Tidsskriftet Tidsskrift for Den norske legeforening

Anaphylactic reaction to MRI contrast agent

SHORT CASE REPORT

LINE BROCH

E-mail: line.broch@vestreviken.no
Department of Neurology
Drammen Hospital Vestre Viken Hospital Trust
and
Department of Neurology
Oslo University Hospital, Ullevål
Line Broch, senior consultant and specialist in neurology, and PhD candidate.
The author has completed the ICMJE form and declares no conflicts of interest.

SIMONE WESTER

Department of Anaesthesia, Intensive Care and General Surgery
Drammen Hospital Vestre Viken Hospital Trust
Simone Wester, senior consultant and specialist in anaesthesiology. She has completed the European Diploma in Intensive Care Medicine parts 1 and 2.
The author has completed the ICMJE form and declares no conflicts of interest.

ANNIKEN HASLUND

Department of Diagnostic Imaging
Drammen Hospital
Vestre Viken Hospital Trust
Anniken Haslund, specialist in radiology, senior consultant and head of section.
The author has completed the ICMJE form and declares no conflicts of interest.

ASTRID EDLAND

Department of Neurology
Drammen Hospital
Vestre Viken Hospital Trust
Astrid Edland, senior consultant and specialist in neurology.
The author has completed the ICMJE form and declares no conflicts of interest.

BACKGROUND

Magnetic resonance imaging (MRI) is a non-invasive test with a low complication rate even when using contrast agents. Nevertheless, even routine examinations can cause serious complications, as this case illustrates.

CASE PRESENTATION

Approximately one minute after receiving an intravenous contrast agent containing gadoteric acid (Dotarem®), a woman suffered a generalised seizure and lost

consciousness. The staff immediately moved her out of the MRI examination room. She was flushed, unconscious and had weak pulses. The resuscitation team suspected an anaphylactic reaction. She was treated with epinephrine and IV fluids, but soon developed pulseless electrical activity. She received advanced cardiopulmonary resuscitation with a chest compression device. The contrast agent was removed by dialysis. She gradually recovered and was discharged without significant sequelae eighteen days after the incident.

INTERPRETATION

Anaphylactic shock can occur as a rare complication of MRI with contrast enhancement. The examination should not be performed without a clear indication.

Magnetic resonance imaging is a non-invasive test with a low complication rate even when using contrast agents. Nevertheless, even routine examinations can cause serious complications, as this case report illustrates.

A woman in her fifties was to undergo an outpatient magnetic resonance imaging (MRI) scan with contrast as part of the follow-up of a chronic neurological disorder. She had never been exposed to MRI contrast agents before, but had no known drug allergies.

Approximately one minute after the administration of a gadolinium-based contrast agent, gadoteric acid (Dotarem®), the patient suffered a generalised seizure and became unresponsive. She was immediately removed from the MRI machine and wheeled out of the room on a gurney.

A 'code blue' alert (cardiac arrest) was triggered and a specialty registrar in anaesthesiology arrived one minute later. The patient was by then lying in the recovery position with spontaneous agonal respiration. She was unconscious, flushed over her entire body, and had a weak radial pulse bilaterally. An anaphylactic reaction was immediately suspected, and she received 0.5 mg adrenaline intramuscularly followed by 0.05 mg adrenaline intravenously. Her first measured blood pressure was 144/90 mm Hg and she had sinus tachycardia of 110–130 beats per minute. She was transferred to the intensive care unit with ongoing assisted ventilation. She did not have bronchospasm.

She was intubated immediately upon arrival owing to a Glasgow Coma Scale (GCS) score of 3 and partial airway obstruction. Upon intubation, she received 50 mg esketamine (Ketanest), 0.1 mg fentanyl and 75 mg suxamethonium (Curacit), all intravenously. The placement of the tube was confirmed with end-tidal CO_2 capnography, and the initial reading was 4.5 kPa, indicating adequate circulation. One litre of Ringer's acetate was infused. During further attempts to establish invasive arterial pressure monitoring, pulseless electrical activity (PEA) was detected. Ultrasound revealed no detectable brachial, radial or femoral pulse, despite a stable sinus rhythm of 70 beats per minute. Advanced cardiopulmonary resuscitation was started 27 minutes after the code blue alert.

Echocardiography during resuscitation revealed cardiac standstill, and 1 mg adrenaline was then administered intravenously in each loop. Pulse monitoring revealed persistent pulseless electrical activity. The LUCAS mechanical chest compression system was used for ten minutes before the patient regained a pulse-generating rhythm, as revealed by pulse pressure measured at the femoral artery. She received advanced cardiopulmonary resuscitation for 17 minutes in total. She remained hypotensive with systolic blood pressure below 80 mm Hg, and infusion of adrenaline 0.2 µg/kg/min was started. In addition, she received intravenous infusion of 6 000 ml of Ringer's acetate over the first two hours, followed by infusion at 300 ml/h.

