarbonate}}$$

Delta = Δ
Δ Anion gap = calculated Anion gap - normal anion gap
Δ Bicarbonate = measured HCO3 - normal HCO3

حاصل قسمة ال
Δ Anion gap ÷ Δ bicarbonate
هيطلع رقم معين
هيفيدنا فى ايه ؟

- لو الرقم طلع قيمته من

1 - 2	Pure high anion gap metabolic acidosis (pure HAGMA)
< 1	Mixed {high anion gap metabolic acidosis HAGMA} + Normal anion gap metabolic acidosis NAGMA}
> 2	Mixed metabolic acidosis & metabolic alkalosis!!

الاخيره دى هل ممكن تدخل الدماغ ؟
ازاى
Metabolic acidosis + metabolic alkalosis
فى نفس الجسم!

دى معناها ببساطه
Hidden underlying pre-existing metabolic alkalosis with recent superimposed metabolic acidosis

بس كانت قوة ال Acidosis
اعلى فخلت الارقام كلها فى صالح ال
Metabolic acidosis

✓ هل ممكن تطبيق ال

Delta Delta gap in practice?

ينفع ،، بس فايدتها ضعيفه شويه ،،

لان الهدف منها تشخيصى وتشخيص كل ما هو مستخبى ومستتر بشكل دقيق جداا ،،

وده عندنا فى مصر مش موجود للاسف

العلاج مش هيتغير طبقا لل

Delta delta gap

ولكن التشخيص و الحاجات المخفيه دى هتفيدنى انى اعالج السبب فقط

ولو انها ممكن تتشخص عن طريق الهستورى او سياق الحاله احيانا ..

والعلاج هو علاج السبب ،، لكن قرار ال

Correction of acidosis

نفسه مش هيتغير كتير طبقا لل

Delta Delta gap

طبعا الامريكان والكتب الامريكيه واسئلة البورد الامريكى بتهتم شويه بالحاجات دى ..

هما متمكنين ومتميزين فى الحاجات الرقميه دى فعلا

✓ اخيرا !! امتى نلجأ لحساب ال

Delta delta gap?

هل فى كل ABG ؟

لا طبعا ،،

فى حالات ال

Primary metabolic acidosis

وكمان عرفنا ال ABG اللى اتشخصت مبدأئيا بال

Compensation
&
Anion gap
العاديين بتوعها

Example:

☒ 25 years old man known to have diabetic , came to ER complaining of vomiting , abdominal pain , excessive urination

RBG: 526

ABG

PH: 7.27	Cl: 90
HCO3: 10	Na: 134
PaCO2: 22	

Answer:

Metabolic acidosis with partial compensation

N.B: American diabetes Association (ADA) diagnostic criteria for DKA

- Hyperglycemia ≥ 250
- PH < 7.3
- HCO3 < 18
- Acetone in urine 2+ or more or B-hydroxybutyrate in blood

****

Example:

☒ 20years old man known to have type 1 DM and having unexplained lethargy & dizziness on standing with craving for salts

He came to ER with dehydration & confusion

ABG shows

PH: 7.21	PaO2: 82
HCO3: 12	SPO2: 96%
PaCO2: 27	Na: 135
	Cl: 105

Answer:

Step 1

- PH: 7.21 ↓ acidosis
- HCO3: 12 ↓ metabolic acidosis
- PaCO2: 27 ↓ respiratory alkalosis

➢ SO, it is *Primary metabolic acidosis with respiratory compensation*

Step 2

- Calculate expected PaCO2
- Expected PaCO2 = HCO3 × 1.5 + 8 (± 2)

= 12 × 1.5 + 8 (± 2)
= 18 + 8 ± 2
= 24 –28

- Measured PaCO2 = 27

➢ SO, it is *Appropriate respiratory compensation*

- Full or partial ؟

PH: 7.21 --> below normal 7.35

➢ SO, it is *Partial respiratory compensation*

Step 3

- Calculate anion gap
- Calculated anion gap = Na - (HCO3 + Cl)

= 135- (12+105)
= 18

- Normal anion gap : 10 – 14
- Median: 12
- Calculated anion gap > 14

➢ SO, it is *High anion gap metabolic acidosis*

طبعا عارفين ال DD بتاعها
بس فى سياق الحاله مشتبه اكتر فى DKA ؟
عشان المريض Diabetic
عشان كده هنقيس Acetone in urine
ولو طلع Possitive
تبقى DKA

في المثال اللي فات

طيب طالما هيا HAGMA

نجرب نحسب ال Delta Delta gap

ونشوف هيا
Pure or not?

