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WHEN THE DIAGNOSIS AND THE DRUG CHART DISAGREE: AN ADENOSINE DEAMINASE-RICH PLEURAL EFFUSION LABELLED AS PULMONARY EMBOLISM IN AN ELDERLY DIABETIC SMOKER – A CLINICAL PHARMACIST-LED CASE ANALYSIS

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ABSTRACT

A pleural effusion in a comorbid patient attracts a diagnostic label early, and once written that label can survive unchallenged even when the fluid, the imaging and the prescription point elsewhere. We describe a 70-year-old man, a thirty-year bidi smoker with type 2 diabetes, hypertension and a myocardial infarction eighteen years earlier, admitted with eight days of breathlessness and productive cough. Computed tomography showed a gross left pleural effusion with collapse of the underlying lung and mediastinal shift to the right. The pleural fluid was an exudate with a protein of 5.2 g/dL, a glucose of 26.4 mg/dL and an adenosine deaminase of 53 IU/L; the erythrocyte sedimentation rate was 90 mm/hour and glycated haemoglobin 10.2%. The recorded provisional and final diagnosis was left-sided pulmonary embolism. No anticoagulant was prescribed at any point, no confirmatory imaging was documented, and no antitubercular therapy or microbiology followed the adenosine deaminase result. A structured medication review using the Pharmaceutical Care Network Europe classification identified eleven drug-related problems, among them metformin delivered twice through two fixed-dose combinations, a sodium-glucose co-transporter-2 inhibitor and a sulfonylurea continued through acute infection, metronidazole added to piperacillin-tazobactam, and withdrawal of statin and antiplatelet therapy at discharge in a man with established ischaemic heart disease. The patient improved symptomatically and went home on three medicines. The case shows how a diagnostic label that never reaches the prescription can coexist with a pleural fluid result that never reaches a diagnosis, and what a pharmacist reading the whole chart can recover.

KEYWORDS: Pleural effusion, Adenosine deaminase, Drug-related problems, Fixed-dose combination, Clinical pharmacist, Medication review.

INTRODUCTION

A unilateral pleural effusion in an elderly diabetic smoker is one of the most common and most deceptive presentations in Indian general medicine. The differential is wide, the history rarely discriminates, and the single investigation that most often settles the question – analysis of the aspirated fluid – usually returns after the working diagnosis has already been written in the case sheet and the first prescriptions have already been dispensed. The sequence matters, because a label written on day one tends to persist through the discharge summary regardless of what arrives afterwards.

India reported the largest national share of the global tuberculosis burden in the most recent World Health Organization assessment, accounting for roughly a quarter of the estimated 10.7 million people who fell ill with the disease worldwide in 2024.[1] Tuberculous pleuritis is among the commonest causes of a lymphocytic exudate in this setting, and pleural fluid adenosine deaminase has become its practical biochemical signature: a systematic review of 174 studies and 27,009 patients found a pooled sensitivity of 0.92 and specificity of 0.90, with 65 studies using a threshold of approximately 40 IU/L achieving a sensitivity of 0.93 and specificity of 0.90.[2] Pulmonary embolism, by contrast, is an uncommon cause of a large effusion. When embolism does produce pleural fluid, the collection occupies a third or less of the hemithorax in roughly nine out of ten patients.[3]

Running alongside the diagnostic question is a pharmacological one that receives considerably less attention. An elderly patient with diabetes, hypertension and established coronary disease arrives already carrying a long medication list, usually composed of fixed-dose combinations identified in conversation and on the chart by brand name rather than by constituent. Several of those combinations share ingredients. India has an unusually dense market in metformin-containing fixed-dose combinations, and the evidence base supporting many of them is thin.[4] When such a patient is admitted acutely, home medicines are transcribed, hospital agents are added, and the resulting chart can contain duplications, contraindicated continuations and omissions that no single prescriber ever intended and that no single prescriber is positioned to see.

