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## Iodide Mumps

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### To the Editor,

Iodide Mumps, so-called in medical literature, is a rare, scarcely described adverse effect, consisting of an inflammation of the parotid, submandibular and/or sublingual glands, secondary to exposure to iodine contrasts.<sup>1</sup>

### CASE REPORT

We present a 65-year-old male with a history of long-term arterial hypertension, surgical intervention of thoracic aortic aneurysm and abdominal aortic aneurysm being monitored for vascular surgery, benign prostatic hyperplasia (BPH), right parotid swelling, stage 5 chronic kidney disease (CKD) due to likely nephroangiosclerosis in kidney replacement therapy, on peritoneal dialysis since May 2012. He was admitted to our department due to intense abdominal pain, finding an inguinal hernia on the left side. An

abdominal CT with contrast was performed which showed the hernia to be incarcerated, with decrease in pain following manual reduction. 48 hours after the radiology test the patient presented painless swelling of the right parotid and right submandibular gland (Figure 1). An ecography of the salivary glands was carried out which showed generalised swelling in all glands (Figures 2 and 3). The symptoms were self-limited, with full recovery after 6 days.

### PATHOPHYSIOLOGY

Iodide mumps are characterised by a rapid and painless growth of the salivary glands following iodine contrast administration. It can present itself several minutes after exposure and up to 5 days later. Its incidence is unknown, with few reported cases: 40 until 2012.<sup>2</sup> It was described for the first time in 1956 by Sussman and Miller.<sup>3</sup> It is believed to be caused by iodine contrast, as an idiosyncratic reaction or due to its toxic accumulation which

causes inflammation, oedema of mucous membranes and duct obstruction due to the concentration through the sodium iodide symporter of salivary gland tissue. 98% of iodine is excreted by the kidney and the remaining 2% by the salivary, sweat and lacrimal glands. As a result, CKD could be a risk factor for its development.<sup>4</sup> It can recur with successive exposures to iodine contrast.

### DIAGNOSIS AND TREATMENT

Clinical symptoms serve as a guideline and the ecography showed diffuse glandular swelling, dilated hyperechoic ducts and increase in central vascularisation. A differential diagnosis is made using entities that may put the patient's life at risk (angioedema, haemorrhage).<sup>4</sup> Treatment is conservative, supported with analgesic on demand. Corticosteroids and antihistamines have not proven effective.

### CONCLUSIONS

Iodide mumps is a rare adverse effect of iodine contrast use, although its true incidence is unknown, due to under-recording, with unpublished incidental cases in our nephrology department.<sup>5</sup> We consider that this condition deserves more attention and a suitable register for recurrence, since there are no prophylactic measures and its long term significance is uncertain.

### Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.



**Figure 1.** Swelling of the right parotid and right submandibular gland.



**Figure 2.** Ecography of normal parotid gland.



**Figura 3.** Swelling of right parotid.

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## IgM nephropathy presenting as full blown crescentic glomerulonephritis: first report in the literature

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### Dear Editor,

IgM nephropathy (IgMN) is a relatively newly described, and still contentious, clinicopathologic entity which presents mainly as idiopathic nephrotic syndrome (INS) in both children and adults. Although, it is widely acclaimed that this lesion was first described in 1978 by two groups led by Cohen et al.<sup>1</sup> and Bhasin et al.,<sup>2</sup> predominant IgM deposits in the glomeruli, in fact, were first described in renal biopsies in 1974 by Putte et al.<sup>3</sup> in patients

with persistent or recurrent hematuria. The frequency of IgMN reported in literature has varied from 2% to 18.5%.<sup>4-8</sup> We have earlier reported a prevalence of IgMN of 18.5% in native renal biopsies in children presenting with INS in Pakistan.<sup>9</sup> The disease is defined by its immunopathologic features: the light microscopic (LM) features being highly variable ranging from minimal change lesion to variable degrees of mesangial proliferation to focal segmental glomerulosclerosis (FSGS).<sup>4,5,9</sup> A few studies have also noted small subcapsular crescents in cases of IgMN.<sup>4,5</sup> However, up till now, no case of IgMN has been reported presenting with crescentic GN (CresGN). We herein report a case of an 11-year-old child presenting clinically with rapidly progressive glomerulonephritis (RPGN) and the biopsy features of CresGN.

### CASE REPORT

An 11-year-old female presented with one month history of generalized body swelling, anorexia and moderate degree of hearing loss. On physical examination, her blood pressure was 150/110mmHg. No past medical history of note was elicited. Family history was unremarkable for renal disease. Abdominal ultrasound showed normal size kidneys. Urine examination revealed 1+ albumin, 15-20 white blood cells (WBCs)/HPF and 6-8 red blood cells (RBCs)/HPF. Her blood urea was 198mg/dL and serum creatinine, 4.5mg/dL. Relevant viral serology was negative. Slit-lamp examination for eye abnormalities was negative.

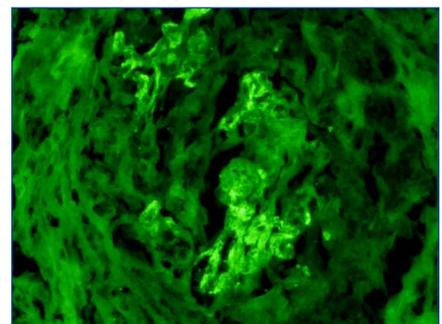
The percutaneous renal biopsy was done, which mostly comprised of medulla with little cortex containing six glomeruli. These showed diffuse severe degree of mesangial proliferation. In addition, three glomeruli showed extracapillary epithelial cell proliferation with the formation of cellular crescents (Figure 1). Mild arteriosclerosis was seen. Mild degree of tubular atrophy and

interstitial scarring was also noted. On immunofluorescence (IF), there was diffuse, bright (3+, on a scale of 0 to 3+) positivity of IgM in the mesangium and peripheral capillary loops, accompanied by C3 and C1q in similar location (Figure 2). IgG and IgA were negative. A diagnosis of IgMN was rendered and lupus serology and serum antineutrophil cytoplasmic antibodies (ANCA) were advised, which returned negative.

She was given three pulses of methylprednisolone and was started on oral steroids in the standard dosage. Her serum creatinine stabilized at 2.3mg/dL and she was discharged with advice to follow-up in the OPD. Unfortunately, she was soon lost to follow-up.



**Figure 1.** Medium-power view showing cellular crescents in all the glomeruli (Silver stain, ×200).



**Figure 2.** High-power view showing diffuse mesangial positivity of 3+ intensity of IgM in the collapsed tufts of the glomerulus on immunofluorescence (IF) microscopy. The surrounding crescent is negative (anti-IgM on IF, ×400).