Delta Delta gap = Δ Anion gap ÷ Δ HCO3

Δ anion gap = calculated - normal = 18 - 12 = 6

Δ Bicarbonate = normal HCO3 - measured actual HCO3 = 24 - 12 = 12

Delta Delta gap = 12 ÷ 6

= 0.5 (يعنى اقل من ١)

ده معناه

Mixed high anion gap metabolic acidosis HAGMA
+
Normal anion gap metabolic acidosis AAGMA
يعنى فيه حاجه تانيه عند المريض ده غير الDKA

DD of normal anion gap met acidosis?

1. Adrenal insufficiency
2. Diarrhoea
3. Renal tubular acidosis

Adrenal insufficiency

اللى هو
Addison
وجاى مع
Type 1 DM

واعراضه هيا

Lethargy
Craving for salts
Dizziness on standing

وبالتالى بعد ما اعالج ال DKA
هاعمل Cortisol a.m
عشان اثبت تشخيص ال
Addison's disease

Example:

☒ 32 years old man came to ER with disturbed conscious level and fits

No lateralization at all

He was previously healthy without any medical problem ،

He lives alone and he came to hospital by the help of his neighbour

MRI brain: normal

CSF exam: normal

Basic labs show

Normal CBC, LFTs, RFTs

Random BG: 126

Coagulation profile

ABG & electrolytes

PH: 7.23
HCO3: 15
PaCO2: 26
PaO2: 78%
SPO2: 94%
Na: 135
Cl: 98
Urea: 42
Measured plasma osmolality: 301

Diagnosis?!

Answer:

✓ طبعا فى الحاله دى ،،واضح جداا انها

Primary metabolic acidosis

اظن كلنا عارفينها

لو حسبنا

Anion gap = 135 - (15 + 98) =22

يعنى دى👇

High anion gap metabolic acidosis

✓ طب ده احتمالاته ايه

DKA

مش ماشيه ... السكر كويس

Renal failure

مش ماشى ... الكريات كويس

Lactic acidosis & sepsis

احتمال ... بس الهستورى مش راكب على الاحتمال ده

Toxicities

دى محتمله جداا لان سياق الهستورى غامض وده راجل ساكن لواحده ،،

محتمل يكون تناول سم فعلا

** Toxicities

Aspirin (Salicylate)

Ethanol

Methanol

Ethylene glycol

✓ كل الToxicities

دى ،، ممكن نعرفهم بحسبه مبدأيه اسمها

Osmolality gap

ايه دى ؟

كنا قولنا واحنا بنشرح ال Plasma osmolality
ان احنا ممكن نقيسها وممكن نحسبها
وان الرقم متقاربين جداا ،،
بس الفرق بينهم لابد يكون اقل من ١٠ ده فى الانسان العادى

لكن لو الفرق اكتر من ١٠
يبقى
Osmolality gap: high

وده معناه ان فيه 👇
Hidden exogenous osmole
جاى من خارج الجسم ومش موجود فى المعادله المشهوره
وده اللى مزود ال measured osmolality
وده بيبقى. Toxins

