

## Clinical Reports

# Submandibular chronic sialadenitis presenting with alcohol-induced pain

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**Summary:** A 32 year old man with alcohol-induced pain over a right submandibular swelling is described. Excision biopsy of this swelling revealed chronic sialadenitis and the symptoms promptly ceased following this excision. We speculate on the possible pathophysiological mechanism.

### Introduction

The association between alcohol-induced pain and Hodgkin's disease was first noted by Hoster in 1950.<sup>1</sup> Since then several other authors<sup>2–6</sup> have also reported similar findings, so much so that alcohol-induced pain is now commonly listed in textbooks as a hallmark of Hodgkin's disease, albeit an uncommon one. Apart from Hodgkin's disease, alcohol-induced pain has also been reported less frequently in other neoplastic diseases like carcinomas of the pancreas,<sup>5,7</sup> bronchus,<sup>5,8</sup> cervix, bladder, nasopharynx, breast and also in non-Hodgkin's lymphoma.<sup>5,6</sup> It has also been reported in a malignant intracranial chordoma<sup>9</sup> and in non-neoplastic conditions like fractured hip bone, acute pyogenic lymphangitis,<sup>4</sup> Brodie's abscess and pyogenic osteomyelitis.<sup>10</sup> We hereby report a case of alcohol-induced pain seen in a patient with chronic sialadenitis of the submandibular gland.

### Case report

A 32 year old businessman was referred with a 3-month history of intermittent neck pain and a 3-week history of a right submandibular swelling. Pain was aching in nature but progressively got more intense and it took the patient some time to realize that the pain usually came on a few minutes after he had drunk some beer. He drank lager beer frequently and was in the habit of taking about 2 or 4 pints at a time. Initially he got some relief from the pain by taking aspirin tablets. About 3 weeks before the patient presented to us he noticed a swelling at the right submandibular area, where the

pain was usually maximal. This swelling gradually increased in size and the intensity of the pain worsened. The patient then reported to his family clinic from where he was referred to us as possibly a case of Hodgkin's disease with an alcohol-induced pain.

He was seen by us during one of his bouts of pain which started about an hour after he drank 2 pints of beer. The pain was so intense that the patient actually rolled on the floor and needed pethidine 100 mg intramuscularly to control. Apart from the firm, slightly tender and non-mobile lump at the right submandibular region measuring about 2 cm by 3 cm, there were no other physical findings. The patient was admitted for excision biopsy. The histopathological diagnosis was chronic sialadenitis of the submandibular salivary gland with no evidence of malignancy. We have since followed him up for 2 years and there has been no recurrence of the neck pain even though he has resumed his beer drinking habits.

### Discussion

Alcohol-induced pain had been previously thought to occur almost exclusively in Hodgkin's disease. However, the finding of this symptom in a variety of diseases, some of which are not neoplastic, indicates that it is not specific for Hodgkin's disease. The mechanism of pain following alcohol ingestion is not known although oedema,<sup>1</sup> alcohol-induced vasodilation,<sup>2,4</sup> and accumulation of acetaldehyde as a result of tumour production of alcohol dehydrogenase in the case of tumours,<sup>11</sup> have been variously suggested as possible mechanisms. It is quite conceivable that our patient's symptoms could be similar to those of patients with

alcohol-associated pancreatitis. Both the pancreas and the salivary glands are exocrine glands with certain morphological similarities. In the case of alcohol-associated pancreatitis, repeated episodes of acute pancreatitis result ultimately in chronic pancreatitis with variable acute exacerbation fol-

lowing each ingestion of alcohol.<sup>12</sup> In a small percentage of these patients only minimal amounts of alcohol are known to trigger off severe pancreatic pain.<sup>13</sup> We speculate that this same pathophysiological mechanism might be responsible for the symptoms in our patient.

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