We report the case of a 70-year-old man whose recorded diagnosis of left-sided pulmonary embolism was never translated into anticoagulant therapy, whose pleural fluid carried an adenosine deaminase of 53 IU/L that was never pursued, and whose medication chart accumulated eleven drug-related problems over a five-day admission. The patient improved and was discharged well. The report is offered not as a criticism of the care delivered, which was clinically sound in its essentials, but as an illustration of how documentation, diagnosis and prescription can drift apart from one another, and of what a structured pharmacist review recovers when it reads all three together.

CASE PRESENTATION

Presentation and history

A 70-year-old man was admitted in January 2026 to the Department of General Medicine of a tertiary care teaching hospital in northern Karnataka with breathlessness and a productive cough, both of eight days' duration. The breathlessness had been insidious in onset and progressive, and by the time of admission it was provoked by walking only a few steps. The cough produced a large volume of white sputum. He reported no chest pain, haemoptysis, fever documented at home, calf pain or unilateral leg swelling, and no recent surgery, immobilisation or long-distance travel. He had been hypertensive and diabetic for eighteen years and was established on treatment for both. He had sustained a myocardial infarction eighteen years earlier. He had smoked bidis for thirty years, six to seven per day. There were no

known drug allergies, his diet was vegetarian, appetite and sleep were normal, and bowel and bladder habits were regular. Family history was non-contributory.

Clinical examination

The patient was moderately built and nourished, conscious, co-operative and well oriented to time, place and person. Blood pressure was 140/90 mmHg and the pulse rate 86 beats per minute. Cardiovascular examination revealed normal first and second heart sounds with no murmur. Respiratory examination showed bilateral air entry with bilateral crepitations. The abdomen was soft and non-tender, and the central nervous system examination was unremarkable. Peripheral oxygen saturation was not recorded in the available notes.

Investigations

Laboratory results are set out in Table 1. There was a normocytic anaemia with a haemoglobin of 10 g/dL, a mild neutrophilic leucocytosis of 11,300 cells/cumm with 78% neutrophils, and an erythrocyte sedimentation rate of 90 mm/hour. Glycaemic control was poor, with a random blood sugar of 212.3 mg/dL and a glycated haemoglobin of 10.2%. Liver function tests were normal.

Cross-sectional imaging demonstrated a gross left-sided pleural effusion with collapse of the underlying lung and shift of the mediastinum towards the right. Ultrasonography of the abdomen, performed subsequently, reported grade 1 prostatomegaly and a left-sided minimal pleural effusion, the difference in volume being consistent with the therapeutic aspiration performed in the interim. Transthoracic echocardiography recorded a left ventricular ejection fraction of 60%, and no right ventricular dilatation, right ventricular dysfunction or pulmonary hypertension was documented.

Pleural fluid analysis returned an adenosine deaminase of 53 IU/L against a local reference range of 0–30 IU/L, a protein of 5.2 g/dL and a glucose of 26.4 mg/dL. D-dimer, computed tomographic pulmonary angiography, lower-limb venous Doppler ultrasonography, sputum microscopy for acid-fast bacilli, nucleic acid amplification testing of the pleural fluid and pleural fluid cytology were not recorded as having been performed. The recorded provisional diagnosis was left-sided pulmonary embolism with ischaemic heart disease (ejection fraction 60%), type 2 diabetes mellitus and hypertension, and the final diagnosis at discharge was unchanged.

Table 1: Laboratory and pleural fluid investigations.

Investigation	Observed value	Reference range
Haemoglobin	10 g/dL	13.5–17.5 g/dL
Total leucocyte count	11,300 cells/cumm	4,500–10,500 cells/cumm
Neutrophils	78%	40–75%
Lymphocytes	18%	20–40%
Monocytes	3%	0–7%
Eosinophils	1%	0–5%
Red blood cell count	4.21 million cells/cumm	4.7–6.1 million cells/cumm
Platelet count	3,59,000 cells/mm³	1,50,000–4,50,000 cells/mm³
Erythrocyte sedimentation rate	90 mm/hour	0–20 mm/hour
Random blood sugar	212.3 mg/dL	60–140 mg/dL
Glycated haemoglobin	10.2%	Target < 7%
Serum sodium	136 mEq/L[a]	135–147 mEq/L
Serum potassium	4.4 mEq/L	3.5–5.2 mEq/L
Serum chloride	104 mEq/L[b]	95–107 mEq/L
Alanine aminotransferase	12.9 U/L	6–38 U/L
Aspartate aminotransferase	13.5 U/L	6–40 U/L