➢ اما بالنسبه للناس اللى بتتلخبط فى موضوع الارقام والمعادلات او مش واخده عليها

قولنا قبل اهم شئ هو قراءة ال ABG

بطريقه Simple

دى مش محتاجه معادلات ،،

موضوع ال Compensation

ده غالبا بتجيب اغلبه من قرايتك لل ABG ببساطه ،، لو عايز تبقى دقيق احسب بالمعادله

مش حافظ المعادله سجلها عندك ع الموبايل او احسبها عن طريق ال MD calc

اما اللى لازم تحسبه اجبارى هو ال Anion gap

لو حتى من ال MD calc

لان ده بيتوقف عليه تشخيص

لو انت شاطر اوى فى الباطنه ممكن تشخص السبب بالهستورى والتحاليل التانيه

All what you need to know bout 👇👇👇

Respiratory Acidosis

PH ↓: PaCO3 ↑: HCO3↑

- زياده ال PaCO3 بتزود ال Bicarb
  وبتقلل ال PH فى حالة لما تكون simple
- كل اسباب respiratory acidosis نتيجة مشاكل فى اى جزء فى ال respiratory system ككل
  من اول respiratory center مرورا بعضلات التنفس سواء كانت المشكله فيها او فى الاعصاب المغذيه ليها واخيرا فى الرئتين نفسهم .
- كل اسباب ال respiratory acidosis
  بتكون مصاحبه ب hypoxia بسبب
  Impaired ventilation --→ hypoxic hypercapnic
  Type 2 respiratory impairement وده بنسميه

➢ There are 2 Types Respiratory Acidosis

I- Chronic respiratory acidosis: due to chronic CO2 retension

- Due to either

A) chronic lung disease : COPD

B) chronic neuromuscular

- diseases like Motor neurone
- disease & severe
- Kyphoscoliosis (rare)

II- Acute respiratory acidosis

- Either due to

A) Acute on top of chronic :

- Acute exacerbation of COPD (Very Very Common.)

B) Acute on top of normal lungs :

- Respiratory center depression by drugs high dose: like Morphine& opiates, hypnotics.
- Acute neuromuscular disorder like Myasthenic crisis, Guillian Barre Syndrome

➔ Any disease leading to acute type 1 respiratory impairement (hypoxia with hypocapnia)

--> If not treated rapidly, tachypnea will lead to respiratory muscle fatigue due to hypoxia

--> Accumulation of lactic acid

--> Bradypnea--> RR starts to become slow with CO2 retention (hypercapnia)

--> Type 2 respiratory failure (hypoxic hypercapnic)

Ex:

- pulmonary edema (cardiogenic & non cardiogenic: ARDS {alveolar diseases}
- severe COVID-19
- acute massive pulmonary embolism (perfusion defect)

➢ **How to differentiate between**

Acute respiratory acidosis and chronic respiratory acidosis?!

➔ Look at PH

A) If it is between **7.33 - 7.39**

It is likely to be chronic (not sure 100%)

➔ Then the next step 👇

compensation احسب ال (مهم جداااا جداااا)

- In **Chronic** respiratory acidosis
  - Every 10 mmHg increase in PaCO2 >40
    Lead to → 4 mmHg increase in HCO3 > 24

Or

- Every 1 mmHg increase in PaCO2 >40
  Lead to → 0.4 increase in HCO3 > 24

Example:

Patient has COPD on treatment
Has ABG showing:
PH: 7.34, PaCO2: 60, HCO3: 32

Is it acute or chronic?

Answer:
PH: 7.34 --> more likely chronic

**** if Not sure?**

Let's check compensation 👇

- Change in PaCO2 = 60 - 40

= 20

الرقم اللى حسبناه ده بنسميه معدل زيادة ال PaCO2

هنضرب الرقم ده فى 0.4 ونضيفه على قيمة متوسط ال bicarb ونشوف هيطلع كام

- Expected HCO3 in chronic = 20×0.4+ 24

= 8+ 24 = 32

هنا طلع نفس الرقم

والتشخيص هنا👇

Chronic respiratory acidosis with appropriate respiratory compensation

- **Next step** in therapy

- No Ventilation either Non-invasive (NIV) or invasive ventilation (mechanical)

- No admission at ICU

- Just control disease by ttt at home.

B) If PH < 7.33

It is likely to be acute

- Is it acute on top of normal?

Or

- Acute on top of chronic!؟

You will be guided by history & examination.

