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RESEARCH ARTICLE

PULMONARYEMBOLISMREVEALED BY ABDOMINAL PAIN: A CASE REPORT

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Abstract

Introduction: Pulmonaryembolism (PE), one of the leading causes of morbidity in the world, isassociatedwithsignificantmortality, The diversity and lowspecificity of clinical manifestations of PE often lead to misdiagnosis . Correct earlydiagnosis and prompt treatment are therefore key factors in reducing the mortality rate of PE.We report the case of a 58-year-old man treated in the out-patientdepartment of the Mohammed VI ArraziUniversity Hospital in Marrakech (Morocco) for pulmonaryembolism as the reason for revelation of abdominal pain

Observation:A 58-year-old man with a history of hypertension, a twelve pack/year smokerwhopresentedwith abdominal pain in the right hypochondrium of suddenonset for twodays, radiating to the right flank, and evolvingperiodically, aggravated by inspiration and feeding. On abdominal ultrasound no gallstones, no distension of the gallbladder and a negativeMurphy'ssign. Abdominal CT scan showed a small right pleural effusion withatelectasis at the right base on slices through the chest, with no evidence of acute intra-abdominal process.Thoracicangioscanrevealed acute pulmonaryembolism in the segmental branch of the right lower lobe extendingdistallyinto the subsegmental branches; the infiltrate in the base of the right lung base mostlikelyrepresents an infarctedlung. It wasdecided to performthrombolysisduringresuscitation, sohewasthrombolyzedwithtwo doses of retaplasehalf an hourapartwithtolerance of the treatmentwithoutsigns of bleeding. Shortlyafterthrombolysiswithretaplase, hiscyanosis and PJV improveddramatically .

Discussion: Abdominal presentation of PE isdescribedwith high frequencyit, can manifest as an acute abdominal picturewith right upper abdominal pain with or withoutdefensiveness. The abdominal pain isthought to resultfrom irritation of the pleura on contact with the diaphragm or fromliver congestion secondary to IVD.

Conclusion:PE can take on the appearance of variousotherentities and physiciansshouldbeaware of the differentsigns, abdominal pain shouldalert the clinician to the possibility of an extra-abdominal aetiology, including PE, especially in the presence of dyspnoea or polypnoeaincludingsymptoms and radiographicfindingsthatmay lead to this life-savingdiagnosis.

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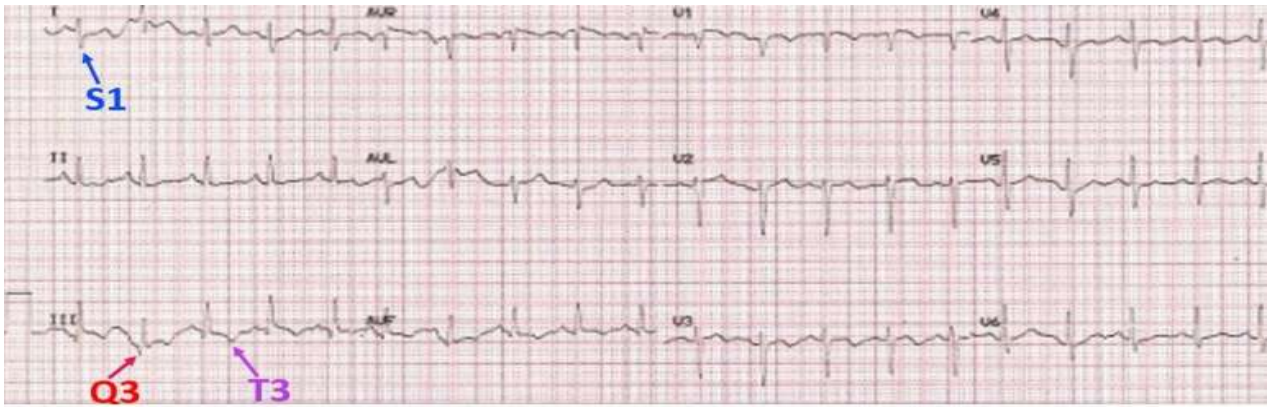
Introduction:-

