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When Contrast Leaves the Lungs Wet: Recognising Non-cardiogenic Pulmonary Oedema After Contrast CT

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Abstract

Contrast media are widely used in diagnostic and interventional imaging and are generally considered safe. Non-cardiogenic pulmonary oedema after intravenous iodinated contrast can be difficult to recognise, particularly in patients with pre-existing left ventricular dysfunction, in whom it may be mistaken for cardiogenic pulmonary oedema. We report a case of a 73-year-old man with a documented left ventricular ejection fraction (LVEF) of 39% and scarring consistent with a previous myocardial infarction who developed acute pulmonary oedema 30 minutes after receiving Iohexol (Omnipaque) for outpatient CT coronary angiography.

The patient's primary complaint was breathlessness, and he denied any chest pain. A chest X-ray taken in the emergency department showed florid pulmonary oedema. He was initially given a glyceryl trinitrate infusion and a single dose of 80 mg of intravenous furosemide, following which he developed hypotension. He subsequently received 250 mL fluid boluses (Plasma-Lyte 148), to a total of 1000 mL of intravenous fluid. Non-invasive ventilation (NIV) was initially used and was later changed to oxygenation via nasal cannula. He improved symptomatically, and his blood pressure recovered; he was successfully weaned off oxygen the next day. His CT coronary angiogram performed before the presentation was reviewed by the Radiology team, which did not demonstrate obstructive coronary disease, and a large-volume pulmonary embolism was also ruled out. Two high-sensitivity troponin results were negative, and his N-terminal pro-B-type natriuretic peptide (NT-proBNP) level, measured the following day, was 687 pg/mL. A consensus was reached that this was an uncommon reaction to the contrast.

Non-cardiogenic pulmonary oedema should be considered in any patient who develops acute respiratory distress following contrast administration. Early recognition, exclusion of other cardiac causes, and prompt supportive care can result in rapid recovery. Clinicians and radiologists should be aware of this reaction, because its management, which involves NIV and cautious intravenous fluid replacement, differs from that of cardiogenic pulmonary oedema.

Categories: Radiology, Cardiology, Internal Medicine
Keywords: contrast induced pumonary edema, iodinated contrast media, iohexol, non-cardiogenic pulmonary oedema, n-terminal pro-b-type natriuretic peptide, reaction to non ionic contrast dye

Introduction

CT with intravenous iodinated contrast media is a routine investigation that is used widely across hospitals. The introduction of non-ionic, low-osmolar agents (a type of iodinated contrast) has substantially reduced the frequency of adverse reactions. In a large Japanese cohort of over 330,000 patients, adverse reactions occurred in 12.66% of patients receiving ionic contrast media compared with 3.13% of those receiving non-ionic agents, and severe reactions occurred in 0.22% and 0.04%, respectively [1]. With non-ionic agents in contemporary practice, allergic-like reactions occur in approximately 0.6% of patients, and most are mild [2]. The common side effects of contrast media include nausea, vomiting, urticaria, and pruritus [3]. More severe reactions include anaphylactic shock, severe angioedema, pulmonary oedema, and cardiac arrest, which have been reported to occur rapidly, within 20 minutes of injection [4]. Both allergic (IgE-mediated) and non-allergic mechanisms have been proposed for these hypersensitivity reactions [5].

Pulmonary oedema following contrast administration may be cardiogenic, resulting from raised pulmonary capillary hydrostatic pressure, or non-cardiogenic, resulting from increased permeability of the alveolar-capillary barrier [6]. This distinction is clinically important because management differs. In contrast-induced non-cardiogenic pulmonary oedema, the recommended treatment includes continuous positive airway pressure (CPAP) and intravenous fluids, while avoiding diuretics, and fatal outcomes have been reported [7]. Cases have also been described with non-ionic, low-osmolar agents used for coronary angiography [8]. We report a case of non-cardiogenic pulmonary oedema as a side effect of iohexol (Omnipaque), a non-ionic, water-soluble, low-osmolality contrast medium, in a patient with pre-existing left ventricular systolic

dysfunction, in whom the initial clinical picture suggested a cardiogenic cause. The treatment differs from that of routine management of cardiogenic pulmonary oedema, and this case highlights the diagnostic challenge and specific management considerations in contrast-induced pulmonary oedema.