- In acute on top of normal

PaCO2 is not too much (around 60)

- In acute on top of chronic

PaCO2 is high (> 80, may reach 90, 100 or more)

Because, the baseline is high unlike

Acute on top of normal (normal baseline PaCO2)

****if Not sure Acute or chronic?**

compensation احسب ال

In acute respiratory acidosis

- Every 10 mmHg increase in PaCO2 >40

Lead to →1 mmHg increase in HCO3 > 24

Or

- Every 1 mmHg increase in PaCO3 >40

Lead to → 0.1 mmHg increase in HCO3 > 24

Example

Patient with ABG showing

PH: 7.18, PaCO2: 60, HCO3: 26

- أول حاجه نشوف ال PaCO2 زاد بمعدل كام وبعدين نضرب الرقم فى 0.1 ونجمعه على 24عشان نحسب ال

Expected HCO3

Expected HCO3 = [60 - 40] ×0.1 +24

= 20 × 0.1+ 24

= 26

- هنا ال expected HCO3 طلع نفس رقم ال bicarb عند المريض لو حسبنا على ال acute

وده معناه ان ده 👇

Acute respiratory acidosis on top of normal for sure.

والاحتمالات هنا زى ما شرحت فوق

- لكن لو بصينا فى مثال تانى

Example

Patient known COPD, admitted with confusion, cough

ABG show PH: 7.12, PaCO2: 90 ،HCO3: 33

Answer:

PH < 7.33 → acute for sure

ثانيا ال PaCO2 عالى اكتر من 80 فى مريض معروف ان هو COPD

تبقى دى👇

Acute on top of chronic Respiratory acidosis (acute exacerbation of COPD)

****if Not sure?**

Calculate expected HCO3

If Current HCO3 > Expected PaCO2

It is acute on top of chronic for sure.

Expected HCO3 = [90 - 40] × 0.1 + 24
= 50 × 0.1 + 24
= 29

Here, Current Bicarb in this patient > expected

And hence, PH is < 7.33

Final diagnosis

Acute On top of chronic Respiratory acidosis (exacerbation of COPD)

**** Treatment:**

- ABC, ICU admission
- If PH: 7.25 - 7.35 (high dependency unit)
- Give him low flow O2 with target O2 saturation: 88 - 92% by monitor
-Nebulizers / 15 minutes (SABA or SAMA)
- IV hydrocortisone 100 mg/8rs
- IV antibiotics (infection)

Levofloxacin 750 / 24 hrs IV + fluids

- Monitoring of ABG every 1 - 2 hrs

↳ If no response and PH still 7.25 - 7.35

Consider Non invasive ventilation (NIV)

BIBAP better than CPAP

The patient must be conscious

If worsening CO2 retension & more ↓PH

Invasive mechanical ventilation

N.B if PH < 7.25 + confusion

Consider start of mechanical invasive ventilation from the start.

** **Treatment of the causes of acute respiratory acidosis on top of normal**.

* Respiratory center depression by

-Morphine >> ICU
-Antidote >>> Naloxone
- If no response: mech .ventilation.

* Myasthenic crisis

ICU, Pulse steroids, + IVIG + plasmapheresis
If no response --> mech Vent.

* Guillian Barre Syndrome (GBS(

ICU, Either IVIG or plasma exchange
If no response --> mech .Vent.

* Transverse Ascending myelitis

ICU, pulse steroids ,
If no response --> Mech. Ventilation

****

Example

☒ 62 years old man came to you in the casualty in confusion with intermittent cyanosis
His son reports that, he has developed acute productive cough with fever over the previous 48 hours with worsening dyspnea
There was past history of chronic chest problem due to smoking for which he takes bronchodilators
Chest: bilateral scattered insp. crepitations on the back
With expiratory rhonchi
Raised JVP
Bilateral pitting LL edema
ABG

PH: 7.18	PaO2: 53
PaCO2: 95	SPO2: 74 %
HCO3: 30	

Answer:

Straightforward case of
Acute respiratory acidosis on top of chronic acidosis due to COPD mostly, complicated with pulmonary HTN {RSHF}

