NON-THROMBOTIC PULMONARY EMBOLISM FOLLOWING HYALURONIC ACID BREAST FILLER INJECTION: A CASE REPORT
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ABSTRACT

Hyaluronic acid (HA) is a commonly used injectable breast enhancement material. In the right set of hands, it is usually a safe injectable material. We present a case of a 35 year old female who underwent hyaluronic acid injection by an unlicensed practitioner, presenting 2 weeks later with acutely worsening 4 days history of shortness of breath and pleuritic chest pain. She had low grade pyrexia at 37.8°C. She was tachycardic and tachypnoic, with her oxygen saturation around 85% on air during presentation. Her serum white cell count was normal. She underwent an urgent CT pulmonary angiogram on day 1 of admission, which revealed no pulmonary artery clots but extensive bilateral peripherally located wedged-shaped ground glass opacities with no lobar predisposition. Given her recent history of breast filler injection, she was treated as non-thrombotic pulmonary embolism (NTPE). She was started on deep vein thrombosis prophylaxis, commenced on corticosteroids therapy and started on antibiotics. She continued to have supplementary oxygen and was discharged with temporary oxygen supplement at home. Follow-up CT done a few months later demonstrated practically complete resolution of the initial changes. In our poster, we briefly outlined some literature review of NTPE including its aetiology, imaging characteristics and management.

Keyword: Non-thrombotic pulmonary embolism, breast filler, hyaluronic acid

CASE PRESENTATION

A 35 year old female presented to the emergency department with 4 days history of worsening dyspnoea and pleuritic chest pain, associated with low grade fever and dry cough. She denies any sore throat or other relevant symptoms. Her oxygen saturation on arrival was 85-90% on room air. Her electrocardiogram (ECG) demonstrated the presence of sinus tachycardia.

Her serological infection markers were entirely normal. However her arterial blood gas analysis returned borderline respiratory alkalosis (pH 7.46, pCO₂ 27.8, HCO₃ 22.1). Her initial chest radiograph demonstrated the presence of bilateral lower zones reticular opacities, superimposed with rather opaque looking breast shadows.

On further questioning, she reported undergoing HA injection for breast fillers by unlicensed practitioner about 2 weeks prior - raising the suspicion of possible non-thrombotic pulmonary embolism. She then underwent an urgent CT pulmonary angiogram.

The CT demonstrated no filling defects within the pulmonary artery circulation. However numerous wedge shaped air space ground glass densities with lower lobe predominance observed. Widespread nodular densities are also observed within both breast parenchyma.

She was treated with supplementary oxygen therapy, for which she continuously had up at home until 1 month following discharge. She was also commenced on tapering steroid regime, as well as started on DVT prophylaxis in ward.

DISCUSSION

Non-thrombotic pulmonary embolism (NTPE) is defined as pulmonary arteries embolization by typically microscopic fragments of non-thrombotic material; which can consist of tissues, organisms, foreign material, chemical agents or gas(1). Compared to the typical bland pulmonary thromboembolism, NTPE is far less common. NTPE often presents with atypical clinical features and is often associated with quite specific clinical setting (2).

Our patient was afflicted with the so-called particulate material embolism. This usually occurs in the context of intravenous injection of illicit drugs or drugs intended for extra-vascular administration. We postulate that some of the hyaluronic acid injected may have incidentally introduced into the intraparenchymal vessels, resulting in incidental embolism.

The pathogenesis of NTPE is also more complex than mechanical vascular obstruction and is related to the underlying embolic material (1). The intra-vascular injection of particulate material is hypothesised to induce pulmonary granulomatosis, occurring due to delayed hypersensitivity response. The granuloma formation and subsequent fibrosis result in eventual distortion of lung architecture. Pulmonary hypertension may occur in chronic cases (1).

Imaging findings in NTPE vary according to offensive material. In previous reports of HA embolism, some authors have described diffuse ground glass densities with basal predominance similar to our case (3). Dilatations of the pulmonary arteries and right ventricular enlargement have also been found. These imaging features are non-specific and can occur in other conditions (2). NTPE is typically diagnosed based on clinical history coupled with imaging findings. However in a few cases, diagnosis is made either via tissue biopsy or post-mortem diagnosis.

REFERENCES