Case Presentation

A 73-year-old man who was previously fit and well was undergoing investigation for syncopal episodes. Both his transthoracic echocardiogram (ECHO) and cardiac MRI (cMRI) demonstrated moderately impaired left ventricular systolic function, with an ejection fraction of 39%. There was evidence of basal to mid-lateral wall thinning and scarring, consistent with a previous myocardial infarction. Given the suspicion of underlying coronary artery disease, a CT coronary angiogram was arranged.

The patient underwent the CT scan on Day 1 at 09:49 am, during which he received iohexol (Omnipaque), a non-ionic, water-soluble, low-osmolality contrast medium. Following the scan, he remained well and was discharged home. However, he re-presented to the hospital at approximately 2:00 pm with acute-onset breathlessness. His symptoms had started approximately 30 minutes after the scan. He also reported chest tightness but denied any chest pain. On arrival, he required supplemental oxygen via a non-rebreather mask at an FiO_2 of 60% to maintain an oxygen saturation of 92%. Clinical examination revealed a regular pulse and normal heart sounds. Bibasal crepitations were present on auscultation, while his abdomen was soft and non-tender. There was no evidence of peripheral oedema. His admission electrocardiogram (ECG) demonstrated normal sinus rhythm, with no acute ST-segment changes (Figure *1*). His chest X-ray revealed florid pulmonary oedema, without significant pleural effusions or lobar collapse (Figure *2*).

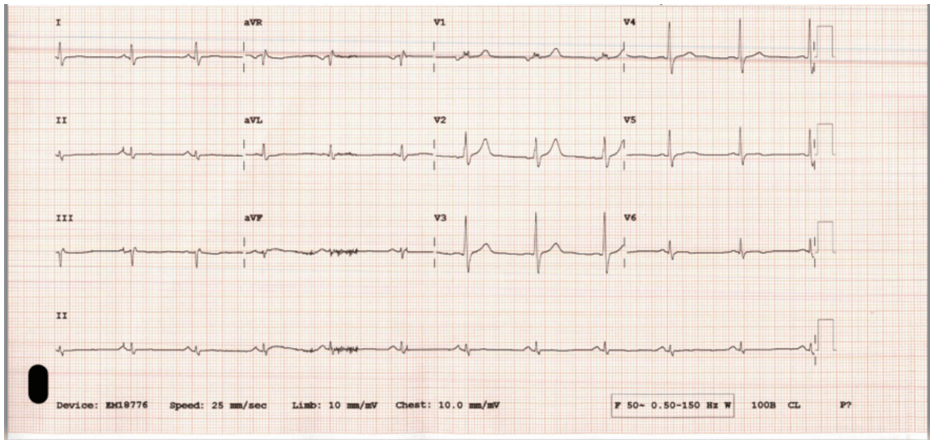


FIGURE 1: ECG on Day 1

Twelve-lead electrocardiogram on Day 1 showing sinus rhythm at 67 beats per minute with a normal axis, with no acute ST-segment or T-wave changes

ECG: electrocardiogram

The emergency department resident initiated non-invasive ventilation (NIV) and a glyceryl trinitrate infusion, which was continued for two hours before being discontinued due to the development of hypotension. The patient also received a single 80 mg dose of intravenous furosemide. He subsequently improved, transitioning from a 15 L non-rebreather mask to nasal cannula oxygen, requiring an FiO_2 of 36% to maintain oxygen saturations of 94%.

However, due to persistent hypotension, with a blood pressure of approximately 82/50 mmHg (compared with an admission blood pressure of 150/67 mmHg), he was administered a 250 mL crystalloid fluid bolus (Plasma-Lyte 148). He responded to the fluid bolus and subsequently received a total of 1,000 mL of intravenous fluids overnight, administered in small volumes. By the following morning, his symptoms had improved, his blood pressure had recovered, and he was successfully weaned off supplemental oxygen. A repeat chest X-ray demonstrated resolution of the pulmonary oedema (Figure *3*).