LSHF might be an underlying cause of cardiac symptoms, but, if there are strong certain clues like S3 gallop, cardiomegaly with displaced apex, history of PND, this would be assessed certainly by bedside Transthoracic ECHO

طبعا سبب ال

Acute exacerbation of COPD here is **pneumonia**

{Bronchopneumonia} evidenced by acute onset of fever, productive cough, with scattered inspiratory crackles on the chest which are superimposed on the exppiratory wheezes

****

Example

☒ 70 years old patient came with acute progressive dyspnea with orthopnea
Clinically
Raised JVP
Pansystolic murmer on apex
Bilateral diffuse crackles on the back
ABG

PH: 7.28	PaO2: 54
PaCO2: 55	SPO2: 90%
HCO3: 25	

Answer:

* Primary disorder :
Respiratory acidosis .

* Cause:
Acute pulmonary edema

* Expected compensation:
= (55 - 40) x 0.4 = 15 x 0.1 = 1.5
= 24 + 1.5 = 25.5
Acute compensated respiratory acidosis
Type 2 respiratory impairment

بالنسبه للحاله دى

Cardiogenic pulmonary edema or non – cardiogenic?

و هل ال

Focal signs on the patients can explain that is it acute heart failure or

Acute pulmonary edema on top of CHF?

↳ This case of acute dyspnea with type 2 hypoxic hypercapnic respiratory acidosis .

Clinically, the patient has signs from which we can suspect the cause :

#Bilateral diffuse crackles suggests severe pulmonary edema mostly with exhausted patient with respiratory muscle fatigue that cause him to be in respiratory acidosis,

#Pansystolic murmer over apex which may suggest Mitral regurge or VSD (we should go to axilla and listen, if it is heared then its MR) (also we must ask about history and previous Echo to know that this MR is acute or chronic, if acute we must suspect Infective endocarditis or Inferior MI if patient with chest pain or without chest pain in diabetics and elderly so ECG is important also) (if murmer is of VSD and new and not in the previous Echo , then suspect Anterior MI.

So clinically it all points to cardiogenic pulmonary edema .

****

Example

☒ 56 years old man was admitted with fever associated with confusion

2 days previously, he has had frequent micturiton and dysuria

Past history was remarkable for COPD and Type 2 DM

He is on insulin mixtard and occasional inhalers

His ABG shows

PH: 7.05	SPO2: 89%
HCO3: 15	Na: 141
PaCO2: 60	K: 5.2
PaO2: 64	Cl: 97

How would you interpret such ABG?

Answer:

High gap metabolic acidosis mixed with Respiratory acidosis and metabolic alkalosis

PH ⬇
PaCo2 ⬆
HCO3 ⬇

* AG

=Na - (Cl + HCO3)

141- (97+15) = 29

High anion gap metabolic acidosis

* Delta ratio

(29 - 10) / (24 - 15)
= 19 / 9
= > 1
* Sepsis highly suspicious

Q urosepsis to be confirmed clinically by

Rapid qSOFA or SOFA

Also by urine analysis, culture + blood culture, CRP & procalcitonin

You have to exclude DKA, as the patient has DM, check ketone bodies in the urine

Respiratory acidosis might be chronic because PaCO2 is not high enough to suspect acute on top of chronic

Also SPO2 value lies at this range (88 - 92%)

Also, for more chest evaluation, auscultation, imaging

Respiratory Alkalosis

PH ↑: PaCO2 ↓: HCO3 ↓

- If it is simple pure Resp .alkalosis, metabolic compensation would occur by reduction in HCO2 value (metabolic acidosis)

N.B:

Respiratory Alkalosis = Hyperventilation

Causes:

Either

A} Hyperventilation due to Stimulation Of respiratory centers in the brain Stem

i) Central causes:

- Encephalitis
- Extensive Brain hemorrhage
- Infarction
- Extensive brain trauma

ii) Metabolic causes:

- Hepatic
- Encephalopathy
- Fever: important

iii) Psychogenic hyperventilation

- Panic attacks,
- hyperventilation syn

All these causes hyperventilation and CO2 wash without Hypoxia or Hypoxemia

(O2 sat ≥ 95% & PaO2 ≥80).