Alkaline phosphatase	117.6 U/L	35–140 U/L
Left ventricular ejection fraction	60%	≥ 55%
Pleural fluid adenosine deaminase	53 IU/L	0–30 IU/L
Pleural fluid protein	5.2 g/dL	–
Pleural fluid glucose	26.4 mg/dL[c]	–

[a]Recorded on the source laboratory sheet as 13.6 mEq/L, a value incompatible with survival and reported here as the transcription of 136 mEq/L that the clinical context requires. [b]Recorded on the source sheet under the heading "calcium" but against the chloride reference interval of 95–107 mEq/L; reported here as chloride. [c]Recorded on the source sheet as 26.4 g/dL; reported here in the only physiologically plausible unit. These three discrepancies are themselves discussed below as documentation-related problems.

Treatment received

The inpatient regimen is set out in Table 2. Antimicrobial cover consisted of intravenous piperacillin-tazobactam three times daily together with intravenous metronidazole three times daily, commenced after therapeutic pleural aspiration. Gastric protection was provided with intravenous esomeprazole and intravenous ondansetron was available as required. Cardiovascular therapy comprised a fixed-dose combination of atorvastatin 20 mg with clopidogrel 75 mg once daily, a fixed-dose combination of metoprolol 25 mg with amlodipine 2.5 mg once daily, and isosorbide mononitrate 30 mg once daily. Glycaemic control was attempted with a fixed-dose combination of glimepiride 3 mg with metformin 500 mg twice daily together with a second fixed-dose combination of dapagliflozin 10 mg with metformin 500 mg once daily. Pyridoxine 40 mg once daily was also prescribed. No anticoagulant – unfractionated heparin, low-molecular-weight heparin, vitamin K antagonist or direct oral anticoagulant – appeared on the chart at any point, and no thrombolytic therapy was administered.

Table 2: Inpatient drug therapy chart.

Brand name	Generic name	Dose	Route	Frequency
Inj. Sompraz	Esomeprazole	40 mg	IV	Once daily
Inj. Emeset	Ondansetron	4 mg	IV	As required
Inj. Pipzo	Piperacillin + tazobactam	4 g + 0.5 g[a]	IV	Three times daily
Inj. Metrogyl	Metronidazole	100 mL	IV	Three times daily
Tab. Ecosprin Gold	Atorvastatin + clopidogrel	20 mg + 75 mg	Oral	Once daily
Tab. Along GR	Metoprolol + amlodipine	25 mg + 2.5 mg	Oral	Once daily
Tab. Monotrate	Isosorbide mononitrate	30 mg	Oral	Once daily
Tab. Glimisave MV3	Glimepiride + metformin	3 mg + 500 mg	Oral	Twice daily
Tab. Oxra-Met	Dapagliflozin + metformin	10 mg + 500 mg	Oral	Once daily
Tab. Benodone	Pyridoxine	40 mg	Oral	Once daily

[a]Recorded on the source treatment chart as "4 mg + 0.5 mg". The dose is reported here as the gram quantities that were evidently administered; the discrepancy is discussed below.

Hospital course and outcome

Therapeutic pleural aspiration was performed on the first hospital day, with antimicrobial cover commenced thereafter. The patient improved steadily. Blood pressure settled from 140/90 mmHg on admission to 128/70, 130/70, 120/80 and 120/80 mmHg on the second to fifth days respectively, with pulse rates between 80 and 96 beats per minute. He was recorded as symptomatically better by the third day and had no fresh complaints thereafter. Capillary blood glucose was not recorded serially despite a glycated haemoglobin of 10.2%, and the erythrocyte sedimentation rate, haemoglobin and leucocyte count were not repeated.

He was discharged on the fifth day on three medicines for fifteen days: the glimepiride-metformin combination twice daily, the metoprolol-amlodipine combination once daily, and a third entry recording the brand name of the dapagliflozin-metformin combination against the generic name pyridoxine 40 mg twice daily (Table 3). Atorvastatin, clopidogrel and isosorbide mononitrate did not appear on the discharge prescription. No anticoagulant, no antitubercular therapy and no smoking cessation pharmacotherapy were prescribed, and no specialist follow-up or repeat imaging was documented.