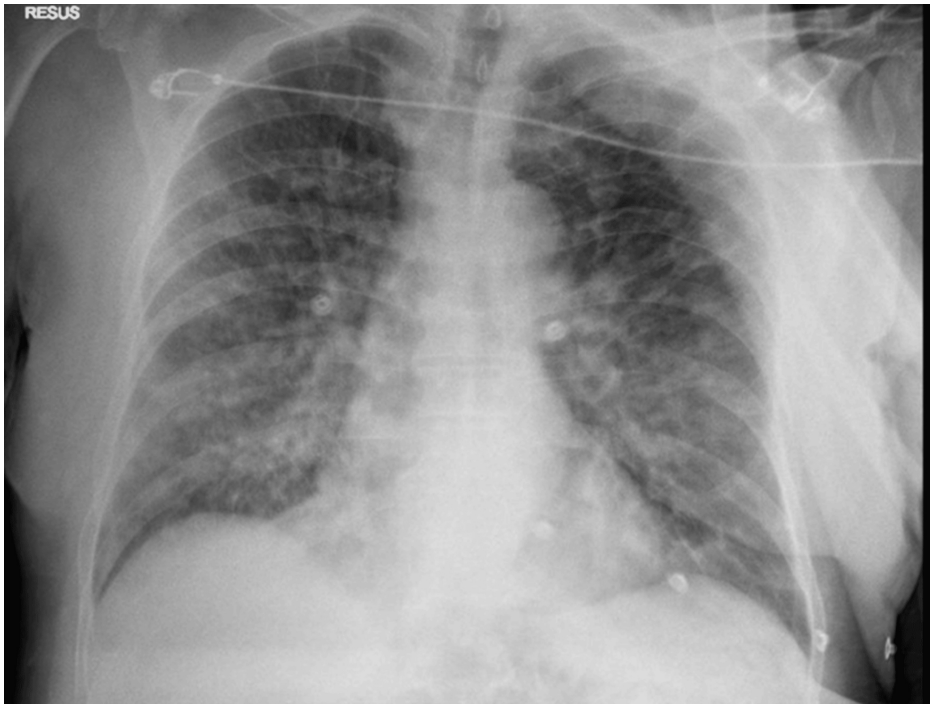


FIGURE 2: Admission Chest X-ray demonstrates florid pulmonary oedema

Changes seen are bilateral diffuse alveolar and interstitial opacities, most marked in the perihilar regions and lower zones, without significant pleural effusion or lobar collapse, keeping in with florid pulmonary oedema

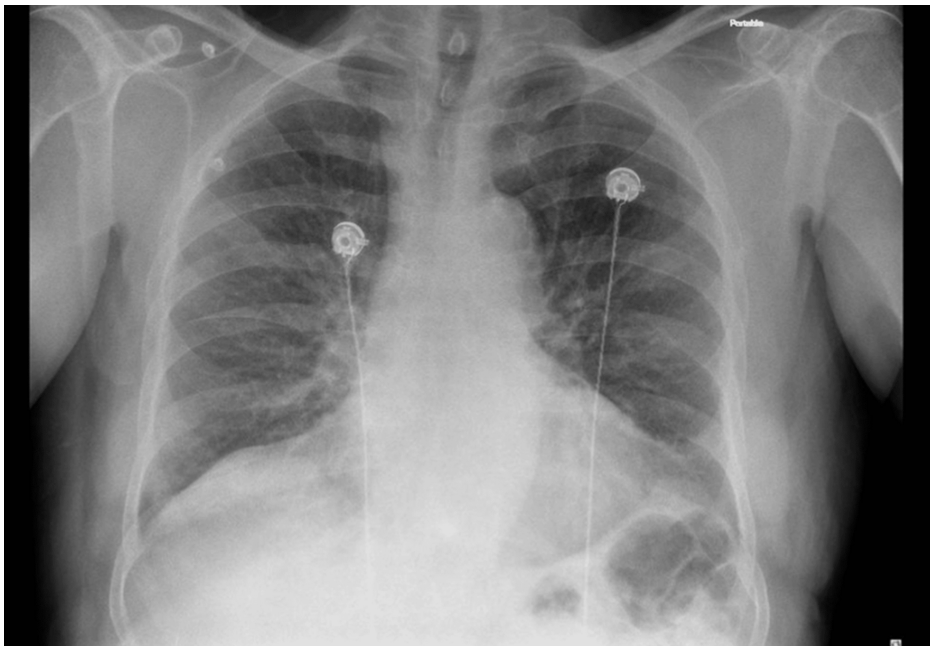


FIGURE 3: Chest X-ray from Day 3 demonstrates resolution of florid pulmonary oedema

