Recognition and treatment of abdominal wall pain

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Summary

In some patients with abdominal pain, the source of the pain may be the abdominal wall. A simple test is described which allows these patients to be identified and treated with injections of local anaesthetic and steroid. Twenty-six patients were studied, 20 of whom were available for follow-up. Sixteen of these 20 were symptom free or improved at a median follow-up period of 29 months. Failure to recognize abdominal wall pain may lead to unnecessary investigation.

Introduction

The investigation and management of chronic abdominal pain is a common problem in surgical clinics. The pain is usually assumed to have a visceral origin; however the abdominal wall is another important source of symptoms. Failure to recognize this may result in prolonged and unnecessary investigation. A study is described in which an attempt was made to identify patients with pain originating in the abdominal wall, and to assess the efficacy of local anaesthetic injections.

Method

Over a seven year period patients attending one consultant's surgical outpatient clinic were evaluated for abdominal wall pain by taking a history, performing a routine examination and, in particular, using the following test, first described by Carnett[1]. When localized abdominal tenderness was elicited during palpation the patient was asked to contract his abdominal muscles by raising his head from the couch. During this manoeuvre the pressure of the examining fingers was maintained and the patient was asked if there was any alteration in the tenderness. Carnett's hypothesis was that if the pain was arising from visceral disease the tensed muscles now protected the underlying organs so that the tenderness disappeared or became substantially reduced. On the other hand, if pain originated from the parietes, the tenderness persisted or increased.

Using Carnett's method we identified a number of patients with one (sometimes more than one) tender spot in the abdominal wall.

Once an area of abdominal wall tenderness had been identified, its position was localized as accurately as possible with a single finger tip. Provided that the clinical picture suggested no other course of action, the tender spot was injected with a mixture of 1 ml 1% lignocaine and 25 mg hydrocortisone acetate using a 21 gauge needle. To start with, a small bleb was raised in the skin overlying the tender spot. The needle was then inserted, and its point moved around in the tissues until the patient complained of pain which was similar to his original symptom. The injection was made into that point and into the immediately surrounding area.

Follow-up was at intervals of four weeks and the assessment repeated. The treatment was considered effective if the symptoms and signs had resolved completely. In cases where temporary or no relief had been obtained, further injections were given, always provided that the clinical picture did not warrant another line of treatment.

Results

Twenty-six patients were identified as having pain arising in the abdominal wall (m : f 5 : 21). The age range of the group was 19-70 years (mean 40.5 years), and the median duration of symptoms 8.5 months (range 1-84 months). The site of the pain and the male to female ratio at each site is shown in Figure 1. The right iliac fossa (nine cases) and the right hypochondrium (six cases) were the most frequently affected, with a preponderance of females at both sites. Sixteen patients had undergone abdominal surgery prior to presentation, although in no case had this been performed for the presenting symptoms. In nine of these patients the pain was related to the abdominal scar. Ten patients had undergone unsuccessful investigation in the past with a total of 20 negative radiological procedures being performed.

Twenty-two patients were relieved of their symptoms with between one and five injections (median, one); four patients failed to respond.

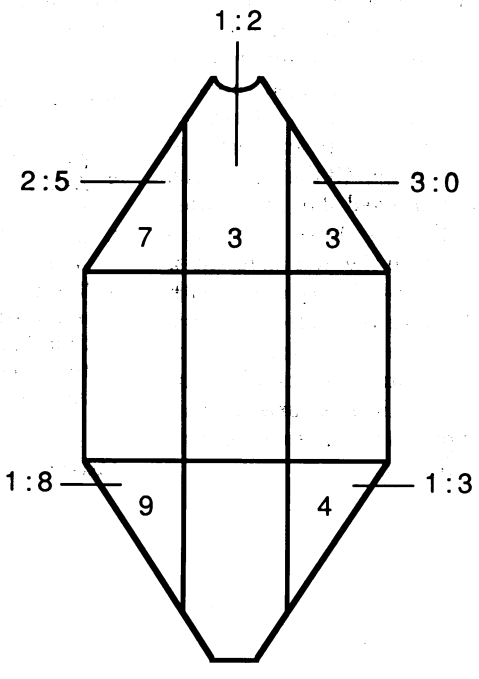

Figure 1. Distribution and male : female ratio of the sites of abdominal wall pain

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At the end of the study period an attempt was made to contact all patients who had been treated in order to ask about the long term effect of the treatment on their pain.

