Low HbA1c levels in a poorly controlled diabetic

Q1: What is the differential diagnosis and what would you do next?

If a laboratory error can be ruled out (repeated samples need to be obtained), the main differential diagnosis is of an abnormal haemoglobin variant. Some causes of abnormal HbA1c values are listed in Box 1. The next step is to perform haemoglobin electrophoresis. The following results were obtained in this patient:

- Alkali/acid elution: haemoglobin A + J (confirms the presence of abnormal haemoglobin J).
- Globin fast beta chain variant (abnormal beta chain).
- Isoelectric focusing: haemoglobin A + J.
- Abnormal haemoglobin: 48% (a high percentage of haemoglobin J).
- Haemoglobin A2: 2.58% (normal <3%).
- Sickledex: negative (no evidence of sickle cells).
- Haptoglob: 0.28 g/l (normal 0.7–3.19).

These tests are consistent with a diagnosis of haemoglobin beta chain variant. J trait.

Q3: What is the pathophysiological basis of the discrepancies observed and how would you assess this man’s long-term glycaemic control?

Non-enzymatic binding of glucose to the valine residue of the beta chain of the haemoglobin molecule gives rise to glycated haemoglobin (HbA1a, HbA1b, and HbA1c). The level of HbA1c reflects ambient blood sugar concentrations during the life span of the patient’s red cells (half-life about 6–8 weeks)—that is, uncontrolled hyperglycaemia results in high HbA1c levels. Current guidelines recommend HbA1c levels of less than 7% as a target for satisfactory control. HbA1c can be measured chemically, chromatographically, and electrophoretically. Most autoanalysers use the chromatographic method. Haemoglobin variants may affect chromatographically measured HbA1c levels either by inducing an abnormal peak and thereby making the estimation of the fraction of HbA1c unrelatable, or by reducing time available for glycation as a result of reduced red cell survival. These two effects may coexist. Physicians should be aware of the potential pitfalls of HbA1c as a measure of long term diabetic control. Apparent discrepancies between glycaemic control reflected in day to day blood glucose concentrations, and HbA1c values should be noted. HbA1c levels are inappropriately affected by factors other than long term glycaemia as shown in boxes 1 and 2. Schenld et al reported a prevalence of abnormal haemoglobin variants of 0.6% among 15 000 HbA1c estimations in a period of over six years. In such individuals a method unaffected by abnormal haemoglobin variants, such as a turbidimetric inhibition immunoassay or alternate methods of chromatography should be used.

REFERENCES


A bed bound patient

Q1: What is the differential diagnosis and the most likely diagnosis?

The differential diagnosis is wide (Box 1, which is not exhaustive) and illustrated somewhat by the past medical history. However, the most likely diagnosis is polymyositis. The presentation with proximal muscle tenderness, and importantly weakness, together with a raised ESR and raised creatine kinase is typical. The clinical picture may evolve over several weeks or months, as in this case.

Had the appropriate skin manifestations been present, dermatomyositis would have been an important consideration. Dermatomyositis is easily recognised and diagnosed because of the characteristic rash that may either accompany or precede the onset of muscle weakness. Classically there is a purplish discoloration of the eyelids (heliotrope rash) often associated with periorbital oedema and papular, erythematous, scaly lesions over the knuckles (Gottron’s sign). In addition, a flat, erythematous, sun sensitive rash may appear on the face, neck, and anterior chest (V sign), on the shoulders and upper back (shawl sign), and on the elbows, face, and malleoli. The nail beds often have dilated capillary loops and calcifications may be found in the subcutaneous tissues, although this is much less common in adults than children with this condition.

Inclusion body myositis is characterised clinically by the insidious onset of slowly progressive weakness. The slow evolution of symptoms contributes to the delay in diagnosis, which averages six years. Distinct from polymyositis and dermatomyositis, males are much more commonly affected than females and the clinical hallmark is early weakness of the quadriceps, wrist and finger flexors, and the ankle dorsiflexors. Irreversibly, the manual muscle scores of the finger and wrist flexors are lower than those of the shoulder and pelvic flexors. Typically the serum creatine kinase is normal or only mildly raised. The high ESR and the patient’s age in combination with the marked muscle tenderness may point to polymyalgia rheumatica. However, although this condition is characterised by pains and early morning stiffness in the proximal muscles of the shoulder and pelvic girdle, the hands and feet are not affected and there is no muscle weakness. There are also usually some systemic features of a low grade fever or malaise. It is three times more common in women than men.

