

Iodide-induced sialadenitis following percutaneous coronary intervention: A case report

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D Curley , D Gent  and S Patel

Abstract

Iodide-induced sialadenitis is a rapid, painless enlargement of salivary glands following administration of iodine-based contrast agents. It has been reported numerous times in the literature; however, the pathogenesis remains unclear. This case report demonstrates how this may present following a coronary angiogram.

Keywords

Iodine, iodide, sialadenitis, percutaneous coronary intervention

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Introduction

Iodide-induced sialadenitis is a rare complication of iodine-based contrast agents. A recent meta-analysis identified 77 cases of iodide-induced sialadenitis worldwide; more work is needed to both increase our understanding of the underlying pathophysiology and potential treatment options in order to educate clinicians about timely recognition of this complication. Iodide-induced sialadenitis is a benign and self-limiting process, and a conservative approach with observation is recommended. The duration of symptoms may be related to advanced age and longer time to symptom onset.¹

While the pathogenesis of iodine-induced sialadenitis is still unknown, it is thought to be an idiosyncratic reaction due to toxic accumulation of the contrast agent with inflammation and oedema. Another theory is that the reaction is due to a pseudo-allergic reaction caused by the accumulation of iodine in the salivary ducts. It is not thought to be a true allergic reaction, and multiple cases have demonstrated negative skin testing results.^{2–4} Imaging of the glands with ultrasound, computed tomography (CT) and magnetic resonance imaging (MRI) shows diffuse gland oedema and enlargement, and routine imaging is not recommended given an appropriate patient history.¹

Case

A 77-year-old lady was admitted from the accident and emergency department with an increased frequency of angina. She had an extensive medical background including

an ST elevation myocardial infarction 4 years earlier treated with percutaneous coronary intervention (PCI) with a drug eluting stent to her right coronary artery, end-stage renal failure with a cadaveric transplant 10 years ago, transient ischaemic attack, hypertension and hypercholesterolaemia. Her baseline creatinine was 170 micromoles/litre.

She lived independently and was a current smoker with a 60 pack year history but did not drink alcohol. There was no significant family history of cardiac disease. She did not have any drug allergies.

The initial examination was unremarkable. Blood tests revealed a stable creatinine of 183 micromol/L, Hb 110 g/L and no high sensitivity Troponin T elevation. Her chest x-ray was unremarkable, and the baseline electrocardiogram (ECG) showed sinus rhythm with an old left bundle branch block. A trans-thoracic echocardiogram on admission showed a reduced left ventricle ejection fraction (45%–50%) without regional wall motion abnormalities. A dobutamine stress echocardiogram (DSE) was performed which had to be terminated early due to the development of non-sustained ventricular tachycardia.

Based on the history and equivocal DSE results, a decision was made to proceed to coronary angiography which

Department of Cardiology, Queen Elizabeth Hospital, London, UK

Corresponding Author:

D Curley, Department of Cardiology, Queen Elizabeth Hospital, London, SE18 4QH, UK.

Email: Daniel.curley@nhs.net





Figure 1. Swelling predominantly in the right parotid and submandibular glands with minor swelling of the left parotid and submandibular glands. Lower facial asymmetry is noted.

revealed significant disease in the first obtuse marginal (OM1) branch and proximal left anterior descending (LAD) artery. The OM1 and LAD stenoses were both functionally significant by fractional flow reserve (FFR) measurement and she was treated with drug eluting stents. Overall, 540 mL of intra-arterial Visipaque (Iodixanol 320 mg/ml) contrast was used, with 500 mL of crystalloid given before and after the procedure.

She developed bilateral neck swelling 6–8 h after angiography (Figure 1) which progressed until 12 h post-procedure. She was reviewed by the on-call medical team and had no airway compromise, cough, fever, night sweats, infectious contacts or symptoms suggestive of thyroid disease. Further questioning revealed that she had mumps as a child and parotid gland swelling 8 years previously following a CT scan which used iodinated contrast medium.

Post-procedure, her creatinine was 163 $\mu\text{mol/L}$, white blood cell count $7.5 \times 10^9/\text{L}$ ($4.3\text{--}10.8 \times 10^9/\text{L}$), free T4 level 15.0 pmol/L (10–20 pmol/L) and thyroid stimulating hormone level 2.46 mIU/L (0.4–4 mIU/L).