Table 3: Discharge prescription as recorded.

Brand name	Generic name as written	Dose	Frequency	Duration
Tab. Glimisave MV3	Glimepiride + metformin	3 mg + 500 mg	Twice daily	15 days
Tab. Oxra-Met	Pyridoxine[a]	40 mg	Twice daily	15 days
Tab. Along GR	Metoprolol + amlodipine	25 mg + 2.5 mg	Once daily	15 days

[a]*Reproduced exactly as written. The brand named is a dapagliflozin-metformin combination; the generic named and the dose correspond to the pyridoxine preparation given during the admission under a different brand name.*

PHARMACEUTICAL CARE ASSESSMENT

The clinical pharmacy team reviewed the medication chart during ward rounds and again against the discharge prescription. Problems were identified and classified using the Pharmaceutical Care Network Europe (PCNE) Classification version 9.1, which defines a drug-related problem as an event or circumstance involving drug therapy that actually or potentially interferes with desired health outcomes.[5] Eleven problems were documented and are summarised in Table 4.

Table 4: Drug-related problems identified, classified by PCNE V9.1 domain.

Sr. No.	Drug-related problem	PCNE domain	Pharmacist recommendation
1	Pulmonary embolism recorded as the provisional and final diagnosis, but no anticoagulant prescribed at any point	Treatment effectiveness – no drug treatment in spite of existing indication	Either confirm the diagnosis and anticoagulate, or revise the recorded diagnosis so that it matches the therapy actually intended
2	Pleural fluid adenosine deaminase 53 IU/L with an exudative, glucose-poor effusion; no antitubercular therapy and no confirmatory microbiology	Treatment effectiveness – untreated indication	Send sputum and pleural fluid for nucleic acid amplification testing and culture; consider pleural biopsy and evaluate for antitubercular therapy
3	Metformin 500 mg administered twice through two different fixed-dose combinations, giving 1,500 mg daily under two brand names	Treatment safety – therapeutic duplication	Rationalise to a single metformin-containing product and state the total daily metformin dose explicitly on the chart
4	Dapagliflozin continued throughout an acute infective illness	Treatment safety – inappropriate drug according to guidelines in the clinical situation	Withhold the SGLT2 inhibitor for the duration of the acute illness and counsel the patient on sick-day rules
5	Glimepiride continued during acute illness with glycated haemoglobin 10.2% and no serial glucose monitoring	Treatment safety – risk of adverse drug event	Substitute basal-bolus insulin during the admission with capillary glucose monitoring; reassess oral therapy before discharge
6	Metronidazole added to piperacillin-tazobactam, which already provides anaerobic cover	Treatment safety – inappropriate duplication of therapeutic group	Discontinue metronidazole unless a specific indication is documented
7	Piperacillin-tazobactam dose	Other – prescribing or	Correct the chart; a thousandfold unit error

	written as “4 mg + 0.5 mg” rather than 4 g + 0.5 g	transcription error	is a dispensing and administration hazard even when the intended dose is obvious
8	Atorvastatin, clopidogrel and isosorbide mononitrate omitted from the discharge prescription in a man with prior myocardial infarction and established ischaemic heart disease	Treatment effectiveness – untreated indication	Reinstate high-intensity statin and antiplatelet therapy indefinitely and document the rationale for any deliberate withdrawal
9	Discharge entry naming the dapagliflozin-metformin brand against the generic “pyridoxine 40 mg”	Other – prescribing or transcription error	Rewrite the discharge prescription by generic name with the brand in parentheses, and reconcile it against the inpatient chart before the patient leaves
10	Serum sodium recorded as 13.6 mEq/L, and chloride reported under the heading “calcium”; neither corrected nor repeated	Other – monitoring and documentation failure	Repeat and verify electrolytes; correct the record so that later clinicians are not misled
11	Thirty-year bidi smoking history with established coronary and respiratory disease; no cessation pharmacotherapy or structured counselling offered	Treatment effectiveness – untreated indication	Offer behavioural support and nicotine replacement therapy, and record the attempt