B} Hyperventilation due to Hypoxia

Hypoxic Hypocapnic (Type 1 respiratory impairement)

I.e (Acute lung condition)

Causes:

- Pneumonia
- Acute severe asthma,
- tension pneumothorax
- Pulmonary edema.
- Acute severe asthma,
- tension pneumothorax
- Pulmonary Embolism (very very Important (easily missed in practice)

الخطوه الاولى بعد ما تشخص انها primary Respiratory alkalosis ←
انك تشوفها

Pure simple resp alkalosis or mixed

عن طريق حساب ال

Compensation

ملحوظه: المعادلات هنا مش مهمه

- In acute resp .alkalosis

→ Every 10 mmHg decrease in PaCO2

→ 2 mmHg decrease in HCO3 < 24

- In chronic resp .alkalosis

→ Every 10 mmHg decrease in PaCO3

→ 4 mmHg decrease in HCO3 < 24

Treatment:

A} Respiratory alkalosis without hypoxia

- You have to detect the cause by taking through history and thorough examination:

- Treatment of the cause

B} Respiratory Alkalosis with hypoxia

I. suspected pneumonia by history& Examination

- Treatment by High flow O2 with target O2 sat ≥ 95%... If refractory hypoxia & RR > 30

- Consider mechanical ventilation

- Proper Antibiotics accordingly

ii. Pulmonary edema

- High flow O2 as before

- If cardiogenic --> IV diuretics ± nitrates if high BP ± Inotropes if low BP

- If hypoxia is refractory: CPAP

- If ARDS: Mechanical Ventilation

- If refractory: ECMO

iii. Acute severe asthma

- High flow O2 + nebulizers

- IV solucortif 100 mg / 6 hrs

-If refractory: IV magnesium

If refractory: Mechanical ventilation

iv. Tension Pneumothorax

- High O2 + urgent Intercostal tube insertion under water seal

v. Pulmonary Embolism

- Suspect if hypoxic hypocapnic pattern with normal chest auscultation

- Particularly if there are risk factors

- DVT, bed ridden,
- recent surgery, cancer
- OCP,
- thrombophilia (suspect if it is unprovoked DVT/Pulmonary embolism)

- Calculate Well's score

  - If ≥ 4 ---> begin LMWH (therapeutic)
  - high flow O2
  - And arrange

    D.dimer + CT pulmonary Angiography to confirm the diagnosis

Example

52 years old woman came to the casualty with sudden onset of chest pain and dyspnea

The chest pain was vague and was not compressing in nature and was not referred to any site...

Bed side SPO2: 91%

Auscultation of the chest: free

Heart: free

JVP: not high

ECG: sinus tachycardia

CXR: normal

ABG

PH: 7.51	PaO2: 62
PaCO2: 24	SPO2: 91%
HCO3: 16	

Answer:

Respiratory alkalosis with partial metabolic compensation

Type1 respiratory failure

Ddx:

- Pneumonia
- Acute sever athma
- Tension pneumothorax
- Pulmonary edema
- Pulmonary embolism

Most likely pulmonary embolism due to acute onset, free chest, sinus tachy
So ask about risk factor:
DVT, obese, recent surgery, tumor, OCP, vircaw triad , bed ridden ,thrombophilia
Then do:
- D dimer, ECG, CT angio

Treatment:

- High oxygen
- Anti coagulant
- Surgery:
- Filter
- Catheter

هل اى مريض تشك ان عنده
Pulmonary embolism
??????????
الصح انك تحسبله
Well's score
لانه بيتوقف عليه ال
Degree of propability
وحسب الرقم بنشوف هل هنعمل
CTPA
على طول لانه غالبا هيطلع
Pulmonary embolism

Well's score

Well is a Canadian scientist who put the famous international prestest probability for pulmonary embolism as follow ↴