An early diagnosis of contrast induced sialadenitis was made and the patient was monitored closely. We did not feel she warranted any imaging given the satisfactory history. She did not receive any specific treatment and her swelling improved over the next 24–48 h with complete resolution 4 days post-procedure.

Discussion

Diagnoses for parotid gland swelling can be divided into infective and non-infective causes. Infective agents include mumps virus (which this patient suffered as a child), Epstein–Barr virus, parainfluenza, adenovirus, influenza A, parvovirus B19 and HIV. Non-infective causes include salivary stones, metabolic disorders (diabetes, cirrhosis and uraemia) and autoimmune diseases (sarcoidosis, Sjogren’s and granulomatosis with polyangiitis). Iatrogenic causes include thiazide diuretics, phenothiazines, thiouracil and iodine contrast media.

The signs and symptoms, alongside the absence of obvious infection, the acute nature of onset and her prior history of neck swelling after a contrast CT scan support the diagnosis of iodide-induced sialadenitis (or ‘iodine mumps’).

Sussman and Miller⁵ first described this in 1956 as a rapid, painless enlargement of the bilateral or unilateral salivary gland. It is a rare but well-recognised complication of iodine-based contrast agents.^{1,6}

It has been postulated that renal disease may be a risk factor⁷ as it appears that the condition is more common in patients with renal dysfunction. There have also been case reports of iodide-induced pancreatitis which is thought to share a similar aetiology.⁸ Imaging was not performed in this case; however, ultrasound findings in similar cases have demonstrated diffuse gland swelling, increased vascularity and prominent ducts. In addition, CT has demonstrated gland enlargement and no fat stranding while MRI has shown oedema of the gland.¹

Iodide-induced sialadenitis is a self-limiting reaction which usually lasts for 2–5 days with complete resolution in less than 2 weeks.⁹ Our patient noticed complete resolution within 4 days. Numerous proposed treatments within the literature include corticosteroids (including pre-medication of ‘at risk’ patients), non-steroidal anti-inflammatory drugs and dialysis. None of these treatments expedited the resolution of symptoms except dialysis which completely resolved the swelling in patients with end-stage renal failure in hours.^{10,11} To date, there have been no reported major complications of iodide-induced sialadenitis. Although rare, it is important that clinicians involved in administering iodine contrast or ordering diagnostic tests which involve iodine-based contrasts are able to identify this complication and reassure their patients if they develop similar symptoms as described.

It is unclear if the dose of iodine-based contrast affects the likelihood of a reaction occurring. A total of 540 mL intra-arterial Visipaque contrast was used in this case and the patient had reported symptoms of sialadenitis following a previous contrast CT scan where she received less than 100 mL of intravenous contrast; 13 months after this case, the patient was admitted with a non-ST elevation myocardial infarction and had PCI to a severe in stent restenosis in the OM branch. She received 69 mL of intra-arterial Visipaque (Iodixanol 320 mg/ml) contrast during this procedure and did not have evidence of sialadenitis post-procedure. The findings in this case challenge the idea that the reaction does not seem to be dose dependent as our patient did not have a reaction with the lowest volume of contrast (69 mL). In

addition, the reaction seems to be independent of the route of administration of the contrast with both intra-arterial and intravenous administration causing a reaction.

As there has been no evidence of major complications to date, it would not be pertinent to deny patients iodine contrast following an episode of iodide-induced sialadenitis, but close monitoring is required. Uniquely, this patient described similar symptoms following a CT scan performed with iodine contrast 8 years previously. This suggests that patients who have developed this reaction in the past are prone to develop this reaction in the future.

Conclusion

Iodide-induced sialadenitis is a rare complication of iodine-based contrast media and its pathogenesis remains unclear. It is a self-limiting condition with no evidence of major complications. Our patient had recurrent reactions which is not uncommon. The route of administration of contrast did not affect outcomes in this case. It is not clear whether the reaction is dose dependent, but our patient did not have any reaction with the smaller volume of contrast. Clinicians should be aware of this reaction following administration of iodine-based contrast media and further work is required to further understand the condition.

Declaration of conflicting interests

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Ethical approval

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Informed consent

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ORCID iDs

D Curley  <https://orcid.org/0000-0003-2864-6877>

D Gent  <https://orcid.org/0000-0002-0365-913X>

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