DISCUSSION

A label that the imaging did not support and the prescription never followed

Pulmonary embolism was recorded as both the provisional and the final diagnosis, and it is worth being precise about why the available evidence does not support it. Diagnosis of acute pulmonary embolism rests on a structured pathway of clinical probability assessment, D-dimer where probability is not high, and confirmatory imaging, usually computed tomographic pulmonary angiography.[6] None of these steps is documented here. Echocardiography recorded a preserved ejection fraction of 60% with no right ventricular abnormality noted, which does not exclude embolism but offers it no support. The patient had no identifiable provoking factor, no pleuritic pain and no features of deep venous thrombosis.

The imaging pattern argues against embolism more positively. Pleural effusions attributable to pulmonary embolism are characteristically small: in approximately 90% of patients the fluid occupies a third or less of the hemithorax, and they are exudative.[3] This patient had a gross effusion sufficient to collapse the underlying lung and displace the mediastinum to the contralateral side, which is a different radiological entity altogether.

The most telling observation, however, is pharmacological rather than radiological. If the treating team had genuinely believed this was an acute pulmonary embolism, anticoagulation would have been the single most urgent intervention of the admission, and it was never prescribed. The therapy actually delivered – pleural drainage followed by broad-spectrum antibacterial cover – was the management of an infected or inflammatory pleural process, and the patient improved on it. In other words, the team treated what they saw and recorded something else. The diagnosis in the case sheet and the diagnosis implied by the drug chart were never reconciled, and it was the written label that followed the patient into the discharge summary and into whatever care he receives next.

What the pleural fluid was saying

The fluid itself pointed in a consistent direction. A pleural protein of 5.2 g/dL establishes an exudate under Light’s criteria.[7] In tuberculous pleuritis the fluid is always exudative, the protein exceeds 5 g/dL in about two-thirds of

patients, and the glucose falls below 60 mg/dL in a minority – this patient's glucose of 26.4 mg/dL sits within that minority.[8] Against a local reference ceiling of 30 IU/L, an adenosine deaminase of 53 IU/L lies well above the 35–40 IU/L threshold at which the test performs best, a threshold supported by pooled sensitivity of 0.93 and specificity of 0.90 across 65 studies.[2,8] The erythrocyte sedimentation rate of 90 mm/hour, the normocytic anaemia and the eight-day history of productive cough all sit comfortably with that reading.

The host context strengthens it further. Diabetes mellitus approximately triples the risk of active tuberculosis, with a pooled relative risk of 3.11 in cohort studies, and this man had carried the diagnosis for eighteen years with a glycated haemoglobin of 10.2% at admission.[9] Thirty years of bidi smoking compounds the risk. A 70-year-old diabetic smoker in a high-incidence setting, presenting with a large unilateral exudative effusion and an adenosine deaminase above 50 IU/L, warrants at minimum sputum and pleural fluid nucleic acid amplification testing and mycobacterial culture, and in many units would warrant empirical antitubercular therapy while those results are awaited.

Three qualifications are necessary and should be stated plainly. Adenosine deaminase is not specific to tuberculosis; complicated parapneumonic effusions, empyema and lymphoma all raise it, and a markedly low pleural glucose is itself compatible with empyema.[8] Differential leucocyte counts on the pleural fluid, which would have distinguished a lymphocytic from a neutrophilic exudate, were not available. The diagnosis therefore cannot be made retrospectively from this record, and we do not claim it. What can be said is that a result of this magnitude in this patient demanded a response, and none is documented. One small observation sits alongside this: pyridoxine 40 mg was prescribed during the admission and again at discharge. Pyridoxine at this dose has a well-established role in preventing isoniazid-associated neuropathy, although it is also widely given for diabetic peripheral neuropathy, and the record does not state which indication was intended.