- Clinical symptoms of DVT --> score **3**
- other diagnosis less likely than pulmonary embolism --> score **3**
- HR > 100 --> **1.5**
- immobilization more than 3 days or surgery within the previous 4 weeks --> **1.5**
- previous DVT /pulmonary embolism --> **1.5**
- Hemoptysis --> **1**
- Malignancy --> **1**

عشان ما ننساهمش

الاول

Origin: DVT symp/signs

الثانى

History pattern/certain clues of PE

دى معناها تركيبه الحاله لا تفسر الا بال pulmonary embolism ،، مثلا

Unexplained acute dyspnea with normal lung auscultation + normal imaging + hypoxia

الثالث

Sign: tachycardia

وعشان هيا غالبا بتبقى موجوده فى PE سكورها 1.5

الرابع والخامس

2 important risk factors

- Immobilization & previous recent surgery 1.5
- Previous DVT /PE 1.5

السادس:

اقل اهميه :Sign

Hemoptysis: 1

السابع:

Risk factor

ولكن اقل اهميه من السابقين

Malignancy --> 1

⬅ لو جمعتهم وطلعوا

Total score > 4

غالبا هيطلع

Pulmonary embolism

بنسبه كبيره ولكن مش **100**%

عشان كده الخطوه اللى بعديها ع طول هنعمل اشعه مقطعيه على الشريان الرئوى

لو ظهر thrombus فى ال PA او احد افرعه يبقى PE

ما ظهرش يبقى مشPE

🡸 طب لو الاسكور اقل من او يساوى 4
يبقى احتماليه انه يطلع Pulmonary embolism
قليله ولكن وارده برضه ،، والخطوه اللى بعدها هو عمل
D.dimer
If positive > 500
هنعمل
CT pulmonary angio
ولو نيجاتيف تبقى دى مش
Pulmonary embolism
Consider another diagnosis

==========

🡸 للتجميع بطريقه سهله

- ✓ Origin of thromboembolism (DVT) ---> 3
- ✓ History pattern consistent with pulmonary embolism

يعنى تركيبة الحاله ماشيه مع pulmonary embolism اكتر من اى حاجه تانيه وده هياخد Score 3

✓ بعد كده

One symptom

Hemoptysis --> 1

لانها مش specific للPE

✓ بعد كده

One sign

Tachycardia, HR > 100

تاخد 1.5لانها common فى حالات الPE

✓ بعد كده ٣ عوامل خطوره

2 very important risk factors

Immobilization for ≥ 3 days or history of surgery within the previous 4 weeks

Previous DVT /PE

ودول كل واحد فيهم بياخد 1.5

✓ فاضل عامل خطوره واحد ولكنه اقل خطوره من السابقين

Malignancy --> 1

حضرتك بتشوف ايه اللى موجود من ال items دى ف الحاله وتحطله الاسكور بتاعه وبعدين تجمع ارقام كل ال items الموجوده فقط وتشوفها اكتر ٤ ولا ٤ فما اقل وتمشى فى نفس ال algorithm اللى قولنا عليه

Example:

☒ 22 years old girl came to the casualty with acute onset of dyspnea with rapid breathing with sense of impending death that is associated with parathesias in the hands and legs which started to develop carpal spasm bilaterally

She has just got separated from her fiancée

On exam

BP: 100/60
Pulse: 140
RR: 25
ECG: sinus tachycaria
CXR: free

Temp: 37.2
SPO2: 99% on room air

ABG

PH: 7.51	PaO2: 83
PaCO2:23	SPO2: 99%
HCO3: 16	

How would you interpret the ABG in the context of history?!

Answer:

* Respiratory alkalosis é partial metabolic compensation
Hypocapnia éout hypoxia ▸ hyperventilation

* Ddx:

RC stimulation

- Central "hemorrhage, infarction, infection, truma"
- Metabolic"heptic encephalopathy, fever"
- Psychogenic

Peripheral parasthesia is an important sign of psychogenic hyperventilation associated with "PANIC ATTACKS"

The respiratory alkalosis per se with excessive CO2 wash will lead to shift of ionized CA leading to carpal spasm

* Treatment: Sedative is enough

ABG will be corrected after the Panic attack has been treated

Metabolic alkalosis

PH ↑: HCO3 ↑: PaCO2 ↑

If it is simple, evey thing is high

الاسهم كلها لفوق

Primary metabolic alkalosis with respiratory compensation!!