Pulmonaryembolism (PE), one of the leading causes of morbidityworldwide, isassociatedwithsignificant mortality. The diversity and lowspecificity of the clinical manifestations of PE often lead to misdiagnosis or misseddiagnosis.Itisreportedthat the misseddiagnosis rate is over 70% and only 7% of patients who die of PE are diagnosedbeforedeath [1].The mortality rate of patients with PE whosediagnosisismissedisgenerallyconsidered to be 30% [1], indicating a catastrophicharmfrommisdiagnosis or misseddiagnosis of PE.Symptoms of PE are oftendescribed as dyspnoea, chest pain, cough, haemoptysiswithoutmentioning abdominal pain [2-4].Similarly, PE isveryrarelymentioned in the differentialdiagnosis of acute abdomen.We report the case of a 58-year-old man treated in the out-patientdepartment of the Mohammed VI ArraziUniversity Hospital in Marrakech (Morocco) for pulmonaryembolism as the reason for disclosure of abdominal pain

Observation:-

A 58-year-old man with a history of hypertension, a twelve pack/years smokerwhopresentedwith abdominal pain in the right hypochondrium of suddenonset for twodays, radiating to the right flank and evolvingperiodically, aggravated by inspiration and feeding. The patient had no chest pain, dyspnoea, chills, no history of thesesymptoms, no notion of recenttravel, no history of clotsurgery. There was no othersignificantmedicalhistory, except for an acute pancreatitis at the age of 20 yearswith no known cause, no medicationwasreported. On admission, the patient wasconsciouswith a Glascow score of 15/15, temperature 38.4°C, blood pressure 137/83 mmHg, heart rate 100 beats per minute, respiratory rate 22 cycles per minute and O2 saturation 96% on room air. Physical examinationrevealed a patient in good general condition, cardiac and pulmonary auscultation was normal,negativehomans sign,. The abdomen was tender in the right upper quadrant and not distended. Murphy'ssignwasnegative. No biologicalinflammatory syndrome, no renalfailure, no ionicdisturbancesLiver enzymes within normal limits, bilirubin 1.6, troponinsnegativetwice.

Arterialbloodgaseswereperformed and revealedsevere mixed acidosiswith pH 6.77, PaCO2 77 mmHg, PaO2 64.7 mmHg, his bicarbonate 7 mmol/L, and his base excess -23.8 mmol/L. Abdominal ultrasoundshowed no gallstones, no gallbladder distension and a negativeMurphy'ssign, abdominal CT scan showed a small right pleural effusion withatelectasis at the right base on slices passing through the thorax, with no evidence of acute intra-abdominal processes, The course wasmarked by increasing abdominal pain becomingexcruciating and haemadynamicinstabilitywith BP 7/4 mm Hg, HR 158 beats per minute, polypnoea 36 cycles per minute and pulse saturation 68% on room air, cold sweats and perioralcyanosis, A bedsideechocardiogramrevealed a dilated right ventricle, a paradoxical septum and an ejection fraction of 60%, the diagnosis of pulmonaryembolismwassuspected and confirmedafter a thoracicangioscan (acute pulmonaryembolism in the segmental branch of the right lower lobe). Admitted to the emergency room, hepresentedwithbradypnoea and thencardiopulmonaryarrest in asystole, followed by cardiopulmonaryresuscitationaccording to the protocol, and thenthrombolysisafterrecovery of spontaneouscardiacactivity, and wasthereforethrombolyzedwithtwo doses of retaplasehalf an hourapartwithtolerance of the treatmentwithoutanysign of bleeding. Shortlyafterthrombolysiswithretaplase, hiscyanosis and PJV improveddramatically. His vital signsbegan to improvedramatically and his condition continued to improvethereafter. A repeatechocardiogramafterthrombolysisshowed a slightimprovement in right ventricular pressure. Subsequently, hewastransferred to the intensive care unit for close monitoring and additional anticoagulation accordingto protocoland stayedthere for five dayswith good improvement and wasthendeemed stable for transfer to a pulmonary unit.