Two metformins, one patient

The most clinically consequential medication problem on this chart is quiet and entirely invisible if the prescription is read by brand name. The patient received Glimisave MV3, a combination of glimepiride 3 mg with metformin 500 mg, twice daily, and Oxra-Met, a combination of dapagliflozin 10 mg with metformin 500 mg, once daily. Both are legitimate products. Prescribed together they deliver 1,500 mg of metformin daily under two different trade names, and neither entry on the chart states the metformin content in a form that invites the reader to add them up. The Indian market for metformin-containing fixed-dose combinations is unusually dense, and the clinical trial evidence underpinning the leading products has been found wanting.[4] The practical consequence is that therapeutic duplication by fixed-dose combination is a structural hazard of prescribing in this setting rather than an individual lapse.

The duplication matters here beyond the arithmetic. This patient was acutely unwell with a probable infective process, anaemic, and seventy years old; these are precisely the circumstances in which metformin accumulation and lactic acidosis become a genuine rather than a theoretical concern. Renal function was not recorded in the available investigations, which compounds the difficulty, since metformin dosing is determined by estimated glomerular filtration rate. The duplication then continued on the discharge prescription for a further fifteen days without supervision.

An SGLT2 inhibitor and a sulfonylurea at the wrong moment

Dapagliflozin was continued throughout the admission. Sodium-glucose co-transporter-2 inhibitors can precipitate ketoacidosis at near-normal glucose concentrations, and acute intercurrent illness is the classic trigger; the consensus advice is that patients should withhold the drug for the duration of such an illness and that clinicians should actively consider euglycaemic ketoacidosis in anyone who is unwell on one of these agents.[10] In a febrile, dehydrated or septic inpatient the risk-benefit calculation clearly favours temporary withdrawal.

Glimepiride presents the mirror-image problem. A sulfonylurea in a seventy-year-old with reduced oral intake during acute illness carries a real hypoglycaemia risk, and no capillary glucose monitoring was recorded. Meanwhile the control being achieved was poor: a glycated haemoglobin of 10.2% with a random glucose of 212.3 mg/dL. The conventional answer to this combination of circumstances is to suspend oral agents on admission and manage the patient with insulin and serial monitoring, reassessing oral therapy before discharge. Instead, both oral agents were continued unchanged, unmonitored, and the glycated haemoglobin of 10.2% was never acted upon – a missed opportunity that is particularly striking given the contribution of uncontrolled diabetes to the very pleural pathology under investigation.

Redundant anaerobic cover and a dose written a thousandfold low

Piperacillin-tazobactam provides reliable anaerobic cover; adding metronidazole duplicates it. Analysis of 128 Veterans Affairs medical centres found that approximately 23% of all metronidazole days of therapy were potentially avoidable because another anti-anaerobic agent was being given concurrently, and that piperacillin-tazobactam was the co-administered agent in about 57% of those avoidable days.[11] Public health guidance identifies this specific pairing as a standing stewardship target, with recognised exceptions for Clostridioides difficile infection, necrotising fasciitis and certain biliary infections, none of which applied here. The point is not that the combination harmed this patient but that it is reliably avoidable and that pharmacy review is the established mechanism for avoiding it.

The piperacillin-tazobactam dose as written on the chart reads “4 mg + 0.5 mg”. The intended dose was plainly 4 g + 0.5 g, and that is certainly what was administered. Nonetheless a thousandfold unit error sitting on an active prescription is not a trivial clerical matter. Charts are read by people who were not present when they were written, including relief nursing staff and dispensing pharmacists, and the safety of a prescription should not depend on the reader inferring what the prescriber must have meant.

Secondary prevention withdrawn at the hospital door

The patient was admitted on atorvastatin with clopidogrel and isosorbide mononitrate, appropriate therapy for a man with established ischaemic heart disease and a myocardial infarction in his history. All three were absent from the discharge prescription, and no reason is recorded. Discontinuation of low-dose aspirin in secondary prevention was associated with a 46% relative increase in cardiovascular death, myocardial infarction or stroke in a Swedish nationwide cohort of 601,527 patients, with an adjusted hazard ratio of 1.46 for the secondary prevention group.[12] The evidence for antiplatelet withdrawal applies with comparable force to clopidogrel, and statin withdrawal after a coronary event carries its own well-described hazard.