The working diagnosis was contrast-induced non-cardiogenic pulmonary oedema. Two serial measurements of high-sensitivity troponin I were 10 and 17 ng/L, respectively, both within the normal range (Table *1*). His blood tests, including urea and electrolytes and full blood count, were unremarkable (Table *1*). C-reactive protein was mildly elevated at 47 mg/L. His N-terminal pro-B-type natriuretic peptide (NT-proBNP), measured the following day after receiving furosemide the previous evening, was 687 pg/mL. This was

within the age-adjusted cut-off of 900 pg/mL for acute heart failure in patients aged 50-75 years [9,10]. A repeat echocardiogram performed the following day demonstrated hypokinesia of the basal to mid anterolateral, inferolateral, and inferior walls, consistent with the findings on previous imaging. The ejection fraction was 53%, representing an interval improvement compared with previous imaging (Video *1*). Serum tryptase levels were also measured the following day, approximately 24 hours after symptom onset; therefore, the results were not considered clinically informative.

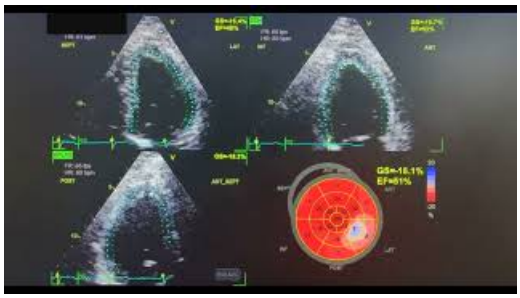
Test	Day 1, 14:00	Day 1, 22:14	Day 2, 05:10	Reference range (units)
Sodium	142	140	-	133–146 mmol/L
Potassium	Haemolysed	4.9	-	3.5–5.3 mmol/L
Urea	4.7	6.3	-	2.5–7.8 mmol/L
Creatinine	85	103	-	60–110 µmol/L
eGFR	78 ↓	62 ↓	-	> 90 mL/min/1.73 m²
Total bilirubin	8	-	-	< 21 µmol/L
Alkaline phosphatase	81	45	-	30–130 U/L
Alanine transaminase	14	-	-	< 40 U/L
Calcium	-	2.07 ↓	-	2.20–2.60 mmol/L
Adjusted calcium	-	2.33	-	2.20–2.60 mmol/L
Phosphate	-	1.26	-	0.80–1.50 mmol/L
Magnesium	-	-	0.78	0.70–1.00 mmol/L
CRP	<1.0	47 ↑	-	< 5 mg/L
High-sensitivity troponin	10	17	-	< 34 ng/L
Total cholesterol	-	4.8	-	< 5.0 mmol/L
HDL cholesterol	-	1.17	-	> 1.0 mmol/L
Non-HDL cholesterol	-	3.6	-	< 4.0 mmol/L
Total cholesterol:HDL ratio	-	4.1 ↑	-	< 4.0
Triglycerides	-	0.4	-	< 1.7 mmol/L
LDL cholesterol	-	3.4 ↑	-	< 3.0 mmol/L
Albumin	37	30 ↓	-	35–50 g/L
NT-proBNP	-	-	687 ↑	< 400 ng/L
Haemoglobin	144	155	-	130–180 g/L
WBC	4.9	10.3	-	4.0–11.0 ×10⁹/L
Platelets	228	225	-	150–450 ×10⁹/L
RBC	5.45	5.72	-	4.5–6.5 ×10¹²/L
Haematocrit	0.442	0.467	-	0.40–0.54 L/L
MCV	81.1	81.7	-	80–100 fL
MCH	26.3 ↓	27.1	-	27–32 pg
MCHC	326	331	-	320–360 g/L
RDW	14	13.2	-	11.5–14.5%
MPV	8.7	9	-	7.5–11.5 fL
Neutrophils (abs)	2.5	9.5 ↑	-	2.0–7.5 ×10⁹/L
Lymphocytes (abs)	1.7	0.2 ↓	-	1.0–4.0 ×10⁹/L