During the first 24 hours, the patient received a total of 12 litres of intravenous fluids. Haemodynamic monitoring with a pulse-induced continuous cardiac output (PiCCO) catheter revealed significant fluid requirements, low cardiac output and high peripheral resistance. PiCCO measurements were used to guide further fluid therapy.

Following the return of spontaneous circulation, glucocorticoids (Solu-Cortef) 250 mg and dexchlorpheniramine 10 mg were administered intravenously. A central venous catheter and dialysis catheter were inserted. The patient began to show signs of responsiveness with the need for analgosedation, and therefore therapeutic hypothermia was not induced.

Dialysis for elimination of the contrast agent was performed for 1 hour and 20 minutes, but was hindered by cardiovascular instability and had to be discontinued owing to catheter dysfunction. Adrenaline infusion was replaced with dobutamine infusion 5 µg/kg/min to reduce peripheral resistance. The dobutamine infusion was phased out over 48 hours and the patient was extubated 46 hours after the code blue alert.

The patient had intact cerebral function, and respiratory and haemodynamic stability. Brain MRI without contrast seven days after the event showed no signs of ischaemic brain damage.

With the exception of anisocoria, the patient had no obvious sequelae. She was discharged home after 18 days.

Discussion

Gadolinium contrast rarely causes serious adverse effects (1). Acute adverse effects can be divided into two groups: non-allergic adverse effects (such as headache, exhaustion/fatigue, arthralgia, nausea, vomiting) and idiosyncratic, allergy-like reactions. The latter are classified as mild, moderate or severe. Mild reactions include urticaria, mild pruritus, and erythema, while moderate reactions include pronounced urticaria, mild bronchospasm and oedema in the face or larynx. Serious adverse effects include hypovolemic shock, and respiratory or cardiac arrest (2).

Gadolinium-based contrast agents can also cause nephrogenic systemic fibrosis, a serious and life-threatening condition that is primarily a risk in patients with severe renal failure (3). It was reported a few years ago that gadolinium can also be deposited in the brain, but the clinical significance of this remains unclear (4).

A retrospective review of 158 792 scans with gadolinium-based contrast agents registered in the U.S. Food and Drug Administration and drug manufacturers' reporting system revealed 94 acute reactions related to the contrast agent (5.9 per 10 000 injections). Of these 94 acute reactions, 4 were classified as severe (approximately 1 per 40 000 injections) based on American College of Radiology (ACR) criteria. Of the four serious reactions, three were cardiac arrests and the other was an episode of severe respiratory distress. Women were more likely to experience adverse effects, with a female-male ratio of 3.3:1 (1).

Another retrospective study of more than 84 000 patients who underwent an MRI scan with gadolinium contrast identified 112 acute hypersensitivity reactions. The incidence was higher in women and in patients with known asthma and allergies, and also increased with the number of times the patient had been exposed to a gadolinium-based contrast agent. Among the patients with reactions, the most common symptom was urticaria (91.1 %). Anaphylaxis occurred in 11 patients (9.8 %) and there was one fatality (5).

In the event of a severe reaction to a contrast agent, it is important that the patient is quickly removed from the magnet room. In a critical situation, there is a high risk that personnel unaccustomed to working with MRI will bring magnetic objects into the room with them. This poses a great danger to everyone in the room. The magnetic field is very powerful and it is always on.

Even though MRI with contrast is considered a safe procedure, serious adverse effects can occur, including fatalities. As with all other diagnostic testing, MRI should only be performed on the basis of a clear indication.

The patient has consented to the publication of the article. The article has been peer-reviewed.

LITERATURE

1. Prince MR, Zhang H, Zou Z et al. Incidence of immediate gadolinium contrast media reactions. AJR Am J Roentgenol 2011; 196: W138-43. [PubMed][CrossRef]

2. ESUR Guidelines on Contrast Agents 10.0. http://www.esur.org/esur-guidelines/ Accessed 17.3.2020.

3. Abildgaard A. MR for radiografer og radiologer. Fysikk og fysiologi. Oslo: Universitetsforlaget, 2016.

4. Gulani V, Calamante F, Shellock FG et al. Gadolinium deposition in the brain: summary of evidence and recommendations. Lancet Neurol 2017; 16: 564–70. [PubMed][CrossRef]

5. Jung JW, Kang HR, Kim MH et al. Immediate hypersensitivity reaction to gadolinium-based MR contrast media. Radiology 2012; 264: 414–22. [PubMed][CrossRef]

Publisert: 25 May 2020. Tidsskr Nor Legeforen. DOI: 10.4045/tidsskr.19.0709
Received 2.11.2019, first revision submitted 19.3.2020, accepted 15.4.2020.