# SELF ASSESSMENT ANSWERS

## Recurrent syncope

### Q1: What does his ECG show?

His admission ECG (fig 1 in questions) shows sinus rhythm, a major prolongation of the QTc interval (660 ms in S2), and a 2/1 atrioventricular block.

### Q2: What diagnosis would you consider?

The diagnosis is long QT syndrome (LQTS) complicated by 2/1 atrioventricular block.

### Q3: What investigations would you consider?

The differential diagnosis includes congenital and acquired long QT syndrome. Information about medication taken is critical for the diagnosis of drug induced QT prolongation. QT prolongation in the course of other diseases (electrolyte disturbances, myocardial dysfunction, neurological conditions…) should also be excluded by history, physical examination, laboratory and imaging studies (blood analysis, echocardiography…). In this case, no acquired causes of QT prolongation was shown or suspected. A family history of sudden death at a young age, a family or personal history of deafness, and QT prolongation on the ECG of the patient' relatives commonly lead to the proper diagnosis of congenital LQTS. Genetic testing for known mutations in DNA samples from studied patients is becoming more accessible in specialised centres. Identification of an LQTS gene mutation confirms the diagnosis; however, a negative result on genetic testing is of limited diagnostic value because many patients with familial LQTS have mutations of yet unknown genes.

### Q4: How would you treat this patient?

β Blocker is the present treatment of choice for congenital LQTS. The impaired conduction is classically functional and because of sinus intervals shorter than ventricular refractoriness. If the block is functional in nature, it will disappear after QT interval shortening under β block. In this case, treatment with oral nadolol (50 mg/m$^2$/day) and temporary pacing was begun, with no recurrence of syncopal events. The 2/1 atrioventricular block persisted despite QTc interval shortening (460 ms in S2) (fig 1). β Blocker treatment was continued and permanent transvenous ventricular pacing was established, with a lowest rate setting of 90 beat/min. At one year follow up, the child remains symptom free.

### Discussion

Congenital LQTS is an inherited disorder characterised by a prolongation of the QT interval on the surface ECG. This cardiac disease is potentially lethal because of polymorphic ventricular tachyarrhythmias leading to recurrent syncope or sudden cardiac death.

LQTS is occasionally complicated by a 2/1 atrioventricular block.[1] This impaired conduction is classically functional and because of sinus intervals shorter than ventricular refractoriness. However, investigations in some cases have also reported abnormal His-Purkinje system refractoriness and histological abnormalities within the conduction system.[1] Such abnormalities may be responsible for the persistence of the atrioventricular block in this case.

LQTS with conduction disorders is rare and has a very poor prognosis despite different treatment modes.[2] This form usually manifests during early life. Most of the cases are sporadic, and de novo mutations[1] or homozygous expression of mutations in several genes coding for cardiac ion channels[3] were suggested.

### References


1 **Gorgels AP**, Al Fadley F, Zaman L, *et al*. The long QT syndrome with impaired atrioventricular conduction: a malignant variant in infants. *J Cardiovasc Electrophysiol* 1998;**9**:1225–32.
2 **Trippel DL**, Parsons MK, Gillette PC. Infants with long QT syndrome and 2:1 atrioventricular block. *Am Heart J* 1995;**130**:1130–4.
3 **Hoorntje T**, Alders M, van Tintelen P, *et al*. Homozygous premature truncation of the HERG protein. *Circulation* 1999;**100**:1264–7.


## An unusual cause of inguinal pain

### Q1: What is the diagnosis?

The diagnosis is osteitis pubis. However, a differential diagnosis of hernia had to be considered because of increase in pain on cough and strain. Multiple myeloma and secondaries were also kept in differential diagnosis on account of age and peculiar site of pain. Avascular necrosis, stress fractures, and bursitis were also kept in the differential diagnosis.

### Q2: What are the diagnostic modalities that are helpful in diagnosis?

Magnetic resonance imaging and technetium-99m MDP bone scan are the most helpful diagnostic modalities in the early phase of disability. However, the most characteristic changes seen are in radiographs. These changes appear two to three weeks after onset of symptoms. Progressive radiographs show resorption of the medial end of the pubic bone, widening of the pubic symphysis, and rarefaction along pubic rami.[1]

### Q3: What is the aetiopathogenesis in this case?

Disease may in some way be related to partial separation of sites of attachment of adductor tendons. This is suggested by complete relief obtained by local anaesthetisation and repeated needling of the area. Thus after transurethral prostatectomy, osteitis pubis may in some way be related to trauma to the adductor tendon attachment during patient positioning and procedure. It may also be related to subclinical or overt infection causing subacute osteomyelites locally. The consistent relation of symptoms with decreasing haemoglobin and rising leucocytosis suggests an inflammatory/infective origin.

### (4) What is the treatment recommended for this condition?

It is a self limiting condition. Treatment is rest and non-steroidal anti-inflammatory drugs. If pain continues, repeated needling of the area may be helpful. Rarely, operative resection of symphysis may be helpful after conservative trial for six months.[2]

### Discussion

Osteitis pubis as a complication of gynaecological and urological surgery has been reported as a rare complication,[3] but this morbidity as a sequel of transurethral prostatectomy has not been reported in the medical literature. Osteitis pubis is a painful, non-infectious, inflammatory condition involving the pubic bone, symphysis, and surrounding structures.[4] It has been reported after various urological and gynaecological procedures. A gradual onset pain in the pubic region is the primary complaint. Eventually, increasing pain makes ambulation difficult and an antalgic or waddling gait develops. Position of comfort is in adduction and flexion.[5]

After transurethral resection of prostrate, mid-inguinal pain may be related to osteitis pubis apart from more common causes like hernia, osteomylites, bursitis, Avascular necrosis, secondaries or stress fractures.

### References


1 **Canale ST**. *Campbell's operative orthopaedics*. Vol 1. 9th ed. St Louis: Mosby, 1998:844.
2 **Grace JN**, Smith, Shives TC. Wedge resection of the symphysis pubis for the treatment of osteitis oubis. *J Bone Joint Surg Am* 1989;**71A**:358–64.
3 **Schnute WJ**. Osteitis pubis. *Clin Orthop* 1961;**20**:187–92.
4 **Wiltse LL**, Frantz CH. Non Suppurative osteitis pubis in the female. *J Bone Joint Surg Am* 1956;**38A**:500–16.
5 **Turek SL**. *Orthopaedics: principles and their application*. Vol 2. 4th ed. Philadelphia: Lippincott-Raven, 1998:1661–2.



**Figure 1** Lead S2 of the ECG obtained at day 6 under β block.