Eosinophils (abs)	0.2	0	-	0.0–0.4 ×10⁹/L
Monocytes (abs)	0.6	0.6	-	0.2–1.0 ×10⁹/L
Basophils (abs)	0	0	-	0.0–0.1 ×10⁹/L
PT	-	12.7	13.3	10.0–14.0 s
PT ratio	-	1.05	1.1	0.8–1.2
APTT	-	31.3	30.1	25.0–36.0 s
APTT ratio	-	1.01	0.97	0.8–1.2
D-dimer	-	-	20,000 ↑	< 500 µg/L FEU

TABLE 1: Laboratory results during admission

↑ indicates a value above the reference range; ↓ indicates a value below the reference range. Reference ranges are for adult males

eGFR: estimated glomerular filtration rate; CRP: C-reactive protein; HDL: high-density lipoprotein; LDL: low-density lipoprotein; NT-proBNP: N-terminal pro-B-type natriuretic peptide; WBC: white blood cells; RBC: red blood cells; MCV: mean corpuscular volume; MCH: mean corpuscular haemoglobin; MCHC: mean corpuscular haemoglobin concentration; RDW: red cell distribution width; MPV: mean platelet volume; abs: absolute count; PT: prothrombin time; APTT: activated partial thromboplastin time; FEU: fibrinogen-equivalent units



VIDEO 1: Transthoracic echocardiogram on Day 2 of admission

The video shows the apical four-chamber (top left), apical two-chamber (top right), and apical long-axis (bottom left) views used to calculate left ventricular global longitudinal strain (GLS), with the corresponding 17-segment bull's-eye polar map (bottom right). Global longitudinal strain was −18.1% (four-chamber: −16.4%, two-chamber: −19.7%, long-axis: −18.2%), with an ejection fraction of 51%. The polar map shows reduced strain in the basal to mid inferolateral and anterolateral segments, consistent with the known previous infarction

View video here: https://youtu.be/KY53pwQYAlk

The CT coronary angiogram performed just before his presentation was reviewed by the Radiology team. It showed no obstructive coronary disease and also excluded any large-volume central pulmonary embolism or lung lesion or scarring (Figure 4). Given these findings, we did not consider further contrast-enhanced imaging necessary. We discussed the patient at the Radiology CT multidisciplinary team meeting, and the consensus was that he had an unpredictable, idiosyncratic reaction to the contrast. A literature review at the time supported the impression that these patients respond poorly to loop diuretics, and that the recommended treatment was a combination of positive-pressure ventilation and cautious intravenous fluid replacement [7]. Alternative diagnoses, including paradoxical embolism and an arrhythmic component, were also carefully considered.

