

white cell count. There were 13 further cases where a peripheral white but not red cell count was available but the haemoglobin was normal. Their results, assuming a normal age-related red cell count, give a mean CSF white cell count 26% of expected. We are not aware of any previous studies on this subject, although Sarff *et al.*¹ found no association between CSF red and white cell counts in a group of high-risk infants without meningitis. The peripheral cell counts were not mentioned.

This study shows that the expected number of white cells are often not found in CSF contaminated with more than $10 \cdot 0 \times 10^9/l$ red cells. Inaccurate counts would not consistently produce the discrepancy found, and some white cells are lost either at the site of bleeding, in the CSF, or during collection and transport. Even with the CSF white cell count less than expected the possibility exists that some of the white cells observed were present in the CSF before contamination occurred and the blood staining has concealed a true meningitis. Therefore

all blood-stained CSF samples should be cultured regardless of the white cell count. Repeat lumbar punctures are advisable if it is necessary to prove the diagnosis of meningitis on the cell count, for instance with partially treated bacterial meningitis. Culture should prove the diagnosis in previously untreated bacterial meningitis. In one of our cases with a disproportionately high white cell count *H. influenzae* was grown: we presume that the other 6 patients had viral meningitis.

Reference

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Correspondence to Dr J Osborne, Bristol Royal Hospital for Sick Children, St Michael's Hill, Bristol BS2 8BJ.

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Precordial catch syndrome

DOUGLAS PICKERING

Department of Paediatrics, John Radcliffe Hospital, Oxford

SUMMARY Seventeen cases of precordial 'catch' are reported. The children varied in age from 8 to 16 years. The characteristics of this painful condition are discussed; in particular, the diagnostic indication of the site of the pain over an intercostal space, with the finger tip, is stressed. Anxiety in the child, or the parents, in relation to the complaint is discussed. Several methods of treatment are recommended.

Richard Asher, in his description of 'a pain without a name', asked at a medical meeting, 'Have any of you ever had, a very brief, sharp, needle-like pain, near the apex of the heart, acutely localised to one point seemingly inside the chest wall, but feeling as if something was adherent to it? Breathing sharpens it, so there is often a disinclination to take a deep breath while it lasts. It comes on out of the blue, it passes off in a few minutes, and although acute it is not at all distressing.'¹

Children often complain of a similar pain due to anxiety from having had the searchlight of attention pointed towards the heart.

The pain is mainly precordial, along the left sternal border or beneath the left breast. It does not

radiate. The pain is described as stabbing, shooting, needle-like, or knife-like. It may be at any grade of severity. The pain may occur at rest while watching television in a slouched posture, or during mild to moderate exercise. It causes patients to hold their breath, or breathe in a shallow fashion while they have the pain; the patient of Miller and Texidor was afraid to breathe 'as if something were catching' when she attempted an inspiration.² The technique of forced deep inspiration when the pain is present, may actually relieve the pain. Change of posture—for example, stretching upright from the slouched position, lying down, massaging the chest, or alternating the respirations between deep and shallow—may also bring relief. The pain is brief, it is generally transient, and it rarely lasts more than a minute. When it does go, it may in some instances be followed by a dull ache. The frequency varies from 3 times in an evening to once in 8 months. It is not related to hyperventilation.

Results

Seventeen children attending either the Radcliffe Infirmary, the John Radcliffe Hospital, Oxford, or

Table Precordial catch in 17 children

Case	Sex	Age (years)	Physical examination	Relevant background or points from referral letter
1	M	11	Innocent murmur	Tense
2	F	14	Innocent murmur	Neurotic
3	M	16	Ventricular septal defect and aortic incompetence	Asthmatic
4	F	14	Atrial septal defect repaired	Nervous, terrified, dizzy
5	M	10	Innocent murmur	Introspective, anxious. Car sick.
6	M	10	Peripheral cyanosis only	Anxious about everything. Parents 'worriers'
7	F	11	Coarctation repaired	Anxious. Mother nervous
8	M	8	Innocent murmur	Anxious in outpatients. Stoic at catheter.
			Secundum atrial septal defect with tricuspid diastolic murmur	Parents anxious
9	F	11	Normal heart	Anxious, obsessive. Mother has ulcers.
10	F	8	Innocent murmur	Vomiting and abdominal pain. History of early coronary heart disease in family
11	F	7	Aortic incompetence (replacement valve for infective endocarditis)	—
12	M	13	Pulmonary stenosis gradient 10 mm	—
13	M	13	Aortic stenosis gradient 30 mm	Migraine. Anxious parents
14	F	11	Innocent murmur. Venous hum	Temperamental, sensitive, faints
15	M	12	Innocent murmur. Venous hum	—
16	M	8	Bicuspid aortic valve	—
17	M	12	Normal heart	Fainted twice

clinics in the Oxford region, between the years 1972 and 1979 had a precordial catch diagnosed as a result of their descriptions of the pain. Fifteen had been referred with a murmur, one with peripheral cyanosis, and one with syncope (Table). There were 10 boys and 7 girls, varying between 8 and 16 years. Each one had been referred by a school or family doctor and had developed pain subsequently. They were of normal growth and normal intelligence.