★Causes of metabolic alkalosis

Either Excess ↑alkali/base or ↓ low acid

A} Excess Alkali in blood:

*Exogenous:

- Excess bicarb infusion.

*Endogenous:

- Excess renal reabsorption
- high steroid (Cushing Syndrome)
- High aldosterone (Conn's syndrome)
- Channelopathies
  E.g Bartter's syndrome, Gitleman's syndrome, Lidddle's syndrome
- Shift of bicarb to blood in cases of hypokalemia {Diuretic use}.

B} low acid

- Loss of HCL from stomach
- Vomiting, Nasogastric tube
- Gastric fistula (important).

لما نشخص

Metabolic alkalosis

pure metabolic alkalosisالمفروض نعرف هيا

mixed disorderولا

عن طريق معادلة ال compensation برضه

وهيا هنا مش مهمه زى قبل كده للى عايز يعرفها

Expected PaCO2 = [0.6 - 0.75 × HCO3] + 40

Treatment:

*If severe --> PH > 7.6 (rare in practice)

➔ Give KCL in cases like severe vomiting.

* Vomiting & GIT losses (saline sensitive):
  - give anti-emetic IV (Primperan, Zofran)
  - Saline infusion

* In diuretic (stop diuretic, give K)

* In saline resistant causes (Cushing, Conn's syndrome)
  - Ttt of the cause

N.B:

Saline Sensitive metabolic alkalosis: e.g Vomiting
Saline resistant metabolic alkalosis e.g Endocrinopathies like Cushing Syndrome

N.B: in Endocrinopathy, metabolic alkalosis is just a diagnostic tool
It is useful in diagnosis not treatment, and it is not specific.

نقص الكالسيوم ده سببه ال Alkalosis
وعلاج السبب هيصلح ال Alkalosis
وبالتالى هيرجع مستوى الكالسيوم المتأين لمعدله الطبيعى وكل الاعراض هتختفى

Example:

☒ 28 years old obese woman came to as she is recently discovered to have recurrent muscle cramps and weakness that was found to be due to mild hypokalemia (K : 2.9 - 3.2)

She complains of fatigue, and she is being treated from anxiety and depression

She denied diarrhoea
The workup for hypokalemia including (serum Mg, 24 hrs K in urine < 20, all labs failed to reveal the cause.

ABG

PH: 7.47	PaO2: 86
HCO3: 29	SPO2: 99%
PaCO2: 49	

How would you interpret such ABG in the context of the history?
What might be the cause?!

Clue for DX:

Metabolic alkalosis with hypokalemia from non-renal source as (24 hrs urine K < 20)

So, it is for sure GIT loss

The most common cause of GIT loss leading to metabolic alkalosis is "repeated vomiting"

So, we have to ask specifically about it, and whether it is spontaneous or self induced

Given the history of obesity and associated Anxiety & depression

It is highly suspected to have Bulimia nervosa

Bulimia Nervosa is a psychiatric disorder Ch ch by binge eating followed by self induced vomiting leading to

Both metabolic alkalosis and hypokalemia

المريضه اللى من النوع ده غالبا هتخبى عليك الموضوع ده ومش هتقولك انها بتاكل بشراهه وبعدين بتحط صباعها فى بقها عشان ترجع (ده غصب عنها) لان ده مرض نفسى ،، وهتخبى عليك موضوع مشكلة الاكل والقيء المتعمد

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سؤال مهم : ايه علاقة البوتاسيوم بال acid base disorders ؟

Acidemia (reduced blood pH) is associated with increased plasma potassium concentration (hyperkalemia)

,whilst alkalemia (increased blood pH) is associated with reduced plasma potassium concentration (hypokalemia)

Mechanism

Acidosis causes potassium to move from cells to extracellular fluid (plasma) in exchange for hydrogen ions ,

And alkalosis causes the reverse movement of potassium and hydrogen ions.