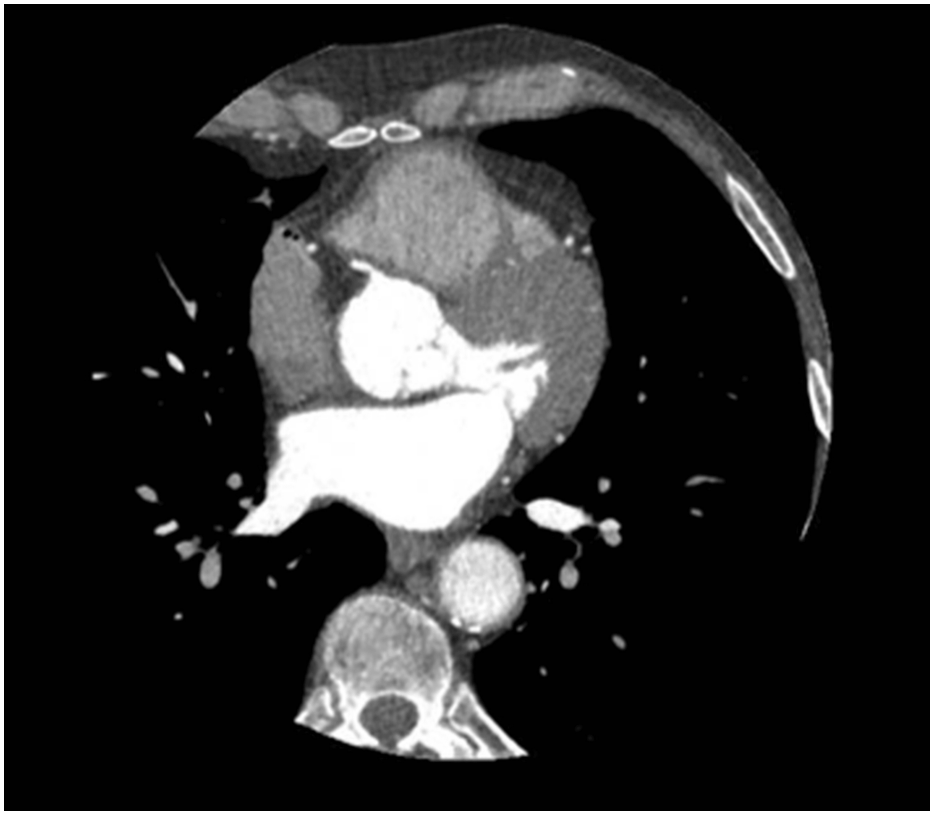


FIGURE 4: Axial (horizontal) cross-section of CT coronary angiogram from Day 1

The image shows a cross-section of the heart, reported to have normal coronaries, no pulmonary embolism, and no lung lesion/scarring

CT: computed tomography

The patient recovered well and was discharged from the hospital. Four weeks later, he underwent a bubble echocardiogram, which excluded a patent foramen ovale. He was then followed up in the Cardiology clinic, where he was offered Holter monitoring and an implantable loop recorder to investigate a possible ventricular arrhythmia.

Discussion

Contrast-induced adverse reactions from intravenous contrast occur in 1-5% of cases annually [3]. These reactions are often not severe and include angioedema, urticaria, and bronchospasm. Rarely, they can be life-threatening, with airway compromise and profound hypotension. Some sources report that these reactions account for 0.01-0.2% of all reactions [11]. Pulmonary oedema is a well-known reaction to intravenous contrast media. It has an overall reported incidence in the region of 0.001-0.008% of administrations, but accounts for 10-20% of fatal contrast reactions [4,7,8]. Within this broader category, non-cardiogenic subtypes are rare, with only a small number of cases reported [4,8,12]. Delayed presentations, such as ours, occurring approximately 30 minutes after exposure to a non-ionic, low-osmolar agent, remain unusual [5,13].

Pathophysiology

The mechanism of contrast-induced non-cardiogenic pulmonary oedema is incompletely understood and likely multifactorial. A popular hypothesis is direct injury to the alveolar-capillary endothelium, mediated by complement activation and the release of vasoactive mediators, including histamine, leukotrienes, and platelet-activating factor [4,12,14]. The resulting increase in microvascular permeability allows protein-rich fluid to extravasate into the interstitial space and alveoli. This produces a clinical and radiographic picture indistinguishable from cardiogenic oedema, but without elevated left atrial pressure [6,14]. Other contributory mechanisms include a direct irritant effect of iodinated contrast media (ICM) on the pulmonary vasculature, transient pulmonary hypertension, and an osmotic shift of intravascular water [4,12]. Because these reactions are not IgE-mediated, prior uneventful exposure does not predict tolerance [14]. This entity may be an analogue of capillary leak syndrome (CLS), in which patients develop increased fluid

extravasation, intravascular hypovolaemia, extravascular oedema, and hypoperfusion.

Diagnostic challenge and management

Distinguishing between non-cardiogenic and cardiogenic pulmonary oedema was the diagnostic challenge in our patient. Our patient had a previously documented left ventricular ejection fraction of 39% with regional wall motion abnormalities from a prior infarction. On the face of it, the picture pointed to a cardiogenic mechanism. However, several lines of evidence argued against this. His CT coronary angiogram performed immediately before symptom onset excluded both obstructive coronary disease and a large central pulmonary embolism. Two sets of high-sensitivity troponin I were negative, making acute ischaemia unlikely. His echocardiogram performed the day after his presentation showed a left ventricular ejection fraction of 53%, an improvement on his baseline of 39%. There was no suggestion of an underlying cardiomyopathy on his previous imaging, including cardiac MRI. The natriuretic peptide level also weighed in favour of a non-cardiogenic cause.