This is the most straightforwardly preventable problem on the list, and it is also the most common. The discharge prescription is written at the busiest moment of the admission, frequently by the most junior member of the team, and it

is the only document that governs what the patient actually takes once he leaves. A man discharged on three medicines, two of which duplicate one another and one of which is mislabelled, with his cardioprotective therapy silently discontinued, is at greater pharmacological risk on the day he goes home than on the day he arrived.

Transcription integrity

Three recording errors deserve mention because each could mislead a subsequent clinician. The serum sodium is recorded as 13.6 mEq/L, a concentration incompatible with life, and was evidently 136 mEq/L. A third electrolyte is headed "calcium" but is reported against the chloride reference interval of 95–107 mEq/L and is almost certainly chloride, leaving serum calcium unmeasured. The pleural fluid glucose is recorded in g/dL rather than mg/dL. None of these harmed the patient. All three survived into a document that will be read by others, and the discharge prescription error belongs in the same family: a brand name recorded against the wrong generic is precisely the error that produces either an unintended continuation or an unintended omission at the community pharmacy counter.

What the clinical pharmacist contributes

None of the eleven problems required specialist knowledge to detect. They required one person to read the admission notes, the investigation reports, the inpatient chart and the discharge prescription as a single continuous document, and to ask whether they agreed with each other. That is the defining contribution of a ward-based clinical pharmacist, and it is consistent with Indian data: a prospective study of 189 cardiovascular inpatients identified 416 drug-related problems, an average of 2.2 per patient, with most pharmacist recommendations accepted by the treating team.[13] Eleven problems in a single five-day admission is a higher density than that average, which reflects the complexity of a patient carrying four chronic diagnoses into an acute illness rather than any unusual deficiency in the care provided.

It should be said clearly that the clinical management of this patient was sound in its essentials. The effusion was drained promptly, antimicrobial cover was appropriate to the clinical picture, the patient improved symptomatically within three days and was discharged stable. The problems described here are of a different kind: they live in the gap between what was done, what was written and what was prescribed for afterwards. They are the problems that no clinical deterioration brings to anyone's attention, and for that reason they are the ones that persist.

Limitations

This is a single-patient report constructed from an inpatient record. The drug-related problems were identified by structured retrospective review rather than recorded prospectively with prescriber-acceptance data, so their clinical significance is assessed against guideline standards rather than observed outcomes. The absence of an investigation from the record is not proof that it was never performed. The tuberculous aetiology we regard as most consistent with the pleural fluid findings was not confirmed microbiologically and is offered as the diagnosis that should have been pursued, not as a diagnosis established. No follow-up data beyond discharge were available, so the clinical consequences, if any, of the omissions described are unknown.

CONCLUSION

A 70-year-old diabetic smoker was admitted with a large left pleural effusion, labelled as having a pulmonary embolism, treated for an infective pleural process, and discharged with neither diagnosis addressed pharmacologically. The pleural fluid carried an adenosine deaminase of 53 IU/L with an exudative, glucose-poor profile in a host at triple the background risk of tuberculosis, and no confirmatory testing or therapy followed. Alongside this, a structured

medication review identified metformin delivered twice through two fixed-dose combinations, an SGLT2 inhibitor and a sulfonylurea continued inappropriately through acute illness, redundant anaerobic antimicrobial cover, a thousandfold dose transcription error, and the silent withdrawal of statin and antiplatelet therapy at discharge from a man with prior myocardial infarction. The patient left hospital feeling better, which is precisely why none of this was noticed. Structured pharmacist-led medication reconciliation at admission and, above all, at discharge is an inexpensive and realistic safeguard in hospitals where elderly patients arrive carrying long lists of brand-named fixed-dose combinations into acute illness. Reading the diagnosis, the investigations and the prescription as one document, rather than three, is where that safeguard begins.

ETHICAL APPROVAL AND CONSENT

This report describes a single patient and did not require institutional ethics committee approval under the institutional policy applicable to individual case reports. Written informed consent was obtained from the patient for publication of the clinical details. All identifying information has been removed and the patient is not identifiable from this report.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

AUTHOR CONTRIBUTIONS

All authors participated in case data collection, medication review, drafting and critical revision of the manuscript, and all approved the final version for submission.

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