

An Iatrogenic Massive Hemothorax

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Traumatic chest injuries pose a diagnostic and therapeutic challenge. In this report we present the case of an iatrogenic massive hemothorax in a multi-trauma patient admitted to our emergency room. The case emphasizes the challenge of admitting a partially treated patient transferred to a tertiary trauma center from a peripheral primary hospital.

PATIENT DESCRIPTION

The patient was a 20 year old male construction worker who had fallen from a platform 3 m high. Prior to transfer, in the peripheral primary hospital the patient underwent a total body computed tomography scan that demonstrated a severely displaced comminuted pelvic fracture and a small

amount of blood in the peritoneal cavity. Due to hemodynamic instability and new-onset peritonitis, the patient underwent an emergency laparotomy with partial small bowel resection (with an automatic stapler) and packing of the abdominal cavity with temporary closure using a bogota bag. During the operation, the patient received 6 units of packed cells, fresh frozen plasma, factor VII and cryoprecipitate through peripheral lines and a right subclavian central line catheter that was inserted in the operating room. The patient was then transferred anesthetized and ventilated to our tertiary trauma center, which is 3 hours away. During transfer, the patient received 2 additional units of packed cells through the central line catheter.

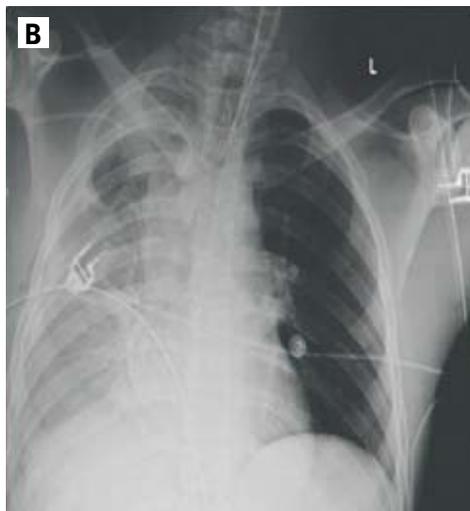
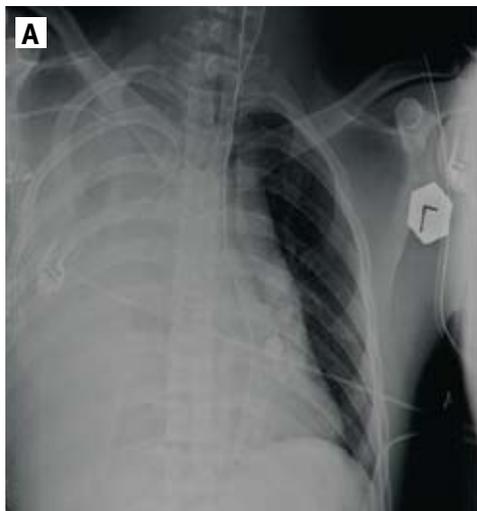
Upon arrival at our center, the patient was hemodynamically stable, anesthetized and ventilated. Oxygen saturation was 93%. A chest X-ray showed a "white lung" on the right [Figure A]. Following the assumption that the patient was suffering from a large hemothorax, a decision was made to insert a chest tube. Upon

insertion 1500 ml of blood were drained. A second X-ray demonstrated some improvement, with the chest tube located in the lower part of the thorax [Figure B]. It is important to note that the CT in the primary hospital did not show a thoracic injury. With a working diagnosis of a diaphragmatic tear with blood from the partially treated abdominal injury entering the thoracic cavity the patient was taken to the operating room [1].

Meanwhile, a second look at the chest X-rays showed an improper location of the central line [Figure A & B] [2]. Upon stopping resuscitation through the catheter the chest tube immediately stopped draining fluid. No diaphragmatic tear was found at surgery.

COMMENT

The issue of recognition and treatment of traumatic chest injuries is ongoing. A review of the Israeli literature of the last few years revealed case reports of chest trauma and its complications such as



[A] Chest X-ray on admission to hospital
[B] Chest X-ray including the wrong position of the central venous catheter

pneumopericardium [3] or injury to the trachea [4]. Our patient demonstrates the challenge of coping with a partially treated patient [5], transferred to a trauma center from a primary care facility. A full reexamination of all tubes and lines entering the patient is crucial. In the current case, a close examination of the chest X-ray would have revealed the reason for the hemothorax, leading to an earlier cessation of use of this misplaced central line.

This case underscores that no assumptions should be made and that the full advanced trauma life support algorithm

be conducted even though our patient was transferred from another hospital. Looking with unbiased eyes, performing the whole ATLS scheme including a new X-ray and not relying on the previous CT scan could help the trauma team reach the correct conclusions.

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ATLS = advanced trauma life support

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Capsule

miR141 and miR-200a act on ovarian tumorigenesis by controlling oxidative stress response

Although there is evidence that redox regulation has an essential role in malignancies, its impact on tumor prognosis remains unclear. Mateescu and co-authors show crosstalk between oxidative stress and the miR-200 family of microRNAs that affects tumorigenesis and chemosensitivity. miR-141 and miR-200a target p38 α and modulate the oxidative stress response. Enhanced expression of these microRNAs mimics p38 α deficiency and increases tumor growth in mouse models, but it also improves the response to chemotherapeutic agents. High-grade human ovarian adenocarcinomas that accumulate

miR-200a have low concentrations of p38 α and an associated oxidative stress signature. The miR200a-dependent stress signature correlates with improved survival of patients in response to treatment. Therefore, the role of miR-200a in stress could be a predictive marker for clinical outcome in ovarian cancer. In addition, although oxidative stress promotes tumor growth, it also sensitizes tumors to treatment, which could account for the limited success of antioxidants in clinical trials.

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Eitan Israeli

Capsule

Differential estrogen receptor binding associated with clinical outcome in breast cancer

Estrogen receptor- α (ER) is the defining and driving transcription factor in the majority of breast cancers and its target genes dictate cell growth and endocrine response, yet genomic understanding of ER function has been restricted to model systems. Ross-Innes et al. have mapped genome-wide ER-binding events, by chromatin immunoprecipitation followed by high-throughput sequencing (ChIP-seq), in primary breast cancers from patients with different clinical outcomes and in distant ER-positive metastases. The authors found that drug-resistant cancers still recruit ER to the chromatin, but that ER binding is a dynamic process with the acquisition of unique ER-binding regions in tumors from patients who are likely to relapse. The acquired ER regulatory regions associated with poor clinical outcome observed in primary tumors reveal

gene signatures that predict clinical outcome in ER-positive disease exclusively. They found that the differential ER-binding program observed in tumors from patients with poor outcome is not due to the selection of a rare subpopulation of cells but to the FOXA1-mediated reprogramming of ER binding on a rapid timescale. The parallel redistribution of ER and FOXA1 binding events in drug-resistant cellular contexts is supported by histological co-expression of ER and FOXA1 in metastatic samples. By establishing transcription-factor mapping in primary tumor material, they show that there is plasticity in ER-binding capacity, with distinct combinations of *cis*-regulatory elements linked with the different clinical outcomes.

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Eitan Israeli

“Every man is a damned fool for at least five minutes every day. Wisdom consists in not exceeding the limit”

Elbert Hubbard (1856-1915), American writer, publisher, artist and philosopher