In this context, the reliability of brain natriuretic peptide (BNP) is unclear. BNP remained normal in a fatal case involving iso-osmolar iodixanol reported by Yi and Li [15], despite radiological evidence of pulmonary oedema. Iqbal and colleagues' case report cautions that reliance on natriuretic peptide values in patients with underlying cardiac disease can lead to misdiagnosis as cardiogenic oedema [16]. Our case reinforces this diagnostic dilemma.

Other investigations completed the diagnostic picture. A bubble echocardiogram performed four weeks later excluded a patent foramen ovale. Ambulatory rhythm monitoring was arranged to rule out arrhythmia. Serum tryptase (a key marker for anaphylaxis) did not add any diagnostic value because it was drawn the next day; the National Institute for Health and Care Excellence (NICE) recommends sending an acute sample ideally within one to two hours and no later than four hours after symptom onset, and a baseline sample at least 24 hours later [17]. An earlier sample would have helped to distinguish a mast-cell-mediated allergic-like mechanism from a purely complement- or osmotic-driven event.

A clinical picture of bilateral crepitations and radiographic pulmonary oedema tends to trigger an automatic instinct to prescribe intravenous furosemide, as happened in our case. The mechanism here was capillary leak, not fluid overload. Intravascular volume is usually normal or low, and diuresis in this setting can worsen hypotension without addressing the underlying lung injury [7,8,12]. The fatal case reported by Paul and George illustrates this danger [7]. Their patient received 60 mg of intravenous furosemide without any positive-pressure ventilation and became progressively hypotensive, dying subsequently. Kang and Nah described a similar pattern of presentation, in which the patient responded poorly to diuretics and improved only after positive-pressure ventilation coupled with cautious fluid replacement [8].

Our patient's clinical course fits a similar pattern. After he received 80 mg of intravenous furosemide and a glyceryl trinitrate infusion, he developed significant hypotension. Symptomatic and haemodynamic improvement, with blood pressure recovery, followed the introduction of non-invasive ventilation (NIV) and 1000 mL of crystalloid fluid given as slow boluses. Our patient has been recorded as having a severe hypersensitivity-like reaction to iohexol (Omnipaque). If future contrast-enhanced imaging is unavoidable, referral to a drug-allergy specialist is recommended, and switching to a structurally different iodinated agent may reduce the risk of recurrence [18].

Limitations

This case report has several limitations. The diagnosis of non-cardiogenic pulmonary oedema is one of exclusion. Even with a supportive NT-proBNP, a mixed cardiogenic and non-cardiogenic picture cannot be definitively ruled out. The NT-proBNP was a single value drawn the next day, after intravenous furosemide administration, and is therefore likely to underestimate the true acute peak. Had the sample been drawn on presentation and before diuretics were given, it would have added more diagnostic confidence. Serum tryptase was sampled outside the recommended diagnostic window and was uninformative. The patient did not have allergy testing, which limits the choice of a safe alternative contrast agent if future imaging is needed.

Conclusions

Non-cardiogenic pulmonary oedema is a rare but potentially fatal complication of intravenous iodinated contrast. It can occur with low-osmolar, non-ionic agents and may present 30 minutes or more after exposure. In a patient with pre-existing left ventricular dysfunction, the diagnosis is easily missed, and reflex treatment as cardiogenic pulmonary oedema with diuretics can cause harm. Acute pulmonary oedema within an hour of iodinated contrast should prompt active consideration of a non-cardiogenic mechanism, even where the cardiac history seems to explain the picture. A natriuretic peptide value that is discordant with clinical severity may be misleading. NIV with maintenance of euvolaemia, rather than reflex diuresis, is the therapeutic cornerstone.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Sugna Divakar Prabhu

Acquisition, analysis, or interpretation of data: Sugna Divakar Prabhu, Sohaib Minhas, Neha Maheshkumar, Satnam Singh

Drafting of the manuscript: Sugna Divakar Prabhu, Sohaib Minhas, Neha Maheshkumar

Critical review of the manuscript for important intellectual content: Sugna Divakar Prabhu, Sohaib Minhas, Neha Maheshkumar, Satnam Singh