A final diagnosis: Abnormal haemoglobin variant.
common in women than in men and usually occurs between the ages of 60–70 years. Chronic inflammatory demyelinating polyneuropathy (CIDP) is an immune-mediated neuropathy characterised by a relapsing or progressive course. By definition, symptoms and signs of the neuropathy must be present for at least two months, which distinguishes CIDP from Guillain–Barré syndrome. With a peak incidence in adults of 40–60 years of age, the majority of patients present with symmetric proximal and distal weakness of the arms and legs. Importantly, at least 80% of patients have both motor and sensory involvement, although one may predominate. Although the hyporeflexia seen in this case is compatible with CIDP, muscle tenderness would not be expected and the ESR and creatinine kinase would typically not be raised. A raised cerebrospinal fluid protein is found in 80%–95% of patients, antibodies directed against myelin proteins are present in a small percentage of patients, and as many as 25% have an IgA, IgG, or IgM monoclonal gammapathy.

This patient’s history of type II diabetes mellitus makes one consider the possibility of a diabetic amyotrophy (also known as diabetic lumbosacral radiculoplexopathy), especially given the pain and the weakness of her quadriceps muscles and the absent knee reflexes. However, this is an arterioles disease, as it does not affect the upper limbs. Most patients have non-insulin-dependent diabetes mellitus and although commonly associated with periods of poor glycemic control, the development of this neuropathy is often unrelated to glucose control or the duration of glucose intolerance. In this patient glycemic control had been improved by a diet and by a glycated haemoglobin (HbA1c) of 6.5%. In diabetic amyotrophy, the neuropathy begins with severe pain in the back, hip or thigh, the pain typically characterized as burning, knife-like, and ach.

On examination there is weakness of hip flexors and extensors, knee flexors and extensors, and ankle dorsiflexors and plantar flexors of varying degree. Profound atrophy of both thigh and at times distal lower extremity muscles develops.

There are many other metabolic and endocrine causes of a proximal myopathy including hyperthyroidism, myxoedema, Cushing’s syndrome, acromegaly, osteomalacia, Addison’s disease, and primary hyperaldosteronism. Those seen in metabolic disease, myxoedema, Cushing’s and Addison’s may all be associated with muscle pain at rest and both myxoedema and acromegaly may sometimes result in an increased creatine kinase. In this patient the hormone profile was normal.

A myasthenic syndrome must always be considered in any case of muscle weakness. It may also cause a mainly proximal muscle weakness, but the lack of demonstrable fatigability and the presence of muscle pain would be atypical. The raised creatine kinase in this patient, is also against the diagnosis.

Q2: How would you confirm the diagnosis?

The diagnosis of polymyositis would be considered on two main aspects: drug response and biopsy. Electromyography is usually abnormal in polymyositis with the following features: increased insertional and spontaneous activity with fibrillation potentials, positive sharp waves, and occasionally pseudomyotonic and complex repetitive discharges; small duration, low amplitude, polyphasic muscle unit poten-

Q4: How would you manage this patient?

The management of polymyositis can be considered from the following aspects: drug treatment of the disease, general supportive measures, and further investigation of the patient. The mainstay of drug treatment remains corticosteroids especially in controlling the early clinical course. Over 80% of patients at least partially improve, while 10%–33% completely respond to prednisolone. Treatment with steroids is usually initiated with prednisolone 0.5 to 2.0 mg/kg per day. Normalisation of muscle enzymes usually occurs within four weeks and the corticosteroid is tapered over one to three months. Once there has been a full response to corticosteroids the patient should be considered for a course of treatment in responders. Improvement in muscle strength occurs later, usually within 3–6 months. Once there has been a full response to corticosteroids the dose of corticosteroid may be gradually reduced (for example, by 5 mg every two weeks). During the period of dose reduction the patient should be monitored closely for evidence of relapse of disease.

