C5-C6 discitis with osteophytosis leading to pharyngeal anatomical distortion and incontinence, and fever. The laboratory analysis showed acute phase reactants elevation. After an exhaustive investigation of infectious etiology and refractoriness to multiple antibiotics, the patient started high-dose corticosteroid therapy with rapid resolution of complaints. The CT image showed C4-C5 vertebrae involvement with calcification and marked thickening of the atlantoaxial ligament complex and peri odontoid soft tissues were identified, with anterior dislocation of the odontoid and stenosis of the vertebral canal at the medullary bulb transition (figure C). Clinically, she reported longstanding episodes characterized by mixed rhythm cervicalgia. She denied a previous history of peripheral arthritis.

Radiographic and ultrasound studies evidenced triangular fibrocartilage of the carpus and knee menisci chondrocalcinosis, and intracartilaginous hyperechogenic images in multiple locations, suggestive of CPP deposition. The patient started therapy with colchicine 1mg/day and cervical orthosis for symptomatic control.

4–Inflammatory discitis: 78-year-old male, hospitalized for inflammatory neck pain, wrist and metacarpophalangeal joints arthritis, dysphagia, dyspnea, urinary incontinence, and fever. The laboratory analysis showed acute phase reactants elevation, without leukocytosis. After an exhaustive investigation of infectious etiology and refractoriness to multiple antibiotics, the patient started high-dose corticosteroid therapy with rapid resolution of complaints. The CT image showed C4-C5 and C5-C6 discitis with osteophytosis leading to pharyngeal anatomical distortion and periodontal and posterior longitudinal ligament calcification (figure D).

Figure 1.

Conclusion: The axial involvement of CPPD can have several clinical presentations, from asymptomatic to acute presentation with manifestations of other organ systems. The imaging findings of axial involvement are distinctive and can be crucial in the diagnosis. The differential diagnosis with other etiologies is situations of acute presentations, namely infectious, is imperative. Recognition of this potential clinical manifestation is essential to avoid underdiagnosis.

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AB1860 CHARACTERISTICS OF IMMUNE CHECKPOINT INHIBITOR INDUCED SICCA SYNDROME

Keywords: Sjögren syndrome, Real-world evidence

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Background: Immune checkpoint inhibitors (ICIs) have revolutionised management of certain cancers and significantly improved mortality. They work by blocking specific checkpoint proteins or ligands: CTLA-4 (ipilimumab), PD-1 (pembrolizumab and nivolumab) and PD-L1 (durvalumab and atezolizumab). This augments anti-tumour immunity through improved T cell function. Unfortunately the augmented immune function can lead to immune related adverse events (irAEs). One of these irAEs is sicca syndrome, which is a discrete entity from Sjögren’s syndrome and was first reported by Capelli in 2017[1].

Objectives: To report on a supra-regional cohort of patients with immune checkpoint inhibitor induced sicca syndrome, exploring clinical and pathological features.

Methods: We evaluated data from patients who were referred with new onset sicca symptoms to Queen Elizabeth Hospital Birmingham’s Sjögren’s service, between 2017 and 2022.

Results: 22 patients were identified (male =12, median age = 66) with underlying cancers (metastatic melanoma =16, renal cell carcinoma = 4, small cell lung cancer =1, hepatocellular carcinoma =1) who were being treated with various ICIs (atezolizumab =2, durvalumab =3, pembrolizumab = 5, nivolumab and ipilimumab combination = 11 and monotherapy nivolumab =1). 4 patients had pre-existing autoimmune disease (autoimmune thyroiditis =2, coeliac disease =1, type 1 diabetes mellitus =1). 21 patients reported xerostomia, with 15 of the 16 patients who performed salivary flow tests having very low unstimulated flow rates. Xerophthalmia was reported by 10 patients; interestingly 4 patients did not report dry eye symptoms but had significantly reduced tear production on Schirmer’s testing. Glandular swelling was only seen in 2 patients. ANA was positive in 3 patients, none had a positive ENA. One patient was rheumatoid factor positive (38.8 IU/ml). 4 had IgG levels >16G/l. No cases of hypocomplementaemia were seen. The median time to symptom onset, after ICI initiation, was 3 months, with varied intensity of onset. The mean oral dryness score, on initial assessment, was 6.5 out of 10 (SD ±2.5). ICI Therapy was stopped in 5 patients due to severity of symptoms. 3 required temporary discontinuation, due to sicca syndrome with an average pause of 9.3 weeks. 13 of the 22 patients had a history of other irAEs, most commonly arthritis, colitis and endocrine issues (new onset diabetes, hypothyroidism, hypercalcaemia). Systemic steroids were required in 12 patients to alleviate their symptoms (starting doses 10-30mg), and 6 of these subsequently started hydroxychloroquine. Mean oral dryness score, following treatment (ICI cessation or aforementioned treatments) had reduced by over half, to a mean of 3.1 (SD ±2.9) at last clinical assessments. Labial salivary gland biopsy was performed in 15 patients; 4 showed focal lymphocytic sialadenitis (FLS; a biopsy finding also associated with Sjogren’s syndrome). None of the patients with FLS had autoimmune disease or dryness symptoms predating ICI. The other 12 samples showed nonspecific chronic sialadenitis, with characteristic atrophy and fibrotic changes. Immunohistochemistry typically showed lightly dispersed CD3+ T cells with an even mixture of CD4+ and CD8+ cells.

Conclusion: We have presented the clinicopathological features of a supra-regional ICI induced sicca syndrome cohort. Our findings mirror those from the US cohort described by Warner et al[2] with significant oral symptoms which occur soon after treatment initiation and may respond to withholding or stopping ICI, steroids and hydroxychloroquine. ICI induced sicca syndrome significantly impacts quality of life and should be recognised as an irAE that in certain cases requires treatment.

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