Supervision: Satnam Singh

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Katayama H, Yamaguchi K, Kozuka T, Takashima T, Seez P, Matsuura K: Adverse reactions to ionic and nonionic contrast media. A report from the Japanese Committee on the Safety of Contrast Media. Radiology. 1990, 175:621-8. 10.1148/radiology.175.3.2343107
2. Wang CL, Cohan RH, Ellis JH, Caoili EM, Wang G, Francis IR: Frequency, outcome, and appropriateness of treatment of nonionic iodinated contrast media reactions. AJR Am J Roentgenol. 2008, 191:409-15. 10.2214/AJR.07.3421
3. Bottinor W, Polkampally P, Jovin I: Adverse reactions to iodinated contrast media . Int J Angiol. 2013, 22:149-54. 10.1055/s-0033-1348885
4. Pincet L, Lecca G: Acute pulmonary edema induced by non-ionic low-osmolar radiographic contrast media . Open Access Emerg Med. 2018, 10:75-9. 10.2147/OAEM.S154081
5. Brockow K: Immediate and delayed reactions to radiocontrast media: is there an allergic mechanism? . Immunol Allergy Clin North Am. 2009, 29:453-68. 10.1016/j.iac.2009.04.001
6. Ware LB, Matthay MA: Clinical practice. Acute pulmonary edema. N Engl J Med. 2005, 353:2788-96. 10.1056/NEJMcp052699
7. Paul RE, George G: Fatal non-cardiogenic pulmonary oedema after intravenous non-ionic radiographic contrast. Lancet. 2002, 359:1037-8. 10.1016/S0140-6736(02)08068-6
8. Kang MH, Nah JC: A delayed, unusual non-cardiogenic pulmonary edema after intravascular administration of non-ionic, low osmolar radiocontrast media for coronary angiography. Korean Circ J. 2013, 43:500-3. 10.4070/kcj.2013.43.7.500
9. Mueller C, McDonald K, de Boer RA, et al.: Heart Failure Association of the European Society of Cardiology practical guidance on the use of natriuretic peptide concentrations. Eur J Heart Fail. 2019, 21:715-31. 10.1002/ejhf.1494
10. Januzzi JL Jr, Chen-Tournoux AA, Christenson RH, et al.: N-terminal pro-B-type natriuretic peptide in the emergency department: the ICON-RELOADED study. J Am Coll Cardiol. 2018, 71:1191-200. 10.1016/j.jacc.2018.01.021
11. Borish L, Matloff SM, Findlay SR: Radiographic contrast media-induced noncardiogenic pulmonary edema: case report and review of the literature. J Allergy Clin Immunol. 1984, 74:104-7. 10.1016/0091-6749(84)90095-2
12. Solomon DR: Anaphylactoid reaction and non-cardiac pulmonary edema following intravenous contrast injection. Am J Emerg Med. 1986, 4:146-9. 10.1016/0735-6757(86)90161-0
13. Bristedt P, Tylén U: Pulmonary edema following intravenous injection of nonionic low-osmolar contrast medium--appearance on HRCT. A case report. Acta Radiol. 1998, 39:81-3. 10.1080/02841859809172155
14. Murray JF: Pulmonary edema: pathophysiology and diagnosis . Int J Tuberc Lung Dis. 2011, 15:155-60.
15. Yi KM, Li X: Fatal noncardiogenic pulmonary edema related to nonionic, iso-osmolar iodine contrast medium: one case report. BMC Pulm Med. 2022, 22:118. 10.1186/s12890-022-01908-0
16. Iqbal A, Mahmood R, Jasti M, Moyine C, Adigopula S: Water lung: an interesting case of noncardiogenic pulmonary edema in a heart failure patient. Cureus. 2023, 15:e48814. 10.7759/cureus.48814
17. Anaphylaxis: assessment and referral after emergency treatment (NICE guideline NG258) . (2026). Accessed: October 19, 2026: https://www.nice.org.uk/guidance/ng258.
18. Mervak BM, Davenport MS, Ellis JH, Cohan RH: Rates of breakthrough reactions in inpatients at high risk receiving premedication before contrast-enhanced CT. AJR Am J Roentgenol. 2015, 205:77-84. 10.2214/AJR.14.13810