Of the original 26 patients replies were obtained from 20. The median follow-up time from receiving the first injection was 29 months. Sixteen patients (80%) had complete or partial relief of symptoms. One patient in the 'cure group' had, elsewhere, an alternative diagnosis of appendicitis made on relapse of her symptoms. After appendicectomy she became symptom free, but the histology of the removed appendix was normal. One patient whose pain was located in an old appendicectomy scar initially responded but subsequently relapsed with symptoms refractory to further injections. She underwent excision of the appendicectomy scar and is now pain free. In the 'no cure' group, two of the three patients contacted had undergone further investigation without an alternative diagnosis being made; in one the pain went spontaneously four months after her initial presentation and in the other the pain had improved to an extent where no further treatment had been sought. The third patient in the no cure group later had a laminectomy and is now pain free. Of the nine patients with pain related to an abdominal scar, six were available for long term follow-up. In five patients, relief of symptoms had been maintained; the remaining case required excision of the scar.

Discussion

The management of abdominal pain has been facilitated by the availability of an increasing variety of diagnostic tests. However, if it is not appreciated at the outset that the source of symptoms can reside in the abdominal wall the patient may be subjected to a fruitless search for visceral pathology. This can only result in anxiety for the patients and frustration for the doctor.

Although this study contains a small number of patients it illustrates the effectiveness of applying a simple clinical test to demonstrate the abdominal wall as a source of symptoms. Subsequent treatment resulted in 80% of our patients being completely or partially relieved of their pain. Other studies have shown similar benefits from recognizing and treating this condition. Ashby[2] obtained prolonged relief of pain for 67.3% of his patients by using an intercostal nerve block, and Mehta and Ranger[3] treated parietal pain with local injections of phenol. Of their patients, 56.3% were pain free or improved 3.5 years after treatment. Thomson and Francis[4] have shown the effectiveness of Carnett's test in the management of the acute abdomen. In their series of 120 patients with acute abdominal pain, 24 were considered to have abdominal wall pain, and in only one case of those 24 was a detectable intra-abdominal cause subsequently found; moreover in that patient there was an inflammatory appendiceal mass involving adjacent abdominal wall tissues.

The right iliac fossa and right hypochondrium were seen as the commonest sites for pain, particularly in women; this clearly has implications for potentially misdiagnosing gallstones and chronic pelvic disorders or 'chronic appendicitis'.

The frequency with which scars proved to be the source of symptoms (nine patients) should be borne in mind when examining patients who have undergone previous surgery. Stulz and Pfeiffer[5] described 23 patients presenting with abdominal pain which was shown to be due to nerve entrapment in lower abdominal scars. In contrast to our study they found that local injection of anaesthetic or steroid failed to produce a lasting benefit and they preferred to undertake neurectomy of the affected nerve. Sixteen of their patients were rendered symptom free by this treatment.

Carnett's test proved positive in one patient who was subsequently diagnosed as having appendicitis (at another hospital) and rendered symptom free by appendicectomy. We have since obtained the pathology report on the appendix and found it to be histologically normal. We do not consider therefore that this case refutes the original diagnosis of abdominal wall pain. It is however important always to take the results of the test in context with the rest of the clinical picture. False positive diagnoses may arise when examining patients with conditions which have produced inflammation in the adjacent parietal peritoneum. This structure forms part of the anterior abdominal wall and therefore has a somatic nerve supply; tenderness will still be elicited on palpation after muscle contraction.

The abolition of symptoms with local anaesthetic strongly suggests a diagnosis of an abdominal wall origin for pain in these subjects. However, this was not a controlled trial and it is therefore possible that the results could be due to a placebo effect. Perhaps such a trial should be done. However, it should be borne in mind that the success rate was high, that there were no complications due to the injections and that many patients had already suffered for some time with symptoms unresponsive to a variety of other treatments. To randomize them to a placebo injection would seem unreasonable.

References

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