Patients who do not respond to corticosteroids could be considered for other treatment modalities such as intravenous immune globulin, which has been shown to be an effective therapy for drug resistant dermatomyositis and polymyositis.

Methotrexate and azathioprine should also be considered in patients who do not respond well to steroids, patients at risk of steroid side effects and those with poor prognostic indicators such as dysphagia, disease duration of three months before treatment, and profound weakness in some patients. Patients at risk of malignancy and in patients with a relapse of malignancy, the temporal relationship being unclear. In some patients, pre-existing myositis recur in patients, in a few days or weeks of diagnosis of malignancy and in patients with a relapse of malignancy, myositis has occurred de novo. Dermatomyositis is associated with underlying malignancy especially gastric and lung in men and ovarian and cervical in women. Polymyositis is also associated with a higher incidence of underlying malignancy at the time of diagnosis, and a lesser extent. In a large Swedish population based study the incidence of cancer in patients with dermatomyositis and polymyositis was compared to that in the general Swedish population. In the 392 patients with dermatomyositis the incidence of cancer was 15% (relative risk 2.4 in men and 3.4 in women). In the 396 patients with polymyositis the incidence of cancer was 9% (relative risk 1.7 in men and 1.8 in women).

In addition to the association with malignancy, myositis with secondary congestive heart failure or conduction abnormalities occur in up to one third of patients with polymyositis. Polyrheitis has been reported in as many as 45% of polymyositis patients at the time of diagnosis. Interstitial lung disease occurs in approximately 10% of polymyositis patients, at least half of whom have Jo-1 antibodies. These are antinucleas associated with a more modest response to treatment and poorer prognosis and which are found in around 20% of patients with polymyositis overall. Other antibodies seen in polymyositis include antinuclear antibodies, seen in 16%–40% of cases, and antibodies against the signal recognition particle, found in 4% of cases and associated with an acute onset of severe weakness, myalgias, and myocarditis.

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mainly composed of CD4+ T cells, B cells, and other immune cells, as compared to the endomysium, and the infiltrate is predominantly perivascular and located in the perimysium rather than the endomysium. Dysphagia occurs in ~1/3 of patients due to oropharyngeal/oesophageal involvement. Jo-1 antibodies occur in ~20% of cases and are associated with a poorer prognosis. Risk of malignancy with polymyositis is lower than with dermatomyositis but higher than in the general population. Inflammation in polymyositis is endomy- sial and involves non-necrotic fibres whereas in dermatomyositis it is perivascular with no involvement of non-necrotic fibres. Majority of patients respond favourably to immunosuppressive therapies but usually require lifelong treatment.

Needle biopsy from quadriceps indicates muscle fibre necrosis and loss together with a modest inflammatory cell infiltrate.

On initial assessment by occupational therapy and physiotherapy, the patient required the assistance of two persons to stand. The next 10 weeks, despite receiving intensive physiotherapy, and a concurrent substantial reduction in ESR (35 mm/hour), there was no improvement in the patient's clinical condition. It was decided that she was refractory to steroid treatment and she was started on a course of intravenous immune globulin at a dose of 0.4 g/kg over five days. She was also started on azathioprine and after eight weeks of azathioprine treatment in combination with steroids she was mobilising with a walking frame under supervision. Her full blood profile and liver function tests were monitored weekly and regular creatine kinase measurements were made to exclude relapse. Serum creatine kinase can be useful in monitoring response to therapy but only in conjunction with the physical examination. The creatine kinase was raised in patients with normal manual muscle testing, while weak patients can have normal levels. Overall, this case emphasises the broad differential diagnosis of a painful proximal condition. It is related to the degenerative facet joint. It might be a very common cause of refractory low back pain with radicular pain; it is very rarely bilateral. It can present, although very rarely, acutely as an emergency and there have been reports of cervical cord compression and cauda equina syndrome after a bleed into this cyst. Elderly patients with low back pain and radiation with a leading symptom of aggravation of pain on standing and walking should be suspected.

Lumbar facet synovial cyst

Q1: What is a lumbar facet synovial cyst?