The ECG performed in sinus rhythmshowed a S1Q3T3 appearance (Figure 2)

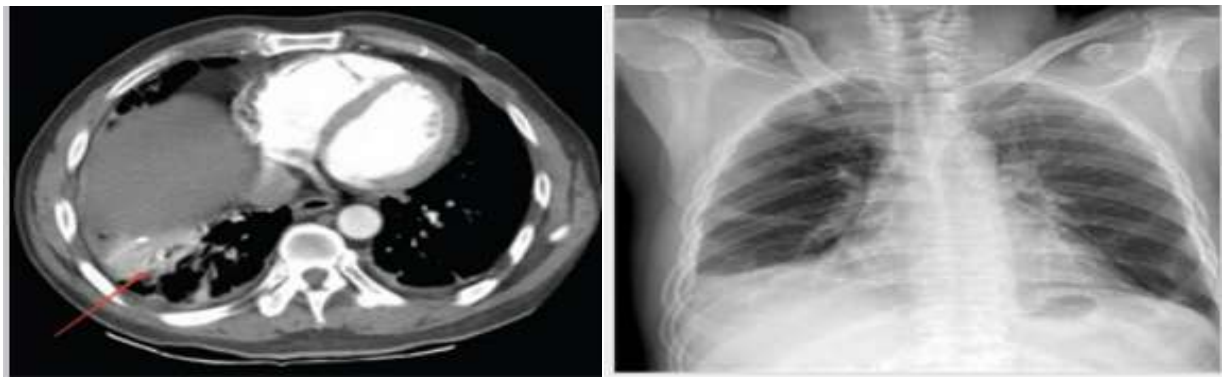


Figure 1:-

Thoracicangioscanrevealed acute pulmonaryembolism in the segmental branch of the right lower lobe extendingdistallyinto the subsegmental branches; the infiltrate in the base of the right lung base mostlikelyrepresents an infarctedlung.

Discussion:-

PE shouldbeconsidered in the differentialdiagnosis of abdominal pain especially in thosewithriskfactorssuch as malignancy, nephrotic syndrome, immobility, diabetes, connective tissue disease, deepvenousthrombosis of the lowerlimbs, pregnancy, oral hormones and contraceptive pills. Earlydetection of bloodoxygen saturation and D-dimer shouldbeperformed, and PE shouldbeconsidered in highlysuspected patients.

The abdominal presentation of PE wasdescribed as early as 1957[1], with a frequency of 6.7%. According to theseauthors, PE can present as an acute abdominal presentationwith right upper abdominal pain with or withoutguarding. The abdominal pain isthought to resultfrom irritation of the pleura on contact with the diaphragm or fromliver congestion secondary to IVD. This article reportedonly cases of CDH or epigastric pain. Subsequently, cases of lefthypochondrial or leftflank pain werepublished [5,6]. Most often, the digestive signs (DA in the foreground) are accompanied by cardiorespiratory manifestations withoutthisbeingsystematic. In several patients, the vital parameterswere normal [6,7], which can lead to misdiagnosis. Sometimesonly the respiratory rate isaccelerated [5,6]. Pain often has a pleuritic component [6-8]. Some patients are febrile [7], which, in the presence of digestive signs, maywrongly point to an intra-abdominal infection.

Varioushypotheses have been proposed to explain the abdominal pain. In addition to liver congestion (for right-sided abdominal pain) and signs of pleural irritation [1], authors have suggested a muscularetiology (intercostal muscle pain) and irritation of the sensory nerve endings of the parietal pleura [8]. Lessobviousmechanisms have been suggested. Hyperaesthesia of the nerve branches providingsensitivity to the flank dermatomes has been suggested [5], in case of irritation of the pleural branches of the intercostal nerves. Morecroft and Learecallthat the respiratorytreedevelopsfrom the primitive foregut [8]. Nociceptive messages from structures originatingfromthis

primitive intestine are poorlylocalised, as are theirrepresentations at the medullarylevel. Theseparticularities of innervation couldexplainupper abdominal pain in PE by a "sensoryoverlap". In fact, the causes of abdominal pain are interrelated and not completelyunderstood.

Without a high index of suspicion, PE is an easilymisseddiagnosiswithdisastrousconsequences. Therefore, itshouldbeconsidered in the differential for a variety of symptoms, including abdominal pain (9,10).

PE can take on the appearance of a variety of otherentities and physiciansshouldbeaware of the varioussigns, symptoms and radiographicfindingsthat can lead to this life-savingdiagnosis.

Conclusion:-

The presentation of PE isprotean and can take on the appearance of abdominal pain, up to and including a surgicalbelly. Itsmisleadingsemiologyrequiresgreat vigilance. The presence of a pleural component to this abdominal pain shouldalert the clinician to the possibility of an extra-abdominal aetiology, notably PE, especially in the presence of dyspnoea or polypnoea.

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