# Muscle involvement in Schönlein-Henoch syndrome

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SUMMARY Three patients with Schönlein-Henoch syndrome developed severe muscle pain in the legs. The pain, so severe that they were bedridden, subsided within a few days and was caused, we believe, by bleeding into the leg muscles.

The classic triad of Schönlein-Henoch syndrome (SHS) symptoms consists of a typical purpuric rash, arthritis, and colicky abdominal pain.[1] Muscle involvement has been reported, but seems to occur rarely. We report three patients with SHS in whom severe muscle pain in the legs was a major complaint, causing a temporary handicap.

## Case reports

**Case 1.** An 11 year old boy presented with abdominal pain and a symmetrical purpuric rash over the buttocks and the legs. SHS was diagnosed, and apart from bed rest no specific treatment was recommended. Three weeks later he was admitted to this hospital because of muscle pain in the legs. His temperature on admission was 38·5°C, blood pressure measured at the forearm was 110/70 mmHg and at the legs 120/80 mmHg. There was tenderness and weakness in the thighs and calves without any neurological deficit or other pathological findings. For three days the pain worsened, the boy was unable to stand or walk, and he was treated symptomatically with analgesics. Four days later his condition improved, the muscle pain decreased, and he was able to stand and walk.

The results of laboratory investigations were as follows: erythrocyte sedimentation rate, 110 mm/1 hour (which dropped to 50 mm/1 hour on discharge home); haemoglobin, 11·7 g/dl; and leucocytes 9100 μl (9·1 × $10^9$/l). The LE test was negative. No antinuclear antibodies were found, and the value of complement components $C_3$ and $C_4$, of creatine phosphokinase, lactate dehydrogenase, and serum transaminases were all within the normal range. An electromyography of the gastrocnemius was interpreted as normal.

**Case 2.** A 5½ year old girl was admitted because of abdominal pain of three days' duration. The abdomen was diffusely sensitive with muscle rigidity and rebound tenderness. A laparotomy was performed, but there were no pathological findings and she continued to suffer from colicky, abdominal pain and increased pain in the muscles of the legs. Eight days after admission a rash of small petechiae appeared over the buttocks and the legs. The muscle pain increased, and as a result the girl became bedridden. After a few days the pain subsided and she was able to walk again. The results of laboratory investigations were: erythrocyte sedimentation rate, 85 mm/1 hour; leucocytes, 7400–16 000/μl (7·4–16 × $10^9$/l). The values of the muscle enzymes were slightly raised: creatin phosphokinase 118 U/l (normal values 5–80 U/l) and lactate dehydrogenase 728 U/l (normal values 150–370 U/l). The values of the serum transaminases and the complement components were normal.

**Case 3.** A nine year old boy was admitted because of a second episode of SHS, and while in hospital he developed severe muscular pain in his legs. As with the previous two patients he was unable to stand or walk and the pain abated after a few days. The values of the muscle enzymes, serum transaminases, LE test, antinuclear antibodies, and the complement components were all normal.

## Discussion

In reviewing the relevant literature we came across very few reports of muscle involvement in SHS. Allen,[2] in a series of 131 cases, mentions only one patient with haemorrhage into the gastrocnemius muscle which required orthopaedic care to prevent contracture, and Meadow[3 4] reported bleeding into the calf which stimulated a deep vein thrombosis.

Having seen many children with SHS in the past, we considered ourselves familiar with the variability of its clinical picture. Nonetheless, these three patients amazed us: the pain and the tenderness in their legs, without any particular external signs, was such that they were practically bedridden and had to be carried or pushed in a wheelchair. This complaint dominated their clinical course for several days and then gradually subsided. Our diagnostic problem during the acute phase was could we relate this pain to SHS or was it related to something else—myositis, neuritis, or deep vein

thrombosis? The classic paediatric textbooks were not of much help since they made no mention of muscle pain in SHS.

The results of the laboratory investigations did not contribute additional information. The muscle enzyme studies showed normal values in two patients and a slight increase in one. Electromyography of the gastrocnemius in one of the patients was normal. Eventually it seemed quite obvious that our patients were just bleeding into their leg muscles and since the symptoms abated so quickly we decided against a muscle biopsy. It is interesting that all our three children were in hospital during a five month period (October 1982–March 1983). By the time the third patient was admitted we felt confident in our assurance to the anxious parents that their son would soon run again.

In conclusion, we do not claim to contribute to the understanding of this enigmatic entity but we would emphasise the form that the muscle involvement in SHS may take, the intriguing predilection of the disease to the lower half of the body, and the fact that muscle pain in all our patients was—how could it be otherwise—symmetrical!

**References**


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# Visceral leishmaniasis contracted in the Mediterranean area

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SUMMARY Two infants who presented with anaemia and hepatosplenomegaly were found to have visceral leishmaniasis. Diagnosis was made immediately after bone marrow aspiration in one infant, but in the other there was considerable delay. Both responded well to a course of sodium stibogluconate.

Visceral leishmaniasis (kala-azar) is endemic in many Mediterranean countries including the popular tourist areas of Southern France, Spain, Portugal, Malta, Italy, and Greece.[1 2 3] We describe two children recently referred to this hospital who were found to have visceral leishmaniasis. In neither case was the diagnosis of visceral leishmaniasis considered initially, although in retrospect both children had the typical symptoms and signs of the disease and had visited or lived in countries where it is endemic.

## Case 1

An 8 month old girl born of English parents on a farming commune in Spain, came to England in March 1980. She had been well until four months of age when she developed a dry cough, progressive anorexia, and lethargy. Her weight had fallen and her development had slowed.

Physical examination showed pallor, low grade pyrexia, pretibial oedema, massive splenomegaly (the spleen extending into the right iliac fossa), and a liver palpable 3 cm below the right costal margin. Investigations showed haemoglobin 6·2 g/dl, reticulocytes 3·5%, white blood count $6·4 \times 10^9$/l (6400/μl), neutrophils 4%, lymphoyctes 84% and platelets $32 \times 10^9$/l (32 000/μl). An initial diagnosis of malignant disease was considered but a bone marrow biopsy showed numerous amastigotes (Leishman-Donovan bodies).

She was transfused with packed cells, started on antibiotics, and treated with sodium stibogluconate (25 mg/kg bodyweight, IV) daily for three weeks. After this time her hepatosplenomegaly had decreased considerably and her haemoglobin, white blood count, and platelet values were returing to normal. Five weeks later she relapsed, with increasing hepatosplenomegaly and marked neutropenia. She was given a further three week course of sodium stibogluconate in the same dose and made an uneventful recovery.

A year later she was followed up and found to be well. It was later discovered that another infant