PET-FDG response in alcohol induced chest pain with Hodgkin lymphoma

*Corresponding Author: James S Veenstra
Email: James.Veenstra@AlbertaHealthServices.ca

Abstract

We present a case of alcohol induced pain in the setting of a mediastinal mass with significant lymphadenopathy. A diagnosis of Hodgkin lymphoma (HL) was made. The alcohol induced pain resolved, correlating with post-chemotherapy treatment response on positron emission tomography/computed tomography (PET/CT) imaging. The incidences and possible causes of alcohol induced pain are reviewed. The use of PET/CT imaging is discussed in relation to decision making for management of lymphoma.

Introduction

Lymphoma is a group of lymphoid malignancies that develop from lymphocytes with two main types: Hodgkin lymphoma (HL) and non-Hodgkin lymphoma. Lymphoma may present with lymphadenopathy and classic systemic symptoms of fever, night sweats and weight loss. Other less common symptoms can include fatigue, dyspnea, anorexia, pruritus and pain with alcohol consumption. Less common symptoms can make it challenging to determine appropriate investigations and achieve a timely diagnosis.

Case report

A 32-year-old male developed left sided central chest pain that started minutes after ingesting small amounts of alcohol that would resolve over a short period of time or with continued alcohol consumption. The pain progressively worsened over six months’ time and was absent with consumption of any other solids or liquids. However, in a six-week time period a new palpable firm two centimeter lesion developed between the left anterior second and third ribs.

He reported mild fatigue, but denied any fevers, night sweats or weight loss. His medical history was significant for Neurofibromatosis type 1. He was a never smoker and consumed daily light amounts of alcohol. Initial testing included a normal barium swallow and chest x-ray showing an anterior mediastinal mass (Figure 1). Computed tomography of the chest showed a bulky anterior mediastinal mass (10.3x10.3x14.5 cm) with sternal/chest wall invasion and mediastinal lymphadenopathy. Positron emission tomography/computed tomography (PET/CT) showed very non-homogeneous fluorodeoxyglucose (FDG) uptake internally with several focal areas of very intense uptake (Figure 2). There was marked metabolic activity within mediastinal, hilar, bilateral retro-pertoral, left supraclavicular and right axillary lymph nodes with increased metabolic activity in an area of manubrial lytic change. Complete blood count, electrolytes, urea, creatinine, liver enzymes and lactate dehydrogenase were normal. Erythrocyte sedimentation rate was elevated at 23 mm/hr. Viral hepatitis and HIV testing was negative.
Core biopsy (16 gauge) of the mediastinal mass showed polymorphous cellular infiltrate consisting of numerous small mature lymphocytes, scattered histiocytes, eosinophils and occasional large atypical cells consistent with variants of Hodgkin and Reed-Sternberg (HRS) cells. Immunohistochemical stains showed HRS cells positive for CD30, CD15 (small subset), MUM1 and PAX5 (weak); and negative for CD45, CD20 and October 2. CD3 highlighted abundant small mature T-cells in the background. An in-situ hybridization of EBV RNA (EBER) was negative within HRS cells. The final diagnosis was Stage IVAEX Classical HL nodular sclerosis subtype.

Treatment was with six cycles of Adriamycin, bleomycin, vinblastine and dacarbazine (ABVD). Chest pain with alcohol consumption and the mass between the ribs resolved after the first treatment. Repeat PET/CT scan after the second ABVD cycle showed considerable decrease in FDG avidity and size of the mass measuring 3.7x2.5 cm. Only a few foci of FDG uptake persisted of similar intensity to background liver (Deauville category 3). Tumor extension into the left parasternal chest wall, supraclavicular, axillary and mediastinal lymphadenopathy had resolved. The manubrial abnormality had become more sclerotic and the FDG uptake had resolved, consistent with a healing response. Bleomycin was removed for the remainder of treatment for long term risk reduction [1]. Consolidative radiation was given (3000 cGy in 15 fractions) due to residual FDG foci in the mass. Repeat PET/CT imaging shows complete metabolic response to treatment (Figure 3). The patient’s disease remains in remission four years after finishing treatment.

Comment

The incidence of alcohol related pain with HL was initially reported in 1950 [2] with an incidence of 1.5-5% of HL cases. The differential diagnosis of alcohol-related pain or intolerance includes HL, carcinoid syndrome, alcohol dehydrogenase 2 mutations, other solid tumor malignancies and disulfiram reactions. Alcohol related pain seems to be more common with HL nodular sclerotic subtype, affecting areas locally involved with HL. Incidences of occurrence range from 7% patients with lymphadenopathy to 20% with bone involvement [3]. Treatment of HL commonly results in resolution of alcohol related pain [4]. The causes of alcohol-related pain in HL are unknown and are hypothesized to be related to vasodilation within the lymph node causing capsular stretch or to acetic acid production in the tissues affected by HL [5,6]. PET/CT is a standard for evaluation, staging and response assessment in lymphoma [7]. Early interim PET-FDG uptake is a negative independent predictor of progression free survival and overall survival [8]. At this time there is no strong evidence to support poorer outcomes for patients with HL and alcohol related pain. In conclusion, lymphoma should
be a diagnostic consideration with symptoms of alcohol related pain. Resolution of alcohol related pain indicates treatment response which can be correlated with radiological imaging. PET/CT imaging is useful in lymphoma treatment planning and in determining treatment response.

References