Lumbar facet synovial cyst was first described in 1968 by Kao et al. It is now being commonly reported with advanced neuroimaging techniques. It can pose serious diagnostic and therapeutic problems. The pathogenesis of this condition is degenerative including osteoarthritis, rheumatoid arthritis, and spondyloarthropathy.

Q2: How is it diagnosed?

Blood tests and radiographs are usually unhelpful. MRI is the investigation of choice. The differential diagnosis with MRI could be a schwannoma, and a extradural space occupying lesion. Pathologists divide these cysts into synovial and ganglion types but they do not have any prognostic significance.

Q3: How is lumbar facet synovial cyst treated?

It is treated only if it is symptomatic. If it is an incidental finding, analgesia can be given. Spontaneous disappearance has been reported in 10% of these cysts. Aspiration has led to recurrence and steroid injection into the cyst has been reported to increase the severity of pain. Surgery is the treatment of choice with excision of the cyst and associated lami- nectomy. If there is associated instability then fusion is the treatment of choice.

References

A misleading swelling of the tongue

Q1: What is the differential diagnosis for this lesion?

A possibility of bacterial, fungal, and viral infections should be borne in mind when establishing a differential diagnosis. Tuberculosis, syphilis, histoplasmosis, and actinomycosis are some of the infections which may produce a sarcoidal type of tissue response or granulomas. Other possible entities are foreign body granuloma and orofacial granulomatosis as well as oral Cohn’s disease, granulomatous cheilitis, and Melkerson’s syndrome. In this patient our clinical diagnosis was minor salivary gland tumour given the site of occurrence, and hard nodular swelling with overlying normal mucosa.

Q2: How will you establish a definitive diagnosis and suggest the investigations necessary for the same?

Clinical features along with histological evidence from tissue biopsy can be supplemented by chest radiography, the presence of tuberculin anergy, a positive Kviem-Siltzbach skin test, a raised serum angiotensin converting enzyme, and by an increased 24 hour urine calcium level. The characteristic histological picture of sarcoidosis in all involved tissues is the non-casing granuloma composed of aggregates of epithelioid cells, scattered multinucleated giant cells, lymphocytes, plasma cells, and fibroblasts. Two other features often seen in the granuloma are lanced concretions composed of calcium and proteins, known as “sarcoid bodies”. Although these two features are characteristic, they are not pathognomic.

Q3: Discuss the prognosis and treatment of this lesion?

Overall, the prognosis of sarcoidosis is good. The drugs of choice are glucocorticoids as they suppress the activated T helper-inducer cell processes occurring at the sites of the disease. Some advocate only surgical excision of the lesion, some medical treatment, while others combine the two modalities. Radiation also has been used as a mode of treatment, while spontaneous healing was documented in a few cases. Our patient responded well to the surgical treatment.

Discussion

Sarcoidosis is a disease that has highest occurrence in the third and fourth decades of life and exhibits a slight female predominance (1:5:1) [3]. In a young adult with constitutional complaints, respiratory symptoms, erythema nodosum, blurred vision, and presence of bilateral hilar lymphadenopathy the epitheloid is almost always sarcoidosis [4]. None of these features were seen in the present case. The most common cervicofacial manifestation, excluding ocular and lacrimal gland involvement, appears to be asymptomatic swelling of the parotid gland or cervical nodes [5]. Intraoral presentation is uncommon, and in most cases systemic sarcoidosis has been diagnosed before the oral manifestation becomes apparent [6]. One study reported the presence of non-casing granulomas in 38%–58% of biopsies of normal appearing oral mucosa of patients with known sarcoidosis [7]. Very few cases of tongue involvement have been reported, [8,9,10] Bilateral hilar adenopathy is the hallmark of this disease and is also seen in lymphoma, tuberculosis, coccidiomycosis, and trichinellosis, and bronchogenic carcinoma [11]. The presence of skin anergy is typical but not diagnostic [12]. The Kviem-Siltzbach skin test yields sarcoidosis-like lesions in 70%–80% of patients, with fewer than 5% false positive results [13]. Angiotensin converting enzyme is raised in the serum in approximately two thirds of patients but is also seen in asbestosis, silicosis, berylliosis, fungal infection, granulomatous hepatitis, hypersensitivity, pneumonitis, leprosy, lymphoma, and tuberculosis. A raised 24 hour urine calcium level is consistent with the diagnosis but is again not specific.