The heart was normal in 3 children. Six of the group had innocent murmurs, two of whom also had venous hums. There were single cases of bicuspid aortic valve, mild pulmonary stenosis, mild aortic stenosis (gradient 30 mm). Two cases followed uncomplicated closure of secundum-type atrial septal defect, and there was a single case each of aortic incompetence secondary to infective endocarditis,³ aortic incompetence secondary to ventricular septal defect, and resection of a coarctation at age 7 years.

Discussion

This syndrome deserves wider recognition because it is rarely discussed in the differential diagnosis of precordial pain. It has been described in adults^{2 4 5} but not in children.

The site of the pain is characteristically described by pointing with the tips of the fingers to an intercostal space, as opposed to anginal pain where the clenched fist or hand held flat is used. The term precordial catch appears to be appropriate and makes no attempt to indicate the aetiology of the pain. It may be related to the reflections of the pleura in relation to the heart. There was a high incidence of

the 'amplifiers being turned up to full volume'. In 9 cases there was a comment in the records of anxiety in either parents or child. The incidence has been assessed by Asher as over one-third¹ in a 'normal' medical audience.

Treatment

It is important to take time over details of the history and examination in order to be in a strong position from which to reassure the parent that there is either nothing clinically wrong, or that there is only a slight abnormality. It should be explained that the heart is the focus of attention, or has been for some time, and hence there is a tendency for chest pain to be focused at the heart. The probable site of the lesion is in the chest wall. Outside activities—such as the cubs, scouts, or ballet classes—should be encouraged to divert attention from the heart, rather than sitting indoors watching television.

During puberty, in cases of bicuspid aortic valve when the gradient may change, non-invasive methods—such as echocardiography—should be used, with the Bennett formula.⁶

Discharge from a cardiac clinic or outpatient follow-up should be carried out if possible. One can always write to the family doctor asking him to refer the patient back if the pain changes in any way. Decreasing the frequency of outpatient visits is always a good boost to the patient's morale.

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Correspondence to Dr D Pickering, Department of Paediatrics, John Radcliffe Hospital, Headington, Oxford OX3 9DU.

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Hypertension and segmental renal hypoplasia causing a syndrome of haemolysis and uraemia

R DUMAS, P BALDET, B ROLIN, AND R JEAN

Clinique des Maladies des Enfants, Hôpital Saint-Charles, Montpellier, France

SUMMARY An 11-year-old girl presented with acute renal failure and severe hypertension. The blood film showed thrombocytopenia, numerous fragmented red blood cells, and a reticulocyte count of 10%. An intravenous pyelogram showed a small contracted left kidney, and plasma renin activity was increased in the left renal vein. Treatment with minoxidil and propranolol controlled the hypertension. After nephrectomy the hypertension resolved. Light microscopical examination of the left kidney showed a segmental renal hypoplasia. Malignant arterial hypertension can provoke a syndrome of haemolysis and uraemia in children. Aggressive lowering of blood pressure leads to an improvement in renal function.

Severe hypertension in haemolytic uraemic syndrome is common, and is often a complication of the renal disease. However, in adults, haemolysis and uraemia are present in some cases of malignant hypertension.¹ This case report shows that this can be so in children too.

Case report

An 11-year-old girl presented with a history of polyuria, headaches, nausea, and vomiting for one year. Blood pressure (BP) had not been measured. Gross haematuria, pallor, and purpura had developed 3 days before admission. The day she was admitted BP was 250/130 mmHg. The liver was enlarged and there was bilateral oedema on the legs. Haematocrit was 23%, haemoglobin 5 g/dl, white

blood count $12.9 \times 10^9/l$, platelets $10.0 \times 10^9/l$. The blood film showed numerous fragmented red blood cells, and the reticulocyte count was 10%. Urine analysis showed protein (+++) and numerous red blood cells. Other values were as follows: blood urea 60 mg/100 ml (9.9 mmol/l) on admission rising to 185 mg/100 ml (31 mmol/l). Plasma creatinine level was 3 mg/100 ml (265 μ mol/l), fibrinogen degradation products were between 25 and 40 mg/ml. The C3 fraction of complement was 135 mg/100 ml and the C4 was 49 mg/100 ml. Plasma renin activity, measured after the administration of 40 mg furosemide and 20 mg hydralazine, was found to be 708 ng/ml per hour. The normal value at this age, in the supine position and on a normal diet, is 3 ± 2 ng/ml per hour in our laboratory. During the next 3 weeks the child received 80 mg hydralazine (4 mg/kg) and 200 mg acetobutolone (10 mg/kg) each day, and intravenous heparin at a dose of 5 mg/kg a day, for 7 days only. After this treatment BP, blood urea, reticulocyte count, and platelet count returned to normal. However, after a few days the blood pressure again rose to 160/130 mmHg, despite continuous antihypertensive treatment. One intravenous injection of 200 mg diazoxide was given every day for 8 days. Treatment with minoxidil was started at a dose of 1 mg/kg a day with propranolol (40 mg daily) and furosemide (40 mg daily). After 3 days the BP had fallen to a normal level. The treatment was maintained at the same dose for 3 months. An intravenous pyelogram showed a small, contracted left kidney. The cystourethrogram was normal, without evidence of reflux. Left renal arteriography showed no evidence of stenosis, but