Final diagnosis

Primary intraoral sarcoidosis.

Learning points

- Primary intraoral sarcoidosis is of rare occurrence.
- Wide excision is essential as well as therapeutic.
- In patients presenting with smooth submucosal swellings of tongue a possibility of granulomatous lesions and infections should always be considered.

References


An unusual cause of a discharging sinus

Q1: What is the diagnosis? Sarcoidosis cutis

Calcinosis cutis is characterized by the deposition of calcium salts in the subcutaneous tissues of the body. Metastatic calcifications can occur in the body in hyperparathyroidism and end stage renal disease. Calcifications can also occur in a variety of other clinical settings. The lesion can present as a mass and is amenable to FNAC [1]. In cytological preparations, deposits of calcium salts can be both amorphous and refractile on Diff-Quik and Papanicolaou stain. However, the material may not be birefringent with these stains. Alizarin red S stain for calcium permits demonstration of the characteristic birefringence.

A group of extremely small bacteria capable of precipitating calcium salts implicated in the pathogenesis of urinary calculi and calcific atherosclerosis have been identified as the nanobacteria. The pathogenesis of calcinosis cutis and its significance in conjunction with a variety of unrelated scarring and pre-existing cutaneous entities are incompletely understood. In a series of cases, including basal cell carcinoma with dystrophic calcification, subepidermal calcified nodule, pilo-calcific, and trichodysplasia. Ultrastructurally examined the presence of Nanobacteria sp. All cases were negative for nanobacteria. Dystrophic calcification that occurs in conjunction with these entities does not likely involve a bacterial-induced aetiology. The cause of these entities remains unknown.

Subepidermal calcified nodule is a form of calcinosis cutis that most commonly occurs in children. The typical clinical presentation is of a solitary verrucous nodule on the face, but occasionally multiple lesions may occur.

Smears usually show amorphous granular material consistent with calcium, and occasional histiocytes. The presence of amorphous calcium salts along with histiocytes in the appropriate clinical setting is diagnostic of calcinosis cutis. The diagnosis can be confirmed on histology.

Although this benign lesion can be suspected on radiological and clinical examination, FNAC and biopsy should always be done to rule out calcification in benign and/ or malignant conditions. Metastasis of this disorder has also been reported, though it is still benign.

Localised lesions can be injected with intralesional corticosteroids that may be beneficial due to the anti-inflammatory effect and inhibitory effect on fibroblast activity. Probenecid and colchicine have been beneficial in some individuals. In patients with hyperphosphataemia, magnesium or aluminium antacids may be effective due to phosphate binding ability. However, use in patients with renal insufficiency may result in magnesium or aluminium toxicity. Sodium thiosulphate and diphosphonates may reduce bone turnover and inhibit the growth of ectopic hydroxyapatite crystals. However, prolonged treatment is necessary, and paradoxical hyperphosphataemia may result. There have been variably beneficial effects with the use of the calcium channel blocker diltiazem. The therapeutic effect of this is believed to be the antagonism of the calcium-sodium ion pump. Surgical excision can be undertaken when there is pain, recurrent infection, ulceration, or functional impairment, though surgical trauma can stimulate calcification and recurrence is not uncommon.
Q2: What other features can be associated with the present clinicoradiological picture? Raynaud's phenomenon, oesophageal dysmotility, sclerodactyly, and telangiectasia combine with calcinosis to form the CREST syndrome.

Q3: Which immunological test is positive in a majority of patients with this clinical condition? Anticentromere antibodies are seen in a very high proportion of patients with CREST syndrome.

Final diagnosis Calcinosis cutis.

References

Learning points
- Not every discharging sinus is due to infection or malignancy.
- Not all that appears radio-opaque on radiographs is solid.
- Examination of hands can provide crucial diagnostic clues even for the lesions in the lower extremity.
- Calcinosis cutis, although more commonly associated with the CREST syndrome, can present as an isolated lesion.
- As in this case, calcinosis can be present without any renal or other systemic disease.
Lumbar facet synovial cyst

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